Carboneum oxygenisatum (Carbn-o.)

Introduction

Carbon monoxide (CO) is an odorless and colorless gas that is produced anytime a fossil fuel is burned. It is a combustible gas that is soluble in water. Inhaled CO binds to hemoglobin, forming carboxyhemoglobin (COHb), as it has a 200–300 time greater binding affinity to hemoglobin than oxygen.

Continuing displacement of oxygen from hemoglobin eventually leads to tissue hypoxia. The half-life of COHb is 4–5 hours at room air, 60 minutes on 100% O2 at sea level and 30 minutes or less in hyperbaric oxygen atmosphere.[1]

Incidentally, our bodies produce small quantities of CO through the degradation of heme, which activates many detoxification processes.[2] The World Health Organization has issued the following guidelines for levels of CO in the air in order that blood COHb levels remain below 2.5%:

- 87 ppm for 15 minutes
- 52 ppm for 30 minutes
- 26 ppm for 1 hour
- 8.7 ppm for 8 hours.[3]

Smokers are an interesting exception to these recommendations. While non-smokers generally have COHb levels of about 0.5%, as a result of normal endogenous production of carbon monoxide, smokers can have COHb levels of up to 13%, yet they seem not to experience the types of effects that do those exposed to ambient CO who attain similar COHb levels. Without understanding why smokers are seemingly “immune” to such levels of COHb, it is clear that they are an exception to the general rule and should not be used to estimate the effects of low level carbon monoxide exposure on non-smokers in the home.[4]

People with blood COHb levels of around 5% or below generally experience no symptoms. However, there is a substantial amount of evidence that such low levels have negative physiological effects. Further, epidemiological studies have shown that levels of CO in the air correlate with mortality rates and hospital admissions for heart failure. This has important public health implications, as heart disease is a leading cause of death in the USA and the UK.[5]

Incidentally, in the preparation of this long and detailed monograph, the author doesn’t have any clue on how useful Carboneum oxygenisatum will become in general homeopathic practice. Similar to Plumbum metallicum, the toxicology re-

ported in the medical literature of CO is simply overwhelming, as in the USA only, about 50,000 persons are admitted in ER for CO poisoning with about 3,500 deaths annually.[6]

Nash had previously said about Plumbum metallicum, “Notwithstanding the very extensive provings [toxicology], this remedy has not been so useful as it would seem it should be.”

Will the fate of Carboneum oxygenisatum be the same as the one of Plumbum metallicum?

I can’t wait to witness how Carboneum oxygenisatum will play out in practice with the advent of this monograph in the context of the current COVID-19 pandemic, its great newly completed proving and the upgrade of our repertory.

Shall we be surprised if it doesn’t find an important clinical niche? Not really, as Carboneum sulphuratum is another remedy with a large materia medica depicting the intoxication of its vapors of industrial workers, which is rarely prescribed and greatly resembles Carboneum oxygenisatum.

However, it must be stated that since 2008 when the MMPP monograph of Plumbum metallicum was completed and additions were made to the repertory, its use in practice has augmented considerably, but still not to the level one could expect by reading the extend of the realm of its intoxication.

However, it should be pointed out that the slow chronic intoxication of CO over many years is associated with a more particular pathogenesis than the acute state of poisoning and may show to be more useful in practice.

Also it is important to point out that there has never been a proving of Carboneum oxygenisatum until 2020 when one was initiated by the MMPP after the remedy was used clinically in COVID-19 patients.

The proving symptoms will be important to establish the most characteristic symptoms of the remedy versus the ones already gathered in victims of intoxication, and help to confirm that some of the symptoms found in these cases of intoxication actually belong to the pathogenesis of Carboneum oxygenisatum and not to the intoxicated victims. For instance, a CO intoxicated person reported having ongoing HA and that the only mean to alleviate them was by sleeping. Was this modality characteristic of Carbn-o. or of it’s the victim of intoxication?

We now have the answer, as the symptom can found in its new proving.

Or a CO poisoned person reported that he had difficulty waking in the morning. Was this symptom characteristic of CO poisoning, or was it a characteristic of the poisoned person that preceded the intoxication incident?

Now we know that it is a characteristic of Carboneum oxygenisatum, as it was found in provers.

Finally, will the symptoms of CO that are related to hypoxia show to be less clinically important than the ones that are specific to CO poisoning, such as the flu-like illness or the changes in personality.

In the proving we found flu-like symptoms but we have not seen the changes in personality in the proving.

Clinically the great variety of symptoms presented by multiple sclerosis patients has been found irrelevant for prescribing, unless it presents unexpected symptoms such as the paresthesia being worse from 3 to 5 PM.

On April 1, 2020, I began supporting Dr. Frédéric Rérolle by following with the night shift nurses the residents of a nursing home in Lyon, France, who were sick with COVID-19. Each evening (the afternoon for me), Dr. Rérolle would send me a report on 5 or 6 febrile patients who were experiencing respiratory distress. We had no subjective symptoms from these patients, as they were too severely mentally handicapped to provide such symptoms. The symptoms I would work with for each patient were as a rule the oxygen saturation index, the pulse rate, the temperature taken on the forehead, the results from auscultation, the description of the respiration and demeanor of the patient.

I quickly realized that we needed to better understand the materia medica corresponding to the later stages experienced by COVID-19 patients, especially when they began to experience severe respiratory distress associated with pronounced desaturation.

As I was continually looking at the literature on the acute respiratory distress syndrome experienced by COVID-19 patients, I came across the following article *COVID-19: Attacks the 1-Beta Chain of Hemoglobin and Captures the Porphyrin to Inhibit Human Heme Metabolism*.[7] Through genomic modeling it was suggested that certain proteins of SARS-CoV-2 attack the heme part of hemoglobin, which incapacitates hemoglobin to carrying oxygen and carbon dioxide.

I then went back to the materia medica to study all the remedies associated with asphyxia and came across the following passages from E.A. Farrington about Carboneum oxygenisatum, “A wide field for study, and once scarcely yet trodden by the therapeutist, is that which gives us substances capable of causing and curing asphyxia.

“Carboneum oxygenisatum, as a remedy serviceable in asphyxia arising from pulmonary affections, it would seem to stand between Carbo vegetabilis and Opium having the hyperemia of the latter with the coldness of the former. Cases of poisoning with the gas have developed pleurisy, bronchitis, emphysema, with bloody sputum, weakened vesicular murmur, and pneumonia. Its subjective symptoms are: ‘Anxiety in the chest or *feeling of a heavy load on the chest,* etc.’ There are also recorded, rattling of mucus in the air-passages, bloody mucus raised from the bronchi, *heat in chest*, and abdomen, extremities cold.

“Want of oxygen in animal tissue invariably leads to a general disturbance, the central phenomena of which appear in respiratory and cardiac symptoms. The blood in the capillaries is retarded in its flow, and at length fails utterly to pass into the veins. Then the heart, which at first worked harder to overcome the resistance, beats more and more quickly, but at the same time more and more feebly, until it finally becomes paralyzed. Such a calamity follows first, because the heart muscle is exhausted by its undue efforts, and secondly, because its blood, deprived of oxygen, fails to impart its essential stimulus.

“The symptoms which more or less characterize asphyxia are: ‘Pectoral anxiety, dyspnea, rapid feeble pulse, surface coldness, restlessness or stupor, with cold blue skin.’

“*The patient soon feels stupid*, confused or acts like one drunk. Respiration becomes *stertorous* and slow; the breath becomes cool, and complete unconsciousness. *The temperature falls perceptibly*.”[8]

I was stuck by the similarity of those symptoms with the ones I had seen in COVID-19 patients. I continued to investigate this trail and began comparing the respiratory pathology of COVID-19 patients with the one of CO poisoned people, and discovered many characteristic similarities and more particularly the pronounced diffuse *pulmonary edema* and the *ground-glass appearance* of the lungs that is characteristically found CO poisoned victims and patients with SARS, MERS and COVID-19. The more I compared the pathologies of CO poisoning with the one of COVID-19 patients, the more I saw similarities. We therefore introduced on the evening of

April 8 Carboneum oxygenisatum in patients with respiratory distress at the nursing home.

On Friday evening April 10, before retiring for the Easter weekend, Dr. Rérolle wrote me in his daily report, “Here is our first case in which Carboneum oxygenisatum was prescribed. I can confirm that the severe forms of COVID-19 that I have unfortunately had in older people have presented a picture very similar to the MM of Carboneum oxygenisatum.

“It is still too early, but I am happy to be able to give you the beginnings of the first results on a patient who has been seriously affected since at least March 23 and who was managed to stay alive but without stable results, forcing us to switch from one remedy to another with a saturation which remained low between 83-(87% under Carb-v.) under O2, an oscillating fever and severe breathing difficulties.

“Under Carboneum oxygenisatum 200 and then 1M since the evening of April 8: the saturation rose quickly to 90, 93% and remains stable, no fever and very good clinical improvement on auscultation. The whole team finds him transformed, rejuvenated! To be followed but after many failures and deaths I regain hope.”

By April 19, the epidemic in the nursing had been completely tamed and three of these elderly and greatly handicapped persons in the severe/critical stage of Covid had been saved by the use of Carboneum oxygenisatum.

Since April 2020, many skilled practitioners have confirmed the beneficial use of Carboneum oxygenisatum in COVID-19 patients, especially in the later and last stages of the disease. Pay attention to cases #15, 16 and 18, which are all extremely severe cases of COVID-19 and see how Carboneum oxygenisatum has saved those lives. Just imagine genuine if homeopathy had been used as official medicine during epidemic how many lives would have been saved?

In case #13, Dr. Michel de Sonnaville from the Netherlands clearly confirmed the use of Carboneum oxygenisatum in COVID-19 patients in both the early and later stages of the disease, but also for prevention and in the treatment of long haulers or long COVID-19.

As far as my experience goes, long-haulers have responded well to the remedy they needed when they were in the first stage of COVID-19, such Bryonia alba, or in the later stage of the disease, such as Carboneum oxygenisatum, or to the remedy they needed before they fell sick to COVID-19, which is a good example of individual susceptibility to chronic diseases, as it was originally reported by Hahnemann in his *Chronic Diseases*.

Practitioners will have the same success with these cases as they would have in their general practice to treat patients with chronic diseases.

I can recall a COVID-19 long hauler patient who began to come out of brain fog and physical exhaustion within 15 minutes that she had been experiencing constantly for about nine months after taking a single dose of Carboneum oxygenisatum 1M.

I therefore recommend all my colleagues a careful study of the materia medica of Carboneum oxygenisatum, as its pathology is most similar to COVID-19. Many practitioners including myself have cleared in patients sequellae of COVID-19 including chronic difficulty breathing and low oxygen saturation index, fatigue and symptoms related to poor circulation/blood clots, such as cyanosis, chilblains, mental confusion, disorientation.

An interesting findings one will notice by reading the materia medica of Carboneum oxygenisatum from Generalities downward, it is that the great majority of the characteristic symptoms reported in the proving that was ably directed by Dr. Klaus Habich greatly confirmed the ones resulting from intoxication with carbon monoxide poisoning and the cured cases as whole greatly confirmed the symptoms from the previous two sources. Great work Klaus and coworkers!

The great clinical successes we have so far obtained with Carboneum oxygenisatum has so far went beyond all our expectations.

CO poisoning has occupied the thinking of physicians since the time of Aristotle, who is credited with the first recognition of CO poisoning in the third century BC: “Coal fumes lead to heavy head and death.” The ancient Greeks and Romans used CO to execute criminals and commit suicide.[9]

Historians have reported that two Byzantine Emperors have suffered carbon monoxide poisoning from the burning of coal inside the room they were sleeping in, a usual method of indoor heating, while on their visit to their kingdom, one, Julian the Apostate, almost died while the other, Jovian, died at the age of 33.[10]

CO was first identified in the 18th century as a combustible gas that burned with a bright blue flame. However, it was not until CO was identified as the toxic substance in coal gas that Aristotle’s observation was finally verified. Numerous case reports of its ill effects followed this recognition.[11] CO poisoning has since been studied extensively.

At the time of the First International Congress of Labor, held in Washington, D.C. in 1910, delegates were asked to call the attention of their respective government to the steady increase in industrial CO and requested to make investigations leading to its prevention. The rapidly increasing use of internal combustion engines was mentioned as one source of this toxic gas.[12]

Incidentally, CO poisoning is repeatedly being stated in the modern medical literature as the leading cause of morbidity and mortality from poisoning in the world.[13]

However, its seems that an entire class of scientists and physicians have a blind spot for the incredibly high mortality associated with prescribed and illicit drugs, as there are about 15,000 deaths annually due to prescribed opioids alone, or heroin alone or cocaine alone in the USA (50,000 cases of poisoning with 3,500 deaths annually in the USA).[14]

If we would exclude intoxication related to prescribed and illicit drugs, CO is likely responsible for more than one-half of all fatal poisonings that are reported worldwide each year.[15]

CO poisoning destroys lives and families, and takes the sparkle of life out of survivors (see the case of this 52-year old woman in the Generalities section).

However, it is estimated that at least one-third of nonfatal cases of CO poisoning usually go undetected and undiagnosed and are found through inspection.[16] For instance, in a 1945 study that evaluated 5,000 drivers of vehicles operated by charcoal gas in Finland, symptoms of CO poisoning were found in 67.5% of the cases. The symptoms of acute poisoning were complained of by 3,069; symptoms of chronic poisoning, by 2,506 drivers. In 530 cases the poisoning had caused inability to work.[17]

Several studies have attempted to determine levels of carbon monoxide in people and in their homes. In a 2002 study, the COHb blood levels were measured in about 3,000 children in the USA, as well as CO concentrations in 1,820 randomly selected homes. It was found that *22.5% of children* (n = 517) had blood COHb levels above 3%. Highest levels were around 8%. Of the homes tested for CO concentrations, *about 17% (n = 306) had levels above 10 ppm.* These elevated levels of CO were primarily caused by defective or improperly vented fuel burning appliances. However, passive tobacco smoking was not taken into account, and it is known that this can play an important part in raising indoor concentrations of CO. *Nevertheless, this study suggests that a large proportion of the population may be unknowingly exposed to carbon monoxide levels above those considered safe*.[18]

There is a great variation in the intensity of the symptoms experienced by the victims and bodily damage and as well as which tissues and organs is most affected for the same exposure, which depends on individual susceptibility.

Any one who reviews the literature on CO poisoning is impressed with the great variety of injuries, which it may set up. *In this respect CO differs from every other known poison.* Even the symptoms of acute poisoning vary more than is usual, and when it comes to the after effects, it seems that any organ or tissue of the body may show injury, and it is impossible to predict what the course of any individual case will be.

Further, there is a marked variation in the way different individuals are affected by long exposure to small quantities of carbon monoxide. One person will suffer from ill-health, constant headache, neuralgic pains, perhaps albumen in the urine, after a few months' work in a room where others have passed several years without noticing any symptom.[19]

These characteristics of CO deserved not only the name of "*silent killer*," but also of being "*the great imitator*," because of the myriads of clinical pictures associated with intoxication.

For instance, clinical syndromes and brain imaging have been found identical for Parkinson's disease and multiple sclerosis and CO poisoning. Further, it is interesting to note that that certain classical symptoms in the homeopathic materia medica, such as the desire to be nude, fear of being infected, kleptomania, obsessive-compulsive behaviors, repetitive hand washing, checking behaviors, counting rituals or foolish behaviors have been associated with brain lesions commonly found in CO poisoned victims.

Although the brain and heart are the most sensitive, but other organs can also manifest the effects of CO poisoning. One-fifth to one-third of patients with severe CO poisoning, particularly those requiring intubation, develop pulmonary edema. This is caused in part by cardiac depression directly from CO and acute respiratory distress syndrome (ARSD).[20]

The earliest symptoms associated with CO poisoning are nonspecific and can readily be confused with many other syndromes. In fact, the most common misdiagnosis of CO poisoning is a viral syndrome, such as a flu-like illness.[21]

It has been estimated that 3% to 24% of patients who are admitted into ER in the winter with a flu-like illness have CO poisoning. The most common symptom, headache, is usually described as dull, frontal, and continuous. CO poisoning can

also be frequently misdiagnosed as food poisoning, gastroenteritis, and even colics in infants.[22]

Acute severe CO poisoning may be easier to recognize and diagnose than chronic low-level exposure, which may produce more subtle and vague symptoms that can easily be mistaken for other common illnesses, such as *chronic fatigue syndrome, depression and influenza*.[23]

For instance, the mother of a family that was chronically exposed to CO over several months experienced headaches, dizziness, and extreme exhaustion, which she attributed to a viral infection. Her 2.5-year-old daughter was lethargic and irritable, and vomited on several occasions. During the period of her exposure, this child had eight clinic visits and was examined by three different pediatricians. Her symptoms were repeatedly attributed to “the flu.”[24]

In another instance, a 3 1⁄2-year-old girl was admitted to the hospital several times with acute, life-threatening events, acidosis, and flu-like symptoms. The diagnosis remained elusive until after careful questioning of family members and a home visit, chronic CO poisoning was diagnosed.[25]

In summary, symptoms of CO poisoning can be very diverse, nonspecific and disparate and can at times be very difficult to diagnose without evaluation of the COHb level.[26]

However, CO toxicity is prominent on the organs that are more sensitive to oxygen deprivation, such as the brain, heart and kidneys.

However, no tissue is spared and thus CO can thus present innumerable clinical pictures. For instance in one patient, myonecrosis can lead to compartment syndromes while in another one myonecrosis will lead to acute kidney failure and in a third one to heart failure.[27]

The most common findings in patients severely affected with CO poisoning are abnormalities of the nervous system, lungs, skin and blood pressure, elevated respiration, tachycardia, arrhythmias, dizziness, nausea, vomiting, headaches, excessive sweating, palpable liver, localized edema, mucosal changes, bleeding tendency, fever, leucocytois, proteinuria, glycosuria and abnormal urinary sediments.

Almost every abnormality of mental status has been described and most patients will show some mental abnormality. *Disorientation*, confusion, *indifference*, excitement, depression, apprehensions or personality changes are common symptoms. The neurologic responses vary widely without apparent pattern.

Incontinence of urine and stool is common in severely ill patients. Abnormality in the lungs is frequently seen in the first 48 hours. Fine, medium, coarse or bubbling rales or rhonchi are common signs. Pneumonia is often suspected. Some patients complained of pain or soreness in one or more regions of the body or in their entire body during the course of their illness.

Death is not an uncommon sequela of CO poisoning and there is a significant correlation of abnormal neurologic signs, irregular cardiac rhythm, excessive sweating in the first 24 hours, rales or rhonchi in the chest, elevation or abnormal blood pressure, elevated respiratory rate, high red blood cell count, or evidence of bleeding at any time with subsequent death.[28]

The adverse long-term sequelae of CO poisoning have traditionally been attributed to hypoxic injury secondary to CO occupation of oxygen binding sites on hemoglobin. However, many physiological changes associated with CO poisoning are not seen in other forms of hypoxia. For decades clinicians have noted that the COHb levels of patients do not correlate well with their signs and symptoms or their ultimate clinical outcome. If CO poisoning were a simple reduction in available tissue oxygen and secondary tissue/organ hypoxia, then the relationship between the COHb level and symptom severity should be nearly linear, but this is not the case.

Furthermore, a simple hypoxic injury does not explain the strange cardiac dysrhythmias that often develop in the face of CO poisoning. Likewise, it fails to explain the neurologic and cognitive dysfunction that patients often develop, not immediately, but typically in the weeks and months following acute poisoning. Interestingly, unlike the other mechanisms of hypoxia, CO poisoning can induce neurological damage without loss of consciousness.[29]

It was eventually discovered that CO reacts with a number of other heme compounds besides hemoglobin: myoglobin, hydroperoxidase and cytochrome oxidase and P-450. Myoglobin is an oxygen carrier protein that acts to facilitate oxygen diffusion in skeletal and cardiac muscle cells. CO neutralizes myoglobin, which leads to a decrease in cardiac output and generalized muscle weakness even in cases of mild CO poisoning.[30]

Just by itself the binding of CO to the mitochondrial cytochrome oxidase is enough to disrupt cellular respiration, creating histologic hypoxia and directly compromising life.[31]

However, inactivation of cytochrome oxidase is only an initial part of the cascade of inflammatory events that results in subsequent cellular loss.[32] For instance, acute CO poisoning causes intravascular neutrophil activation due to interactions with

platelets, which is followed by perivascular oxidative stress and a cascade of immune-mediated sequelae.[33]

Immediate death from carbon is most likely cardiac in origin, because myocardial tissue is most sensitive to hypoxic effects of carbon monoxide. Severe poisoning results in marked hypotension and lethal arrhythmias, which may be responsible for a large number of pre-hospitalization deaths.[34]

In summary, CO poisoning pathology is a markedly complex phenomenon that involves extensive disruption of cell signaling and profound oxidative stress, which is sometimes facilitated by *hypoxia but is not entirely dependent upon hypoxia*.[35]

CO intoxication can be either acute or chronic. It could be a recent intense exposure, which usually leads to death or to the ER, or it can be chronic as a minor exposure over days, weeks, months and years, which leads to a slow deterioration of the organism.

Under acute poisoning are included those cases in which the manifestations appear very promptly after the exposure of the individual to the toxic agent, whether such exposure be accidental or intentional. The symptoms include headache, an indefinite feeling of illness with fullness of the head, dizziness, nausea, great muscular weakness, and, if the influence of the gas is continued, drowsiness, unconsciousness and loss of control of the sphincters. Muscular twitching or even convulsions sometimes occur. The skin and mucous membranes may be cyanotic and occasionally an erythematous rash resembling frostbites may be seen over the malar prominences. The sequelae of acute poisoning include pneumonia, cardiac palpitation, localized hyperemias, gastrointestinal disturbances, transient glycosuria, cutaneous eruptions, localized edema and gangrene. In the nervous system, paralyses of central or peripheral type and choreiform movements are seen, persistent headache is complained of and mental changes take place, often only mild hallucinatory states but more commonly distinct confusional insanities.

The first consistent symptom with controlled CO exposure is a mild frontal headache when COHb reached 15–20%. Unfortunately, there were no unique features of these headaches.

One hundred patients referred for hyperbaric oxygen treatment for CO exposure answered questions concerning their headaches. The location was frontal for 66%, while 58% had more than one location. Pain was dull in 72%, sharp in 36%; constant in 74%, throbbing in 41%, and intermittent in 16%. Patients with ischemic heart disease had decreased exercise duration due to increased chest pain when COHb levels were 2.7–5.1%. There is no concentration of CO that does not exert a

significant and measurable untoward effect upon a diseased cardiovascular system.[36]

The frequency of symptoms found in a series of 100 patients with CO poisoning who were admitted to the ER were headaches (55%), ataxia, dizziness and imbalance (49%), loss of consciousness (46%), disorientation (29%), nausea and vomiting (26%), loss of higher brain functions (24%), motor disorders (20%), requiring mechanical ventilation (13%), visual disturbances (10%), abnormal ECG (8%), cardiac arrest (5%), cyanosis (5%), metabolic acidosis (5%), loss of hearing or tinnitus (3%), sensory disorders (3%), hypotension (2%), bradypnea or respiratory arrest (2%), convulsions (1%), atrial fibrillation (1%), urinary incontinence (1%), pulmonary edema (1%), cherry-red coloration (1%).[37]

In another series of 304 cases of accidental poisoning, the main symptoms at admission in the mild poisoning cases (*n* = 180) were headaches (33%), fatigue (31%), nausea (19%) and dizziness (13%). Seventy-four patients (25%) were admitted to the ICU and 46 (62.2%) required mechanical ventilation. The mean duration of the ICU stay was 11 days (range 1–79 days). The mean duration of mechanical ventilation was 8.5 days (range 1–57 days). Poisoning was graded severe in 41 (89.1%) of the 46 patients who required mechanical ventilation and 57 (77.0%) of the 74 patients admitted to the ICU. A total of 10 patients died (3.3%), all were cases of severe poisoning: 7 in the ICU and 3 patients after treatment in the emergency room. Age above 60 years old significantly increased the likelihood of having severe CO poisoning compared with age younger than 19 years (OR = 4.3).[38]

Under the heading of chronic poisoning by CO are placed those cases presenting headache, nausea, vertigo, general weakness and anemia with some slowness of intellectual activity and failure of memory. These are all symptoms of the acute type, but appear gradually in individuals who are chronically exposed to the toxic action of CO and which will lead to slow permanent debility.[39]

Chronic CO poisoning is characterized by three major symptoms, always appearing together, which are, 1) Almost constant tiredness, muscular fatigue, intellectual apathy, depression, slowness of speech and impaired memory are typically found, with occasional concurrent sexual impotence. 2) Severe headaches that tend to be persistent, and usually more severe during exposure, although it may continue for several hours after work. 3) Vertigo that tends to precede faintness.[40]

Retinal hemorrhages occur with exposures longer than 12 hours. Cherry-red skin coloration occurs only after excessive exposure (it was found in 2 to 3% of cases

who were referred to a hyperbaric center). Another classic but uncommon phenomenon is the development of cutaneous bullae after severe exposures. These bullae are thought to be caused by a combination of pressure necrosis and possibly direct CO effects in the epidermis.[41]

The bigger problem with CO poisoning is the associated morbidity that survivors risk even after acute treatment, and the most serious of these is persistent or delayed neurologic and cognitive sequelae, which occurs in up to 50% of patients with symptomatic acute poisonings.[42]

Neurocognitive deterioration is delayed and preceded by a lucid period of 2 to 40 days after the initial acute poisoning. In patients admitted to an intensive care unit for severe CO poisoning and treated with 100% oxygen, 14% of survivors had permanent neurologic impairment. In a Korean series of 2,360 CO-poisoned patients, 3% continued to show memory failure or parkinsonism 1 year after exposure. In another series of 63 seriously poisoned patients memory impairment was found in 43% and deterioration of personality in 33% at 3-year follow-up. Children also develop behavioral and educational difficulties after severe poisoning. However, patients older than 30 years of age are more susceptible to the development of delayed sequelae. Most cases of delayed neurocognitive sequelae are associated with loss of consciousness in the acute phase of toxicity.[43]

The delayed effects of CO poisoning are varied, unpredictable, and often characterized with apathy, mutism, amnesia, and urinary incontinence after 2 to 4 weeks of apparent recovery.[44]

Reported cognitive symptoms associated with delayed effects of CO poisoning in a series of 86 patients include *apathy* (100%), *disorientation* (100%), amnesia (100%), hypokinesia (95%), mutism (95%), irritability (91%), apraxia (76%), bizarre behaviors, such as silly smile or frowning (70%), mannerism (41%), irrational confabulatory talking (30%), insomnia (19%), depression (15%), delusions (12%), echolalia (2%), elated moods (2%); and reported neurological symptoms include urinary or fecal incontinence (93%), gait disturbance (91%), glabella sign (91%), grasp reflex (87%), increased muscle tone (86%), retropulsion (72%), increased deep tendon reflexes (22%), flaccid paralysis (19%), tremor (14%) and dysarthria (9%).[45]

Almost every known neurologic and psychiatric syndromes have been associated with CO poisoning, including Korsakoff's syndrome, amnestic syndromes, cortical blindness, multiple sclerosis, Parkinson’s disease, peripheral neuropathy, dementia, obsessive-compulsive disorders, psychosis, including manic-depressive psy-

chosis, Tourette syndrome, Wernicke's aphasia, apraxia, agnosia, chorea and incontinence.[46,47]

In 10 patients who developed psychiatric syndrome following CO poisoning, five were diagnosed with acute functional psychosis, three with major depression and two with schizophrenia. On neurological examination, glabella sign, grasp reflex, increased deep tendon reflexes, increased muscle tone and retropulsion were found in 8 out of 10 of these patients.[48]

Neurocognitive sequelae probably involve lesions of the cerebral white matter. Weeks after exposure, autopsies show necrosis of the white matter, globus pallidus, cerebellum, and hippocampus. Magnetic resonance imaging studies confirm the damage to the white matter and hippocampus. Animal studies show that having a markedly elevated COHb level alone cannot cause similar white matter lesions but that there must also be an episode of hypotension. The fact that the areas permanently damaged in serious CO poisoning cases are the areas with the poorest vascular supply in the brain is consistent with these findings.[49]

The examination of thirty-two brains of victims of CO poisoning showed a characteristic symmetrical softening of the globus pallidus, with degenerative changes in the arterial walls leading to the deposit of lime salts even by the third day.[50]

“Psychic akinesia” is a syndrome which has been described after basal ganglia damage, especially located in the globi pallidi, caused by CO poisoning (also in a case with wasp stings). Incidents of CO poisoning seems to be particularly lethal to the basal ganglia, but the extent and degree of the lesions vary in accord with the severity of CO poisoning.[51]

Three hundred and forty-five patients with a mean COHb of 21% treated with normo- or hyperbaric returned to baseline immediately or within a matter of days. Ninety-seven percent of these patients were able to return to work in one month. Persistent symptoms in 32% included fatigue, headache, memory and sleeping problems, decreased concentration, behavioral and visual disturbances. Also the accidentally poisoned with no cognitive sequelae at 6 weeks were seven times more likely to have depression and anxiety at 12 months than at 6 weeks.[52]

However, it is interesting to note that a number of patients with cognitive or neurologic sequelae and clear brain lesions viewed on MRI can begin to recover until no obvious signs or symptoms of deficits can be noticed.[53]

In one review, recovery from delayed neurologic sequelae occurred within 1 year in 75% of the patients.[54] In a series of 242 patients with CO poisoning, 9.5% devel-

oped parkinsonism with a median latency of 4 weeks and mild to severe impairment of cognitive functions, gait disturbances, urinary incontinence, and mutism. There was no resting tremor but some had an intentional tremor. Other signs included small stride, hypokinesia, masked faces, rigidity, release reflexes, and retropulsion. Abnormalities in the white matter and globus pallidus seen on computed tomography were similar to CO-exposed patients who made a full recovery. Standard drug treatment for Parkinson's disease was not effective in this study, but in other case reports, patients responded favorably to L-dopa medications. Sixteen patients followed for 1 year had recovery at 6 months in 81%.[55]

Blood flow remains elevated until loss of consciousness, when transient cardiac compromise causes blood pressure to decrease. Because of this, autoregulation until cardiovascular homeostasis is exhausted and asphyxia or apnea begins, brain hypoxia is probably not an initial feature of CO poisoning. Acute and intense CO poisoning can lead directly to diffuse hypoxic–ischemic encephalopathy predominantly involving the gray matter. There is a predilection for the temporal lobe and the hippocampus. The globus pallidus is the most common site of involvement in CO poisoning. The damage usually occurs immediately. The predilection for the globus pallidus is unclear. Necrosis of the globus pallidus is not necessarily related to the development of parkinsonism and vice versa, probably because the damage to the nigrostriatal pathway is incomplete. The caudate nucleus, putamen, and thalamus occasionally are involved in CO poisoning but less so than the globus pallidus. The lesions usually appear as asymmetric hyperintense foci. Energy production and mitochondrial function are restored after carboxyhemoglobin levels decrease, but the transient changes can cause neuronal necrosis and apoptotic death, which lead to diffuse brain atrophy.56

Peripheral neuropathies occurring in the cases of CO poisoning show histologically demyelination with preservation of axons.[57]

Survivors of CO poisoning also face potential impairments in their cardiac function, and exhibit an increased risk for cardiovascular-related death in the following 10 years.[58]

Globus pallidal lesions provoke poverty of movements, inactivity and a cataplectic state resembling the "status adynamicus," i.e., *a state of decreased spontaneity and initiative*.[59,60]

Perhaps some of you remember the book and movie *Awakenings* in which psychiatrist Oliver Sacks discovered a number of survivors of the encephalitis epidemic that occurred around the world between 1915 and 1926, which was called enceph-

alitis lethargica, or sleepy sickness. Nearly five million people were affected, a third of whom died in the acute stages. No recurrence of the epidemic has since been reported, though isolated cases continue to occur. Many of those who survived never returned to their pre-existing "aliveness". The disease affects the pallidal area of the lentiform nucleus with lesions that are also found in CO poisoning, leaving some of their victims in a statue-like condition, speechless and motionless.[61]

They would be conscious and aware—yet not fully awake; they would sit motionless and speechless all day in their chairs, totally lacking energy, impetus, initiative, motive, appetite, affect or desire; they registered what went on about them without active attention, and with *profound indifference*. They neither conveyed nor felt the feeling of life; they were as insubstantial as ghosts, and as passive as zombies.[62]

It is interesting to note that numerous accounts of encephalitis lethargica have been reported throughout history, such as:

- In 1580, Europe was swept by a serious febrile and lethargic illness, which led to parkinsonism and other neurological sequelae.

- In 1673–1675, a similar serious epidemic occurred in London that Sydenham described as “febris comatosa.”

- In 1712–1713, a severe epidemic of encephalitis lethargica occurred in Germany.

- Between 1750 and 1800, France and Germany experienced minor epidemics of “coma somnolentum” with parkinsonism, including hyperkinetic hiccough, myoclonus, chorea and tics.

- Between 1848 and 1882, Charcot documented many isolated cases of juvenile parkinsonism associated with diplopia, oculogyria, tachypnea, retropulsion, tics and obsessional disorders, which were likely post-encephalitic in origin.

- In 1890 in Italy, following the influenza epidemic of 1889–1890, a severe epidemic of somnolent illnesses (nicknamed the "*La Nona"*) appeared. Almost all of the few Nona survivors developed parkinsonism or other serious sequelae.[63]

Further, encephalitis lethargica assumed its most virulent form between October 1918 and January 1919, which coincide with the second and third and most deadly waves of the Spanish flu.

Many of the survivors of encephalitis lethargica appeared to have recovered completely and returned to their normal lives. However, the majority of survivors subse-

quently developed neurological or psychiatric disorders, often after years or decades of seemingly perfect health. Post-encephalitic syndromes varied widely: sometimes they proceeded rapidly, leading to profound disability or death; sometimes very slowly; sometimes they progressed to a certain point and then stayed at this point for years or decades; and sometimes, following their initial onslaught, they remitted and disappeared. Post-encephalitic parkinsonism was perhaps the most widely recognized of such syndromes.

In encephalitis lethargica there are elevated levels of serum transaminases and LDH, vascular damage throughout the body, and the rapid progression to multiple-organ failure suggest that hypercytokinemia and injury to blood vessels or vascular endothelia may play important roles in the pathogenesis of influenza-associated encephalitis/encephalopathy, rather than the invasion of the virus in the brain. These characteristics are often seen in virus-associated hemophagocytic syndrome or systemic inflammatory response syndrome. Kawasaki disease, which is characterized by systemic inflammatory responses or vascular injury, is often seen in Japan. Japanese people might have genetic backgrounds that facilitate the development of systemic inflammatory responses or hypercytokinemia; they might, therefore, have a tendency to develop influenza-associated encephalitis/encephalopathy.[64]

In a review of 51 case reports of accidental CO poisoning in pregnancy, stillbirth and permanent fetal brain damage associated with hypoxia were consistent with controlled animal studies. The literature suggests that of the 22 cases of Grade 5 exposures, 17 resulted in stillbirth and 5 in fetal brain damage.[65] Infants have who survived the CO intoxication of their mother during pregnancy are plagued with spasticity, athetosis, convulsions, strabismus, cyanosis, areflexia, hydrocephalus, mental retardation.[66]

A correlation between maternal exposure to CO and low birth weight has also been reported. Exposure to higher levels of CO during the last trimester of pregnancy was associated with lower birth weight in babies. The effect on birth weight of increased ambient CO was as large as the effect of the mother smoking a pack of cigarettes per day during pregnancy.[67]

The most common symptoms of CO poisoning in the mild form include headache, dizziness, nausea, vomiting, visual troubles, general malaise, all simulating flu-like symptoms; neuropsychological impairment with deterioration in memory and cognitive functions may be present also at low level of CO inhalation (17-100 ppm). In the moderate poisoning may be present chest pain, tachycardia and tachypnea, rhabdomyolysis (caused by binding of CO to muscle myoglobulin), neuropsychological impairment and altered state of consciousness, anxiety or depression. The

most severe cases may present with seizures, lethargy, coma, cardiogenic and noncardiogenic pulmonary edema and sudden arrhythmic death. The classic appearance with red cherry lips and cyanosis is less frequent. A worsening may be present of pre-existing diseases like chronic broncopulmonary disease (COPD), cardiac diseases or posttraumatic syndrome.[68]

Low-level exposure to carbon monoxide results in impairment of higher cognitive functions. The lower scores on neuropsychological tests indicate dysfunctions in memory, new learning ability, attention and concentration, tracking skills, visuomotor skills, abstract thinking, and visuospatial planning and processing.[69]

CO poisoned patients showed significantly lower cognitive performance than controls on processing speed, mental flexibility, inhibition and working and verbal episodic memories. Patients were more depressed than controls, and suffered more from post-traumatic stress disorder.[70]

Cerebral damage is greatly mediated by a process of demyelination of the cerebral white matter. On autopsy, scattered punctiform hemorrhages and myocardial fibrosis are found throughout the cardiac walls. Delayed neuropsychological impairment, one of the most feared complications, presenting as cognitive or affective disorders, hearing loss, motor disturbances, psychosis or dementia, may be detected in 2-40% of the patients in the following 2-6 weeks. The pathological lesions underlying these symptoms are demyelination, petechiae, edema and necrosis. Hyperintensities, lesions of the basal ganglia and atrophy of hippocampi and pallidus are seen on MRI, this last being the most frequently damaged.[71]

Even though the *globus pallidus* is the commonest site of abnormality in the brain, the effects of CO poisoning remain widespread throughout the brain.[72]

At one point in the nineteen century, the mental disorders associated with CO poisoning was called “the insanity of cooks” because so many cooks were subject to it.[73]

Many have looked for markers of CO toxicity that better reflect brain pathology. Because of the complexity of the numerous systems affected by CO, all acute-phase reactants and proteins associated with inflammatory responses were examined, including chemokines/cytokines and interleukins, growth factors, hormones, and an array of autoantibodies, and were all to be elevated in CO-poisoned patients with no discrimination by severity of exposure, including patients with normal COHb and brief exposure. Early after CO exposure elevations of interleukin-6 concentration and S100B protein, a marker of neuronal damage after brain trauma, occurred in

cerebrospinal fluid but not in serum and predicted the development of delayed encephalopathy associated with CO exposure.[74]

Some researchers have suggested a mechanism on how CO exposure can increase atherosclerosis through oxidative stress that damages the vascular endothelium.[75]

Comparison of CO poisoned and COVID-19 patients:

- Population at greater risk of health effects to CO exposure include aged people with co-morbidities, such as cardiovascular diseases, anemia and other blood disorders, chronic lung
- diseases such as chronic bronchitis, emphysema, and chronic obstructive pulmonary disease.[76]

- Both COVID-19 and the acute and chronic states of CO poisoning resemble an influenza-like illness with chill, shivering, teeth chattering, fever, body aches and hallucinations.

- Many COVID-19 patients first experience dyspnea with a mild cough and great weakness with slight exertion similar to cases of slow CO poisoning.

- Both can have bloody sputum.

- Both conditions are marked by hypoxia.

- Both can experience shakiness and faintness.

- Both can experience nausea, poor appetite, anorexia, vomiting, diarrhea with abdominal pain, dehydration and weight loss.

- Both the COVID-19 and the CO poisoned patients want to lie down while experiencing extreme fatigue and prolonged sleepiness for several days (“I have never slept so much in my life”).

- Both can experience a burning sore throat and nasal discharges.

- Both can experience burning chest pain with pronounced shortness of breath, wheezing, and chest congestion.

- Both can experience violent or persisting headaches.

- Both present with cytokine storms.

- Both develop pneumonia.

- Both can develop into acute respiratory distress syndrome.

- Both characteristically present with constriction and oppression of the chest with palpitation, tachycardia and tachypnea.

- On auscultation both with present with decreased breath sounds and scattered rales and rhonchi.

- Both will present apnea and Cheynes-Stokes breathing.

- Both develop pronounced non-cardiogenic pulmonary and intra-alveolar edema.

- On gross examination in both COVID-19 and CO poisoning, the lungs are edematous and vivid-red (described as carmine red[77] for CO poisoning) with the absence of mucous secretion or hemorrhage.

- Both show ground-glass appearance of the lungs on X-ray, as with SARS and MERS. (“The ground-glass appearance was the most common finding, usually representing the initial manifestation of acute carbon monoxide poisoning. This was observed in 11 cases: 6 cases as the only manifestation.”[78] The ground-glass appearance was the most common roentgen finding of acute carbon monoxide poisoning, usually representing the initial chest manifestation. This lesion presents as a soft, veil-like, homogeneous density occurring predominantly in the peripheral portions of the lung.[79])

- Both can develop into respiratory failure.

- Both can develop into multi-organ failure.

- Both develop vascular blood clots, which can lead to pulmonary embolism, venous sinus thrombosis, ischemic strokes, heart attacks, intestinal necrosis and deep vein thrombosis.

- Both conditions are characterized by arrhythmias, angina, myocarditis, vasculitis, heart infarction, heart scarring and heart failure.

- Both can end up with kidney injury and kidney failure.

- Both conditions are marked by elevated CK and myopathies. In other coronavirus infections, as in CO poisoning, rhabdomyolysis has been reported.

- Both develop gangrenous sores over the sacrum or other parts of contact.

- Both have chilblains or COVID-19 toes.

- Both can develop neuropsychiatric conditions ranging considerably across both the central and peripheral nervous system, including encephalitis, meningitis, hemorrhagic encephalopathy, short and long-term cognitive impairment, headache and dizziness, tinnitus, extreme lethargy, alterations in consciousness, agitation, anxiety, depression, mood swings, confusion, delusions, delirium, insomnia, rapid mood changes, lapses of memory, word hunting while talking, inattention, difficulty concentrating, thinking, and focusing on simple tasks, disorientation, poorly organized movements, ataxia, seizures, eating disorders, smell, taste and vision impairments, polyneuropathies, all types of paresthesia throughout the body, including tingling, pins and needles and vibratory sensation, and nerve pain.

- Both can present with diffuse corticospinal tract signs with enhanced tendon reflexes, ankle clonus and bilateral extensor plantar reflexes (a.k.a. the Babinski's response).

- Both can develop impaired liver function.

- Both develop reflux and heartburn.

- Both have conjunctivitis.

- Both develop hyperthermia at the beginning and hypothermia as there are progressing in the collapse state.

- Both COVID-19 and CO poisoned patients can develop significant metabolic acidosis.

- Both will present with elevated levels of LDH, CRP, ESR, D-dimer, troponin I, liver enzymes, WBC and neutrophils.[80,81,82,83]

- Both COVID-19 and CO poisoned patients develop erythematous rashes and vesicular eruptions, and gangrene in their later stage.

- Pediatric inflammatory multisystem syndrome temporally associated with SARS-CoV-2 (PIMS-TS) and CO poisoning shares many symptoms such as vasculitis, arrhythmias, and cardiomyopathy, elevated cardiac enzymes, conjunctivitis, skin rash, erythema, edema of the hands and feet, vomiting and diarrhea with abdominal pain, low lymphocyte and platelet count, elevated CRP, ferritin, D-dimer, procalcitonin and interleukin-6, and diffuse edema.

Hyperbaric chamber therapy that was found beneficial in CO poisoned patients could potentially be found useful in Covid patients with ARDS.

Repertory

For this monograph, 2,085 new entries have been added to the CR 4.5, which had 280 and now has a total of 2,365 entries, a 745% increase in the number of entries.

Proving

The first proving of Carboneum oxygenisatum was conducted in the summer 2020 with 8 female provers aged from 44 to 72 years old.

All provers knew they were Carboneum oxygenisatum, but all were informed not to read about its materia medica before or during the proving, according to the MMPP proving instructions. No prover knew who the other examiners were. All provers are homeopathic physicians or practitioners, many of them have already participated in several MMPP provings, some of them since 2011. We would like to thank all provers very much for their commitment and also for the inconvenient they temporarily experienced for the welfare of others.

1- The first of these provings was conducted in June 2020 by a 59 years-old female prover (908.HA.1), which took three doses Carboneum oxygenisatum 30C three globules dry on the the first 3 days of the proving. The symptoms lasted for 11 days. All her symptoms are identified as 908.HA.1.

I want to point an interesting curative symptom experienced by 908.HA.1, which was, “Compelled to breath deeply spontaneously in morning when first woke up and throughout the day. I am able to take deeper breaths now than before the remedy. I can expand my chest in all directions, pleasant feeling.”

It was then proved by seven other provers, the last one ending just last week (September 25, 2020). As you can read below she was obliged to antidote it with Bryonia and Arsenicum album.

The other provers were as follows:

2- 908.HA.2: female 72y. Holding the Carboneum oxygenisatum 200C two times in her hand for 15-20 minutes, symptoms occur only during that time

3- 908.HA.3: female 44y. Took only once one granule of Carboneum oxygenisatum 30C. The symptoms lasted a long time for 25 days in July/August 2020

Took only once one granule of Carboneum oxygenisatum 200C in September 2020, the syptoms lasted 17 days.

4- 908.HA.4: female 61y. The proving was started at the holiday resort in Brissago/Switzerland, i.e. after a car journey of about 1200 km over 2 days. Took Carboneum oxygenisatum 30C 3 days in a row, one granule more every day and observed for 4 days after that. Took Carboneum oxygenisatum 200C 3 days in a row, one granule more every day and observed 21 days after that. Very hot weather during proving

5- 908.HA.5: female 61y. Proving done in July 2020. The prover took 3 doses Carboneum oxygenisatum 30C on the first (1 globule), second (2 globules) and forth day (3 globules) of the proving. The symptoms lasted for 8 days.

Beginning of May 2020 she had COVID-19 infection, the symptoms lasted about 2 weeks, at the end Arsenicum album helped a lot. The remaining symptom after the disease was that she could not breath deeply, deep inspiration caused an irritation of cough. This symptoms became much better during the proving with 30C and disappeared later after a dose of Carboneum oxygenisatum 200C.

This provers added, “Two weeks after my recovery we tested positive for COVID-19 antibodies IgG. I am quite sure I was infected via my daughter. We went hiking and I drank from her water bottle. She had a mild form, loss of smell included, and her test for antibodies turned up negative.

In spite having Bryonia symptoms in the beginning, it made no difference in my opinion. I made a lot of hydrotherapy, wet t-shirt, wool sweater and blankets. The whole thing lasted 16 days. Great weakness the whole time. Loss of smell , poor balance when rising in the morning and pressure on chest during breathing appeared on day 5, on day 14 diarrhea. With the diarrhea I nearly fainted. The Arsenicum album helped like a miracle. The loss of smell lasted weeks and weeks.
It was a good experience though.

6- 908.HA.6: female 58y. The prover took Carboneum oxygenisatum 30C several times over a period of 17 days. Then she observed for another 5 days. Then she took a total of 4 doses of 200C ocer e period of 6 days and observed a week after. Often sultry hot weather during proving.

7- 908.HA.7: female 52y. Took Carboneum oxygenisatum 30C on Day 1 and day 3, very hot weather during proving. The reaction started quickly, but after a few days it was over.

8- 908.HA.8: female 57y. Proving done in August and September 2020. Took one globule of Carboneum oxygenisatum 6C on August 15. Only mild symptoms after 6C for 3 to 4 days.

Took two globules of Carboneum oxygenisatum 30C on September 14. Much stronger symptoms, after a week the proving was antidoted with Bryonia and Arsenicum album, symptoms lasted in less degree for some days after that.

Beware that references 36, 72, 85, 100 and 908 are the only ones that refer to an author rather than to an article from the literature. However, because of the great number of reference article cited in this monograph and the way that reference system of Word, reference numbers 36, 72, 85 and 100 can also refer to an article in the literature. There are over 500 references from the general medical literature on carbon monoxide poisoning and these references are easy to identified as their identification numbers are ordered numerically, and you can deposit your cursor on the number and you should be able to read the reference or by clicking on it you can access the reference in the references section. Reference 908 refers as a rule to information summarized from the literature when it is not related to a proving symptom.

General

Genius

Now that we have an excellent proving of Carboneum oxygenisatum with an extensive toxicology, the clinical test is next in order to better know the clinical sphere of this remedy. The main incentive that led to its proving and the completion of this monograph is the resemblance of someone poisoned with carbon monoxide and a person with acute respiratory distress syndrome met in patients with COVID-19.

At this point, the essentials to this remedy is a **chilly person** who tends to be **QUITE TIRED** and **APATHETIC** and desires fresh air and is **better in fresh air**, **takes deep as if from shortness of breath**; is **worse in hot weather**; **worse from exertion**, becoming **physically weaker with exertion** and is **better from rest** and **sleep**.

Many important points of the symptomatology of Carboneum oxygenisatum are found in the pathology of COVID-19 patients, including **a flu-like illness** with **chills**, **rigors**, **fever** and **GREAT EXHAUSTION**, **DIFFICULTY BREATHING**, **pulmonary edema**, **pneumonia** with the **gound-glass appearance of the lungs**, **rales** and **rhonchi, Cheynes-Stokes breathing**, **palpitation**, **tachycardia** and **tachypnea**, **nausea**, **reflux**, **vomiting, impaired liver function, poor appetite**, **diarrhea**, **hyperreflexia**, **chilblains**, **bedsores** and **gangrene**.

Already Carboneum oxygenisatum has begun to bear fruits as with its help it saved at least three residents in a nursing home in France from the last stage of acute respiratory distress syndrome.

The MMPP proving team went to the task and conducted a proving with provers in three continents and two hemispheres. Provers were able to experience subjectively the state of intoxication of carbon monoxide that previously had only been reported by the objective description of the victims of carbon monoxide poisoning.

The fact that at least four of the first nine provers experienced curative changes and that many practitioners have had success with this remedy particularly with COVID-19 patients is significant and is a good omen on how useful this remedy will be in the future.

However, it is unknown at this point how serviceable Carboneum oxygenisatum will be, if at all, in patients presenting with any of the many acute and chronic neurological and psychiatric syndromes that follow carbon monoxide poisoning, such as **parkinsonism**, **chorea**, **Tourette syndrome**, **multiple sclerosis**, **hemiplegia**, **encephalitis lethargica ("silent madness")**, **psychosis** (including **manic-depressive psychosis**) and **obsessive-compulsive disorders**; or in persons presenting with cardiac (i.e., **angina**, **arrhythmia**, **fibrillation**, **myocardial injury**, **heart block**, **heart failure, premature ventricular contractions**), respiratory (i.e., **acute respiratory distress syndrome**, **pulmonary edema**, **stertorous breathing**, **respiratory failure**), circulatory (**thrombotic events**, **polycythemia**, **strokes**), neurological (**peripheral neuropathy**, **cerebral events**), psychiatric (**violent** and **quarrelsome** personality), renal problems (**uremia**, **renal failure,**) and **multi-organ failure**.

Within the next five or so years we should have a much better idea of the sphere of Carboneum oxygenisatum in both acute and chronic cases.

At this early hour we will try to draw its genius despite little clinical confirmations.

The main characteristic that we can draw for Carboneum oxygenisatum is its great state of **APATHY**.

One prover experienced such a state of apathy that she didn't care writing down the symptoms she was experiencing and when she noticed that she was barely breathing she didn't care to even breathe.

Such a state of apathy is commonly met in victims of carbon monoxide poisoning.

The same prover wrote Carbon oxygenisatum is a slow acting, insidious remedy with no discernible characteristic symptom other than its insidious, unremarkable, and forgettable nature that can manifest in any conceivable way with a degree of

apathy that would be *very difficult (if not impossible) to quantify **and** qualify in practice.*

This great state of apathy will manifest itself in a **great lethargy, lack of ambition, will, initiative and spontaneity, lack of expression, mask face, and total indolence and complete indifference to everything**, even **when developing serious symptoms**.

Indifference to his personal appearance and hygiene was marked in the victims of intoxication.

In the one hand, the victims of intoxication **want to sit or lie still**, **staring thoughtless** and **silent** with eyes open all day long, **totally withdrawn**, which is very similar to the state of victim of encephalitis lethargica, as depicted in Oliver Sack's *Awakening*.

On the other hand, the majority of the provers wanted to **sit or lie down with the desire or not to close the eyes**, and **do nothing,** a state of **quietness**, that felt **better by lying down** or **after having slept**.

Much tiredness and exhaustion was experienced by many of the provers.

A prover reported feeling so **exhausted and lazy** that she couldn't imagine being able to move the body.

Another prover felt so tired that had to go to sleep immediately.

This following state of needing to be quiet and wanting to rest greatly resembles Bryonia (which has also been successfully used in COVID-19 patients): Weakness, with the desire to lie down and rest, not work, like having a flu; desires to stay in bed and not move, and feeling better from resting; "I rested in the afternoon, wanted to sit or lie without moving; all the symptoms seem stronger when I get tired or work too much, they get better when I rest. Walking outside (which usually helps to improve my symptoms when sick) does not make me feel better, I would rather prefer to rest."

Feeling better lying down and resting: Later during the day more weak and strong desire to lie down, while resting in bed, felt almost normal.

With this profound state of **APATHY** there existed a significant **prostration of the mind** expressed as **difficulty thinking or concentrating, dullness of the mind, lack of clarity of the mind, absent-minded, thoughts felt slow and imprecise, difficulty recalling words**.

A prover reported that it is very difficult to think with an inability to follow my own thoughts.

Another important characteristic of the remarkable state of mind of this remedy is the tendency to be **disorientated** and **making mistakes regarding time, place and occupation**.

On the physical level we find **lack of coordination** and **awkwardness**.

When not too apathetic Carboneum oxygenisatum will tend to be **easily irritable at trifles**, especially **when exhausted**, even to the point of becoming **quarrelsome**, which is a common characteristic of the confused Alzheimer patient.

Euphoria is another alteration of the mind observed in victims of intoxication.

It was remarkable that only one prover experienced sadness, and none experienced other emotions such as anxieties, depression, irritability or anger, likely because the apathy extinguished all possible emotions, again like in the patients with encephalitis lethargica.

Another characteristic of the victims of carbon monoxide poisoning but not experienced by the provers is its tendency to be **incontinent of urine and feces**.

Carboneum oxygenisatum tends to be **chilly** with the **desire for open**.

Three of the 9 provers were more **sensitive to hot weather** than usual, and which aggravated of all the symptoms.

The main modalities of Carboneum oxygenisatum that were confirmed by the provers were **better rest** and **better in open air**.

Also in both the state of intoxication and in the proving there were **SHORTNESS OF BREATH** and a **desire to take deep breaths**.

Another general modality is of being **worse from exertion**, as the **muscles becoming weaker with exertion**.

Carboneum oxygenisatum may prove useful in patients with **pain, tension, stiffness and restricted motion of the cervical spine with or without headaches** as it was experienced by three of the first nine provers.

Sensation that **the back will break** was met in two provers.

Most of the provers experienced aggravation of their symptoms in **morning on waking**, including **tiredness**, **headaches**, nausea, floaters in the eyes, earache,

blocked nose, dry throat, sore throat, painful and stiff neck, breast pain, low back pain, diarrhea, **muscle cramps**, **difficult waking** and coldness of extremities.

Sleepiness is a marked symptom of this remedy, so is **disturbed sleep**.

Peculiar symptoms of Carboneum oxygenisatum:

Says he is well when he is very sick.

Two of the provers experienced **pain in sites of old injuries**, which may point to its indications in cases of injury.

Two provers experienced **sore throat better by eating**.

Agnosia (its victim have intact senses but are unable to recognize or comprehend certain common objects (i.e., a fork), sounds, faces, etc.)

Apraxia (difficulty to perform simple motor tasks, such as dressing or eating with utensils.

Premature ventricular contractions on exertion

Stoop-shouldered

A very high pulse (100 - 120 BPM) with almost no respiration.

Loss of vision with headaches.

Chilliness after a headache

Desire to clinch the teeth or clenching of the jaws.

Weakness and pain in the heart.

Great weakness with any combination of **irritability**, **headaches, nausea**, **vertigo** and **tinnitus**.

Many of the provers experienced curative symptoms, such as:

One of the provers had sequels from COVID-19 before doing the proving and she reported: Beginning of May 2020 she had COVID-19 infection, the symptoms lasted about 2 weeks, at the end Arsenicum album helped a lot. The remaining symptom after the disease was that **she could not breath deeply**, **deep inspiration caused an irritation of cough**. This symptoms became much better during the proving of Carboneum oxygenistaum 30C in July when she began the proving and disappeared later after a dose of Carboneum oxygenisatum 200C.

Difficult respiration on ascending and on exertion was permanently cured in a prover and which is also keynote of the remedy.

Dry, barking coughs several times a day suddenly without cause since February before the proving, so for 5 months. This cough disappeared during the proving completely. A corona test in May 2020 was negative

Strange, pleasant clarity in the head, feeling of calmness and balance, slow thinking, more sensitive to outside stimuli and altogether more sensitive.

Carboneum oxygenisatum (Carbn-o.)

Introduction

Generalities

Very tired in the morning. Had difficulty just getting out of bed. The whole day exhausted. Nothing made it better. After 6 P.M. a bit better, but still tired. (Day 4).
908.HA.1

Tired when rising in the morning, worse talking, when walking dizzy, unsteady.
de Sonnaville *

She has severe muscle pain, very nauseous, intestinal distress. Smell and taste diminished. Emotionally very unstable. Tired since then, muscle aches, shortness of breath, difficulty breathing. Can hardly do anything, e.g., after going up and down stairs she has to rest for hours. Not able to do any housekeeping. Long COVID-19.
de Sonnaville *

Unusual exhausted on waking. Can hardly keep eyes open. Slept well during night but still felt exhausted on waking. Lasted most of day. Nothing really made it better or worse, just time. Better by dinner time. Felt hungover most of day. (Day 6).
908.HA.1

Suddenly is prostrated worse with trembling and perspiration, especially about 6 or 7 pm. de Sonnaville *

Extreme exhaustion (lying down all day), **resignation** and **great difficulty breathing**. de Sonnaville *

Enormous weakness, and sudden weakness from the sightless exertion.
de Sonnaville *

Post-COVID-19 weakness. Scholtz *

Trembling of the body. 1202 *

Sluggishness, weak and sleepy whole day, better during driving in car, but came back after returning home (Day 6). 908.HA.3

Low energy at 4 p.m., need to lie down (Day 7 and 9). 908.HA.3

Energy very low with hot weather (Day 11). 908.HA.3

Very sluggish and listless at noon (30 degrees Celsius) (Day 5, 30C). 908.HA.4

Very tired and limp in the afternoon (Day 5 and7, 30C). 908.HA.4

Weak and tired all day long (Day 4 and 5, 200C). 908.HA.4

During the whole day I feel extraordinarily tired and exhausted (Day 1). 908.HA.5

Tired and exhausted (Day 1). 908.HA.5

All day strong lassitude. (after 30C). 908.HA.6

During the proving more sensitive to sultry hot weather than usual. After cooling down to 28 degrees Celsius significant improvement of all symptom. (after 30C). 908.HA.6

Exhausted, lazy, not imaginable to move my body (Day 3). 908.HA.7

Easily exhausted from exercise, desire to stop and to go to sleep, too weak to move (Day 4). 908.HA.7

Heaviness in the whole body, very tired (Day 2, C6). 908.HA.8

Body feels heavy. de Sonnaville *

Mild weakness and feeling somehow sick after rising in the morning (Day 3, 30C). 908.HA.8

Later during the day more weak and strong desire to lie down, while resting in bed, I felt almost normal (Day 3, 30C). 908.HA.8

Weak, want to rest, not work, like having a flu, desire to stay in bed and not move. (Day 4, 30C).908.HA.8

I rested in the afternoon, wanted to sit or lie without moving (Day 5, 30C).
908.HA.8

All the symptoms seem stronger when I get tired or work too much, they get better when I rest. Walking outside (which usually helps to improve my symptoms when sick) does not make me feel better, I would rather prefer to rest (Day 7, 30C).
908.HA.8

On waking (waked by wife at 54), a general vague distress, and on getting up was giddy; tendency to fall to the side obliged him to sit down for security. Then distress in precordium, a kind of anguish, with sighing reap, and slight nausea, caused by this precordial distress; then a free fecal evacuation, painless, like a relaxation of bowels which follows fear or excitement (soldiers going into action), with decided urging; rapid action of heart on exertion; pulsations very audible in left ear. Short cough when moving; precordial distress; had to sit while dressing; sensitive to outer air; circulation very much depressed and did not regain its tone for a long time. No inclination to urinate like the urgency to stool. Headache came on after moving around; is a throbbing headache, a headache similar to his usual exhausted headaches. No appetite, could not eat his usually hearty breakfast Mouth thick and pasty; no bad taste; saliva is thick. Hands moist, inclined to be cool. [84] +

She has a distress across the thorax. The distress in precordium so great that she became moist; skin hot (also sensitive to cold air as usual), with nausea. Free evacuation of bowels—no appetite. Weak, could not get up. This distress seemed so bad before she arose—as though she was dying. It stopped menstruation, then 3 days advanced (usually runs 6 or 7 days). The looseness of bowels continued and gradually produced tenesmus—6 passages in 24 hours. Camphor no use. Coffee gave decided temporary relief for 3 hours. [85] +

Extreme emaciation followed the attack. 36 +

The first effect is a stage of complete rest. 36 +

The second stage is one of excitation, characterized by contractions and convulsions. 36 +

Trembling of whole body. 36 +

Body stiff. 36 +

Hemiplegia (persistent). 36 +

Paralysis of the sphincters. 36 +

Carboneum oxygenisatum

32

Sphincters relaxed. 36 +

Cramps. 36 +

Tonic cramps. 36 +

Spasms, without loss of consciousness. 36 +

Spasms, returning every five minutes, with loss of consciousness and loss of speech; the head was drawn spasmodically backwards, the arms stiffly extended; the spasms especially affected the cervical muscles. 36 +

Violent spasms. 36 +

Violent spasms, at first usually clonic, afterwards generally tonic, becoming tetanic. 36 +

Tonic spasm of most of the muscles of trunk and extremities so that it is difficult to make patient sit or lie. 36 +

Repeated convulsions. 36 +

Epileptiform convulsions, which were renewed every time the patient was touched or spoken to, although he lay quite still and apparently unconscious. 36 +

Disinclination to labor. 36 +

Great lassitude in hands and feet. 36 +

Weakness of the muscles. 36 +

Extraordinary weakness. 36 +

General debility and malaise. 36 +

Felt his strength fail him. 36 +

Every voluntary moment, even speaking, difficult. 36 +

Rising and walking seemed a most tremendous exertion. 36 +

In morning could not rise up. 36 +

Prostration. 36 +

Great prostration. 36 +

Complete prostration. 36 +

Inclination to faint. 36 +

Faint feeling. 1202 *

Fainting. 36 +

Suddenly fell to the ground, as if struck by lightning. 36 +

Very restless. 36 +

An enormous restlessness, with anguish and oppression, forced me to get up and walk in another room where the window was open. 36 +

Constant motion, continually flexing and extending the right arm with considerable force. 36 +

The father was constantly moving about and complaining of his head. 36 +

Continually tossing about. 36 +

Four children were lying in bed senseless, tossing to and fro. 36 +

Extraordinary sensitiveness. 36 +

The third stage is one of anesthesia, characterized by partial or absolute insensibility. 36 +

General insensibility. 36 +

Tactile sensibility was greatly diminished; patient evinced no pain when pinched and pricked quite severely. 36 +

Sensibility of sight, hearing, smell, and taste also greatly lessened. 36 +

Blunted sensibility of the whole skin. 36 +

Sensibility of sight, hearing, smell, and taste also greatly lessened, Blunted sensibility of the whole skin. 1202 *

Anesthesia of the skin (persistent). 36 +

Anaesthesia of the skin. 1202 *

34

Anesthesia of skin, but the slightest touch with a hot iron recalls the sensibility. 36 +

The sensibility of the skin is completely lost, especially to mechanical irritation; only a glowing hot iron causes a reaction; this insensibility is at first noticed on the extremities, whence it gradually extends to the trunk, last of all involving the mammary glands and the fossae under the clavicle and in the axilla. 36 +

Complete anesthesia. 36 +

Felt very tired and fatigued. 36 +

General, indefinable malaise; feeling of painful weariness; dull pain in the limbs and loins. 36 +

Dread of every noise or jar, which shoots through the body like an electric shock; continues for several minutes; this conditions gradually changes to a kind of insensibility, which is especially noticed in the tips of the fingers, and it varies in intensity with the condition of the atmosphere. 36 +

Body all sore. 36 +

Whole body sore to touch. 36 +

Soreness of all the muscles, as after excessive fatigue (second day). 36 +

Generalized soreness. 1202 *

Paralysis of the right arm; involuntary evacuations; anasarca of the right arm; over the body various spots, which looked as though they had been burnt, where blisters developed, followed by suppurating ulcers of the whole right arm from the shoulder to the fingers was very much swollen, painful on pressure and motion; the whole right thigh was swollen, especially on the outer side, the skin seemed infiltrated; skin of the right side swollen, not painful; large suppurating sores formed in the right axilla; gangrenous spots on the back of the right hand; sores on the outer side of the right thigh, and on the inner side of both knees, becoming gangrenous. 36 +

Loss of consciousness for a long time; paralysis of the bladder; paralysis of the lower extremities; *bed-sores over the sacrum*; diabetes; abscess in the breast; violent convulsions. 36 +

Found in deep coma, followed by bronchitis, herpes of the lips, and sugar in the urine for three days after the poisoning. 36 +

Found in a comatose condition; afterward pleuro-pneumonia of long duration, and transient diabetes. 36 +

Coma. 1202 *

Convulsions with coma. 1202 * 1202 *

Excessive secretion from with coma. 1202 *

Paralysis with coma. 1202 *

Decorticate rigidity with damage to the corticospinal tract with coma with convulsion. 1202 *

Cardiac irregularities with coma. 1202 *

Coma with moaning. 1202 *

Pupils insensitive with coma. 1202 *

Eyes wild staring and protruding with coma. 1202 *

Left hemiplegia with coma. 1202 *

Respiratory failure with coma, pneumonia with coma. 1202 *

Paralysis of the rectum and bladder; persistent idiocy. 36 +

Stertorous respiration, paralysis of the limbs, paralysis of the bladder, trismus, convulsions, pneumonia on the right side. 36 +

Paralysis of speech; hemoptysis; recovery only after many months. 36 +

Loss of consciousness. 36 +

Surface of the body cold. 36 +

Stertorous respiration. 36 +

Frothing from the mouth. 36 +

After being aroused he complained of headache, difficulty of swallowing, dryness and scraping in the throat, causing cough; nausea; painful pressure in the epigastric region; confusion of the head; coma; chilliness; numbness of the hands and feet. 36 +

36

On the third day, the feeling of paralysis had almost entirely disappeared, the pulse was 120, temperature 39.1. 36 +

The face was pale, covered with copper-red spots of various sizes and forms, which were also found on parts of the trunk and extremities. 36 +

These spots varied in color, were darkest on the abdomen, and on the anterior portion of the left thigh, where they were as large as the palm of the hand. 36 +

Tongue dry, covered with a brownish coat. 36 +

The mucous membrane of the mouth and fauces swollen and injected. 36 +

Pressure on the stomach painful. 36 +

The bladder was distended above the navel. 36 +

For two days the patient passed urine only with great effort, and very seldom. 36 +

There was a short dry cough. 36 +

Respiration 26; on deep inspiration stitches in the lower portion of the right half of the chest. 36 +

Dullness in the lower portion of the right side of the chest; respiratory murmur impaired, with fine rales; through the other portions of the lungs were coarse rales with increased respiratory murmur. 36 +

Urine evacuated with the catheter reddish brown. 36 +

The bladder continued paralyzed; the patient developed pneumonia with infiltration of the right middle and lower portions of the lungs, inflammation of the pharynx and stomach, the vasomotor paralyzed; general loss of power and emaciation; inflammation of the skin; places formed blisters and ulcers, bed-sores; gradual recovery. 36 +

In all the bodies were cold, the limbs were relaxed, pulse imperceptible, pupils dilated, mouth spasmodically closed, respiration irregular and superficial. 36 +

The blood was very dark-colored. 36 +

On the second day, the skin and mucous membrane of the woman became gangrenous, the urine dark brown, after which she died. 36 +

Head, face, and neck livid, and greatly bloated, so as t destroy all recognizable features; the lower lip immensely swollen and turned outwards; the skin discolored and cold, without moisture; extremities quite cold; pulse varying in force and frequency, at times almost imperceptible, the number ranging at different times from 80 to 120; breathing labored, blowing, and irregular; eyelids closed, balls rolled up slant, pupils varying somewhat but generally dilated; entire insensibility to outward impressions, even of the most painful character; no motion of any muscles except those concerned in respiration, which was chiefly diaphragmatic. 36 +

Lower lip would turn outward and swell transiently. Sebastian *

His appearance was that of a calm and tranquil sleep; countenance was of a pale leaden aspect; his lips and ears were livid; respiration inaudible at a short distance, extremely short and suffocative, with intervals of suspension; pulse rapid small, and at times imperceptible; pupils contracted, but the retina was sensible to the impression of light; muscular system relaxed and powerless; he appeared like one whose functions and powers of the system were almost extinguished. 36 +

Face suffused and purple; expression of agonized suffering; the temporal artery distended and prominent; respiration rapid and sonorous, resembling a groan more than snoring; pulse moderately full but frequent; the muscles seemed rigid and contracted; pupils dilated; vomiting. 36 +

If a person be plunged entirely, with the exception of the head, into a bath of carbonic acid gas at the ordinary temperature, there will be experienced a decided sensation of heat over the parts of the body in contact with the gas. 36 +

This sensation of heat is pleasant, and may be compared with that produced by a fine soft garment or padding applied to the skin. 36 +

If the bath be prolonged for a quarter of an hour, the sensation of heat becomes more intense, and accompanied by a peculiar prickling and tingling; in some persons whose skin is very sensitive it reddens, and there is experienced a burning heat. 36 +

The whole surface of the body then transpires freely. 36 +

The urinary secretion is decidedly increased. 36 +

At last, after the gas has remained in contact with the skin for a longer time (some hours), the skin becomes numb, and anesthesia takes place, so that pricking or pinching can be endured without sensation. 36 +

At 9.46, I entered the cabinet containing the gas, and in one minute afterwards experienced a pleasant sensation of heat over all parts of the body. 9.50, the heat had decidedly increased, was especially pronounced in the pit of the stomach and in the internal portions of the limbs, especially of the thighs, associated with an agreeable tingling in the genitals organs. 9.54, the heat had increased and was very difficult to bear; only the feet were somewhat cold. 9.56, experienced along the superior dorsal region slight prickling. 10.1, pulse remained the same; the body was very red and covered with perspiration. 36 +

Extreme heat was felt over all parts of the body, especially in the palms. 36 +

Two other persons were in the lower part of the same cabinet; they experienced different degrees of oppression. 36 +

In one the respiration was very much oppressed, with very great desire to lie down. 10.7, my two companions were obliged to open the window; though I was not so greatly inconvenienced, still the fresh air was very agreeable. 10.16, my pulse had fallen to 60, all my limbs were very supple; the general sensation of health was increased. 36 +

At another time the pulse fell to 52, and there was some pain in the head. 36 +

Another person in the bath described the sensation as of a band above the eyes, and a very severe frontal pain. 36 +

The baths were frequently followed by violent itching over all parts of the body. 36 +

Better warm bathing. de Sonnaville *

After the bath the saliva was acid. 36 +

The capillary circulation was much more active. 36 +

I entered the bath at 5.10. At 5.13, the heat steadily increasing, the current of gas passing over the body causes an irresistible desire to sneeze. 5.15, burning heat between the shoulders. 5.18, respiration 16, pulse unchanged. 5.22, perspiration begins on the face. 5.25, sensation of burning heat very intense between the shoulders, and drops of sweat rolling down over my chest. 5.30, perspiration general. 5.40, pulse full, regular, 76; perspiration profuse. 36 +

If one plunges the head into a bath of Carbonic acid gas, there is immediately experienced heat, especially about the face and eyes. 36 +

The gas getting into the nose causes prickling in the mucous membrane, sneezing, and profuse secretion of mucus. 36 +

He experienced prickling in the eyes, vertigo, thundering in the ears, suffocation, the face became blue, purple, etc. 36 +

Profuse lachrymation, heat of the face, rush of blood to the head. 36 +

He inhaled the gas mixed in various proportions with atmospheric air. 36 +

The secretions are in general increased, perspiration considerable, urinary secretion decidedly increased, and there were frequent efforts to urinate. 36 +

Often the gas causes rush of blood to the hemorrhoids, increased menstruation, epistaxis, and often hemoptysis. 36 +

In the organs of generation it causes great heat and excitement and turgescence. 36 +

In woman increased menstruation. 36 +

Coma, red streaks running along the forearm in the course of the radial nerve, followed by swelling of the subcutaneous tissue especially on the right side, and along the right supraorbital nerve. 36 +

Subsequent development of herpes on the lips and on the forearm, and along the sciatic nerve; development of bedsore. 36 +

After the poisoning there developed zona on the left side of the face, corresponding to the course of the trigeminal nerve. 36 +

Cherry pink discoloration of the body and organs on autopsy suggested carbon monoxide poisoning. Internal examination revealed cherry red discoloration of blood and tissues (see picture); internal organs were congested with obvious **pulmonary and cerebral edema**. [86] +

We observed a large CO poisoning in a homogenous cohort of 38 male recruits. Within the first day the most prevalent symptoms were **headache and dizziness**, followed by **nausea and a sensation of weakness**. **Clumsiness** and **visual disturbances** were already present when the patient woke up from sleep or coma. **Confusion, dizziness, weakness, headache, palpitations and shortness of breath** were noted after a mean latency of 10 to 20 minutes; nausea and **hearing impairment** started within an hour, whereas **vomiting and chest pain** were late symptoms. Individual symptoms also greatly differed in duration. **Shivering** was an

early and quickly reversible, spontaneously reported symptom in two patients. **Shortness of breath, chest pain, vomiting, palpitations and auditory impairment** usually lasted less than 12 hours, whereas **nausea, weakness, dizziness, and clumsiness** generally lasted up to one day. Headaches, visual changes, and a sensation of confusion often persisted much longer. [87] +

In 160 persons in a high school were poisoned with CO: the frequency of the symptoms was: headache 90%, dizziness 82%, weakness 53%, nausea 46%, **trouble thinking** 46%, shortness of breath 40%, trouble with vision 26% and loss of consciousness 6%. [88] +

Early mild intoxication of the 35 staff members tested, 8 (23%) reported feeling light headed and **euphoric**. Only five children raised concerns, all about **conjunctiva injection**. All parents/caretakers of the 41 children were contacted; of these, 10 (24%) reported that the children appeared **happier or more elated** than usual for the rest of the evening. In this case series of 78 people with early acute intoxication with carbon monoxide, **euphoria** was noted to be a relatively common occurrence, a symptom that to our knowledge had not been previously reported. It is interesting to note that it was the exceptional circumstances of this episode of carbon monoxide exposure that prompted the follow up health questionnaire to be performed, and that euphoria was repeatedly reported in both the children and the staff. As mood elevation has not been reported as a symptom of carbon monoxide poisoning, not all the parents were specifically asked about euphoria; the symptom was reported by the parents when they were questioned about any unusual behavior in the children. [89] +

A group suicide was attempted by 3 young people—a 31-year-old man (patient 1), a 21-year-old woman (patient 2), and a 20-year-old man (patient 3)—by burning charcoal in a closed car. At the emergency department, hypotension and hyperthermia were severe in patient 1, moderate in patient 2, and absent in patient 3, although all the patients were comatose. The initial serum lactate levels were 75.1 mg/dL in patient 1, 41.9 mg/dL in patient 2, and 26.3 mg/dL in patient 3, although the carboxyhemoglobin levels were approximately equal in all the patients. Hyperbaric oxygen therapy was immediately initiated and continued for 10 days in all the cases; however, the outcomes of these patients varied considerably. Patient 1 remained comatose and died on day 31 because of central diabetes insipidus after shock. Patient 2 recovered from coma and was discharged; however, she was rehospitalized for delayed effects on day 45 and recovered completely after another 10-day HBOT. Patient 3 gained consciousness and recovered completely with no sequelae during the 1-year follow-up. From these cases, we can consider that the initial blood lactate may correlate with the patient outcomes and prove to be a use-

ful prognostic factor. Thus, we should particularly consider elevated lactate levels in CO poisoning. [90] +

Ms. A. a 23-year-old graduate student awoke from sleep early that morning with a severe headache and shortly afterwards lost consciousness. When she reached the hospital she was awake, but was irritable, nauseated, complained of headache, and on one occasion vomited. Her face was flushed and she was lethargic and unable to answer questions appropriately. The following day, however, she was readmitted because of generalized malaise, anxiety, and blurred vision, some difficulty with recent memory, apathy, fatigue, irritability, and unprovoked crying episodes. [91] +

Mr. B. a 35-year-old postdoctoral lecturer was mildly confused on admission and unable to recall the events leading to his hospitalization. He complained of a moderate frontal headache. Physical examination was normal with the exception of red-mottled skin and left upper-extremity weakness; pressure-induced mono-paresis of the left upper extremity. Following discharge Mr. B complained of **apathy**, **fatigue, irritability**, and disturbed interpersonal relationships. Whereas he had been an accomplished and enthusiastic sailor, he now complained of relative indifference about the sport and noted an impaired ability to make the fine tactical decisions that had previously earned him considerable racing success. His difficulty with problem solving and lower resistance to the stresses of work hampered him professionally. [92] +

A 23-year-old, nonsmoking Phi Beta Kappa, working as an undertaker, moved into a new condominium. She had an 18-month exposure to CO from a faulty clothes dryer hookup and developed symptoms of **extreme fatigue**, **headache**, fever, pharyngeal pain, nausea and diarrhea, flushing, and palpitations. Later she developed a sleep disturbance. These symptoms were diagnosed repeatedly as viral in origin by several physicians, including a neurologist and an endocrinologist. Over time she also manifested weight loss, tinnitus, chest pain, **irritability**, and emotional changes, including **severe depression** and emotional lability. She was unable to do simple computations such as check writing; she also experienced **blurred vision** and **ataxia**. [93] +

A 36-year-old woman (college-educated, a nonsmoker, and employed in research and social work) stayed in her apartment because she was unable to walk adequately after a work-related accident. She began to have muscle spasms of the neck and shoulders, **fatigue**, **chest pain**, nausea, headache, **tinnitus**, photophobia, **decreased short- and long-term memory**, slowed mentation, and **difficulty with calculations**. [94] +

A 19-year-old woman complaints of **headache, vertigo, nausea, and weakness**. She reported a loss of consciousness, lasting between 5 and 10 min and witnessed by her boyfriend, which had occurred in the subway approximately 1 hour after leaving the church. [95] +

A 37-year-old female farmer: blood pressure was 140/100 mmHg, heart rate 96 beats per minute, and respiratory rate 20 per minute. She complained of dizziness, light-headedness, headache, nausea, a pounding heart, and wheezing. [96] +

An interesting report from France about the effect on 35 individuals of working for periods up to three years in a room with leaking gas pipes. It was found that the air had contained from time to time 1 part of carbon monoxide in 1,000 parts of air to 1 in 10,000 parts of air. Twelve of the 35 had **albuminuria** and three had sugar in the urine, but none had any symptoms of heart weakness or of damage to the blood vessels. They complained of **headache in the forehead** or **temples**, less often in the back of the head. Thirty-two out of the 35 had **neuralgic pains in the lower part of the back** or in the **intercostal nerves**, and in some cases there were areas of great tenderness on the skin and sensation of crawling or pricking. There is great difficulty in diagnosing this form of occupational poisoning, because so rarely are the symptoms at all characteristic, usually there is only complaint of headache, palpitation of the heart, breathlessness, general nervousness, and disturbed sleep. Indigestion (especially for solid food), loss of appetite, and acid eructation two or three hours after meals are very common. There may be sleeplessness at night and drowsiness in the daytime. Often this drowsiness is so great as to lead to the taking of stimulants. The French attribute the alcoholism of male cooks to the effect of gas from the stoves, and say that they suffer from **loss of energy** and **great irritability**. 97 +

A 53-year-old woman reported emesis and diarrhea, dehydration, one-sided headache, paresthesia in one arm, tinnitus, palpitations, and thirst. She was **hypotensive**, with a blood pressure of 95/56 mmHg, had a heart rate of 105 beats/min, a body-temperature of 37.7°C, and a peripheral oxygen saturation of 96%. [98] +

A 52-year old woman received an accidental 12-hour exposure to carbon monoxide poisoning, was drowsy and responded appropriately to pain, but was unable to talk. She had **bilateral upgoing toes** and **diffuse hyperreflexia**. Her complexion was **cherry pink**. Her initial hospital course was complicated by a mild acute respiratory distress syndrome. She developed **bilateral pulmonary infiltrates** and transient mild hypoxemia which was vigorously treated. She was discharged from hospital 7 days after the intoxication and her husband and friends felt that her *"sparkle had gone"* but she was otherwise normal mentally. At 14 days post CO intoxication she

became **listless**, **apathetic** and withdrawn and a definite impairment of memory was noted. At 24 days post CO intoxication she became disoriented and had a more severe impairment of memory involving recent and past events. She would **sit and stare for long periods** and *did not seem to know what to do with herself*. She became **clumsy** and had difficulty doing simple tasks such as dancing which previously had given her no trouble. She began to have trouble with simple **arithmetic**, dividing the food up amongst the family members, and playing cards. She was first examined neurologically at 28 days post CO intoxication. She denied any problems with *her memory and lacked insight into her illness*. General examination was normal. Neurological examination revealed **masked facies** with a flat and often inappropriate affect. She was oriented to her name and place, but **she did not know the exact date**. Her responses were slow, she could remember recent events in the news, but did not remember clearly her recent illness. Her parables were concrete and she had trouble with serial 7's. Her general knowledge was patchy and out of keeping with her knowledge prior to her illness. She could follow simple, but not complicated commands. She could read simple sentences, draw, and write normally. There was a questionable weakness of the left arm and leg and mild bilateral **intention tremors** in the arms were noted. There was continued deterioration in her mental status and she became almost helpless in self-care and had to be told what to order for her meals and later required assistance with feeding. She would put inedible objects in her mouth and spill her food tray if left unattended. There was marked perseveration of motor activities. At 35 days post CO intoxication, she had become **incontinent of urine**. She could no longer write her name and there was evidence of an **aphasia**, constructural apraxia and a positive snout and bilateral grasp reflexes were noted. There was some cogwheel rigidity noted in the left arm which was an evanescent sign. Decreased rapid alternating movements were noted in the left arm as well. The reflexes in the legs were brisker than the arms. By 40 days post COI intoxication, she was unable to follow even simple commands and the degree of apraxia in writing and construction as well as the motor perseveration was more marked. After this her condition stabilized and there was no further deterioration. Her first CT scan at 34 days post COI revealed two small, well-defined rounded areas of decreased density approximately at the level of the globus pallidus. There was no enhancement with contrast. No mass effect was associated with these lesions. Mild to moderate diffuse **cerebral atrophy** was present. Clinically, recovery was gradual, but dramatic. By 47 days post COI, noticeable improvement was observed. Her neurological examination was then normal except for abnormalities of higher mental function. She began to write and behave normally after this date. Neuropsychological tests at day 78 post COI revealed mild residual abnormalities suggesting dysfunction of frontal lobes as well

as a right temporal lobe. She was discharged from hospital by day 105 post COI. She was able to care for herself and gradually resumed all her previous activities such as dancing and driving a car, etc. Neuropsychological testing at 9 months post COI revealed some further improvement of mental functioning, with mild difficulties in concentration and tasks requiring rapid decisions. A repeat CT scan 10 months after the initial examination showed mild enlargement of the anterior aspect of the third ventricle. The previously demonstrated lesions at the level of the globus pallidus on both sides could no longer be identified with certainty. Moderate cortical cerebral atrophy was again noted. Localized widening of the third ventricle supported the contention of a localized atrophic phenomenon at that level. When last seen at 11 months post COI, she was functioning well. Her only complaint was she had slowed down somewhat in general. Her neurological examination was normal.
[99] +

Enlargement of the thyroid (38%), loss of hair (45%), **diminution in potency**.
100 +

Localized pain or soreness, and localized edema. 101 +

The course of 90 cases of carbon monoxide poisoning who were taken to hospitals and therefore were carefully observed. All were unconscious when brought to the hospital. They had been poisoned by gas containing for the most part 5 to 10 per cent of carbon monoxide, but in some instances as high as 20 to 30 per cent. In almost **all cases there was fever**, sometimes preceded by a subnormal temperature. The fever lasted from one day to a week; the maximum duration in any non-complicated case was 22 days. In cases which were not fatal the highest temperature, with the exception of one unusual case in which the temperature reached 108°, was 104.8° F; in fatal cases, 107.5. The pulse was r**apid and weak**, but regular. The respiration was rapid, regardless of the condition in the lungs, and was **labored and jerky**, usually 30 to 36 per minute, *and in one case as high as 80*. In the 29 cases in which blood counts were made, an **increase in white cells** was found in all but two. Of these 27 cases, 18 had counts between 18,000 and 44,000.
102 +

The most frequent complications of CO poisoning: Cardiovascular: **Infarction, chest angina**, **ST change**, **arrhythmia**, **cardiac failure**; respiratory: **pulmonary edema**, **lung infection**, **bronchospasm**; Neurological: **convulsion**, **motor paralysis**; others: **gastrointestinal bleeding** and hypothermia. Major functional manifestations are behavioral change, memory disturbance, motor paralysis, Parkinson's disease 103 +

Intoxications of carbon monoxide are frequent and may affect many systems such as the lungs, heart, and brain, leading to coma or death in severe cases. In this case report, we present two adults who were exposed to the same source of carbon monoxide for a nearly equal period of time. The first patient, a 28-yr-old female, developed massive symptoms including loss of consciousness, respiratory insufficiency, and lung complications resulting in **severe lung edema**. She was intubated and ventilated for 43 hours before she recovered and could be extubated. The other patient, a 22-yr-old male, recovered immediately and was fully orientated after applying an oxygen mask at the scene of incident. After admission to the intensive care unit, both patients showed an equally high serum level of COHb and received hyperbaric oxygen therapy. The male patient was discharged from hospital the following day, whereas the female remained in intensive care for 4 days. A satisfacory explanation could not be found for the difference in the clinical progression in these two cases. However, this case report shows that, in spite of almost equal serum levels of COHb, the individual symptoms can vary extremely. [104] +

The typical progression of symptoms of CO poisoning are tightness across forehead, headache and slight flushing, dilatation of cutaneous blood vessels, **severe throbbing headaches at the temples**, **severe headache with nausea and vomiting**, **dyspnea**, **dim vision**, **confusion**, **muscular incoordination**, **syncope**, **tachypnea**, **tachycardia**, **coma**, **Cheynes-Stokes breathing**, and **convulsions**, **cardiovascular collapse**, **respiratory failure**. Pulmonary edema is a fairly common feature that makes routine chest X-ray mandatory in each CO-poisoned patient. Rhabdomyolysis and its consequence, **renal failure**, as well as **pancreatitis**, are also complications that may be seen in CO poisoning. 105 +

Two cases with severe headaches, extreme fatigue, dizziness, nausea, and palpitations. **Frontal headache with fainting**. 106 +

Thirteen major clinical findings were significantly more common in the severe group, viz., abnormalities of nervous system, lungs and skin; **excessive sweating**, **palpable liver**, **localized edema**, **tendency to bleed**, pseudo-recovery, temperature of 102°F or higher, respiratory rate of 30/min or more, l**eukocyte** count of 18, 000/cmm or higher, **albuminuria**, **abnormal urinary sediment**. Nine other major findings had no clear-cut connection with severity, viz., **suffusion of mucous membranes**, abnormalities of blood pressure, **irregular cardiac rhythm**, **localized edema**, pulse rate 110/min or higher, **vomiting**, **headache**, erythrocyte count of 5,200,000/cmm or higher, and glycosuria. Ninety-eight of the 103 patients showed some sign of mental abnormality on admission. Differential leukocyte counts showed that in the severe group the **neutrophil count was increased** to

three times the normal value during the first 24 hours. The eosinophil count was significantly reduced in this group of patients during the same period. 107 +

Neurological manifestations of acute carbon monoxide poisoning include **disorientation**, **confusion** and coma, **cogwheel rigidity**, **opisthotonic posturing**, **extremity flaccidity or spasticity**, and **extensor plantar response**. Perhaps the most insidious effect of carbon monoxide poisoning is the delayed development of neuropsychiatric impairment. Within 1-3 weeks of poisoning, 15-40% of patients will manifest inappropriate **euphoria**, impaired judgment, **poor concentration**, and **relative indifference** to obvious neurological deficits. Computed axial tomography can show in some patients with poor neurological outcome low density in the area of globus pallidus. But this feature is not specific for carbon monoxide intoxication. **Pulmonary edema** is a fairly common feature of CO poisoning. The appearance of pulmonary edema aggravates tissue hypoxia in adding a respiratory hypoxia to the CO-induced oxygen delivery decrease. It makes oxygen administration less active because of the intrapulmonary arteriovenous shunting and decreases CO elimination. 108 +

Nausea and severe throbbing headache feeling dizzy and unwell, and shortly afterwards lost consciousness disorientated in time and space. She was distinctly flushed with cherry-red coloring of skin and mucosae. Radial pulse was 98/ min. 109 +

A positive left Babinski's sign. Blood pressure was 130/60 mmHg, and pulse rate was 160 beats/min and regular, with spontaneous breathing at a rate of 24 breaths/min, and a rectal temperature of 39.3°C (102.7°F). The CK level was 7,000 U/L, and the CK- MB fraction was 2%. The chest radiograph revealed minimal basal pulmonary infiltration. A moderate reduction in global left ventricular function, with an ejection fraction of less than 30% and moderate mitral regurgitation. She had great difficulty lifting her extremities against gravity, indicating proximal muscle weakness. 110 +

Fatigue, nausea, headaches, disorientation, short-term memory loss, acalculia, difficulty with driving and following directions, depression, and emotional lability. Diminished cognitive, visual, spatial, and memory; headaches; functional thought capacity and memory diminished; and anxiety. [111] +

Headache, nausea, dizziness, **weight loss**, **muscle twitches**, and what she called a complete "nerve collapse;" continued muscle weakness and joint pain, **urinary incontinence**, and difficulty remembering things. She had spent time in a local

hospital with breathing difficulties and continued tremors, muscle twitches, headaches, and arm cramps persisted. [112] +

Bad dreams, headaches, dizziness, tinnitus, and arm numbness. In January 1991, he contacted his family physician, complaining of severe vertigo and increased nocturnal blood pressure. Dull pain and twitching in neck and head muscles, increased sensitivity to loud noises, **nervousness**, **irritability**, fatigue, tinnitus, insomnia, dizziness, motivational difficulty, and muscle twitches. [113] +

A 43-year-old woman was in good health when she moved into a condominium in October 1987. During the ensuing months, she noted increasing difficulties with headaches, nausea with vomiting, dizziness, **somnolence**, coughing with shortness of breath, **difficulty maintaining balance**, falling episodes, and **photophobia.** In December 1987 she had difficulty passing the vision portion of her driving test despite experiencing normal vision up to that time. She described her vision as **blurred** or distorted, as though looking through a haze. Because of her complaints of fatigue, a physician did a complete blood count in late December that documented a mild abnormality in the platelet count. She noted increasing difficulties with reading, misreading of words, and **decreased comprehension**, as well as **word-finding impairment**. She had difficulty remembering information and was unable to dial a phone, to perform simple calculations to maintain her checkbook, and to read a digital clock. She tried to cover up deficits that embarrassed her. Inability to remember details. Nausea and vomiting, headache, dizziness, somnolence, shortness of breath, and falling and inability to read and find words. difficulty reading, blurred vision with photophobia, and was unable to perform simple math skills. Periodic nocturia. Tendency to fall. She reported continued problems in remembering where items were stored in her house. Difficulty driving at night. [114] +

Pulse 112, and the respiratory rate 24. She was somnolent, WBC was 12,900 six days later. Left-sided sensorineural hearing loss developed. Mildly decreased attention and rapid index finger tapping. Depression and anxiety. MRI showed lesions in the globi pallidi. [115] +

A 14-month-old girl, granddaughter of patients 1 and 2, was brought to the emergency room breathing oxygen by mask. Palpated systolic blood pressure was 90 mmHg and stable, pulse 164, respiratory rate 44, and temperature 101.9°F. Single generalized tonic-clonic seizure, MRI showed bilateral parieto-occipital periventricular lesions. [116] +

A 3-year-old boy in a coma, pulse late 12O/min, respiratory rate 36/min. Deep tendon reflexes were increased in the extremities with pathological reflexes. During

the first one hour of admission, he had a transient coarse tremor and generalized convulsion. WBC 28,000/cmm, SGOT 58 KU, LDH 1,242 WU. He had mild weakness of the extremities with symmetric hyperreflexia and the positive Babinski's sign bilaterally. He was found to have absent light perception with intact pupils and normal ocular fundi, suggesting cortical blindness. Cortical blindness did not improve throughout the period of hospitalization and for the following 3 months subsequent to his discharge. Spastic quadriparesis completely improved during the period of hospitalization. [117] +

He was comatose with labored and irregular respirations and unresponsive to painful stimuli. He complained of tinnitus and numbness and weakness of the left upper and lower extremities. There was a hearing loss of 40db at all cycles except for 60 db at 6000 and 8000 cycles on the left. The right ear had a loss of 40 db from 250 through 2000 cycles and 10db from 3000 through 8000 cycles. There was weakness at the left elbow and wrist as well as of grasp. There was weakness of the quadriceps femoris, hamstring and tibialis anterior muscles with a foot drop on the left. The deep tendon reflexes in the left upper and lower extremities were hypoactive. Decreased sensation to touch and pain were noted over the fourth and fifth fingers on the left. The hearing loss, tinnitus and weakness in the left upper and lower extremities gradually improved during the hospital stay. However, atrophy of the left quadriceps femoris and hamstring muscles developed three months after admission, showed *fibrillations at rest* and a remarkable diminution of neuromuscular units in the left quadriceps femoris and tibialis muscles. Ten months later, he still had tinnitus and hearing difficulty. Slight weakness persisted on extension and flexion of the left knee and on dorsiflexion of the left foot and toes. There was persistent atrophy of the left quadriceps femoris and hamstring muscles. [118] +

Deeply unconscious with moderately dilated pupils and no reaction to light; tendon reflexes extremely brisk and equal; both plantars gave an extensor response. The patient regained consciousness within 12 hours and the neurological signs returned to normal except that he remained 'stone deaf.' [119] +

K. S. was a 58-year-old woman who was rescued from an apartment fire. Chest X-ray revealed a perihilar infiltrate. On the second post-burn day polyuria developed with output ranging from 280 to 900 cc/hr. Decreasing pulmonary capillary wedge pressure required increasing amounts of fluid to maintain left heart filling. The serum sodium level increased from 137 to 149 mEq/dl over a 6-hour period. Urine and serum osmolarity were 112 and 319 mOsm/kg, respectively. A diagnosis of **diabetes insipidus** was made. The patient remained comatose and showed wide fluctuations of blood pressure, pulse rate, and temperature over the next 3 days, and suffered a cardiac arrest and died on the sixth post-burn day. Serum creatinine

phosphokinase was elevated to a maximum of 4,068 units/liter but the MB isoenzyme was absent. Postmortem examination revealed **diffuse soft degeneration of the brain**. Microscopic sections showed pyknotic nuclei and shrunken eosinophilic cytoplasm in neurons in the cerebral and cerebellar cortex. The pituitary showed autolysis without a cellular filtrate. There were no vascular thrombi seen. The changes were those of severe anoxic brain damage with subsequent edema and 'respirator brain.' [120] +

Fetal death due to acute carbon monoxide poisoning is rarely reported in the medical literature. Of the eight cases found in literature review, only one documented the fetal carboxyhemoglobin concentration. This paper reports a fetal death due to accidental nonlethal maternal carbon monoxide intoxication in which both maternal and fetal carboxyhemoglobin concentrations were obtained. The corrected carboxyhemoglobin concentration was 61% at the time of death in utero, while the maternal carboxyhemoglobin was measured at 7% after one hour of supplemental oxygen. [121] +

A 24-year old male non-smoker had complained of throbbing frontal headache. He was subsequently found unrousable tachypneic, febrile (temperature 39°C), and had a tachycardia of 160/min. **Tense bullae** were noted over the dorsal surfaces of both feet. Early right sided papilledema. Plantar responses were extensor bilaterally. **Neutrophilia** of 18.9 x 10/9 and a thrombocytopenia of 89 x 10 /1. He died and on autopsy there were a total loss of myelin in areas of the white matter. [122] +

A 2 ½ month-old infant girl. On admission, opisthotonus, high-pitched cry, lethargy, and poor visual tracking. The infant improved dramatically during the three days of hospitalization, but after discharge she developed irritability, poor feeding, and weight loss. She continued to deteriorate and was taken to her local emergency department because of severe lethargy and apnea. Admission physical examination revealed a pale infant with a high-pitched cry. A pulse of 190 beats per minute, respiratory rate of 30 breaths per minute, and blood pressure of 80/60 mm Hg [low blood pressure for a two year old girl]. Neurologic examination revealed an obtunded infant with bilateral ptosis and absence of pupillary, corneal, gag, and oculocephalic reflexes. Hyperactive deep tendon reflexes bilaterally. Prolonged apnea She subsequently developed severe increased intracranial pressure, temperature instability and diabetes insipidus developed. [123] +

A 50 year old male was on the floor, doubled up, incoherent and delirious. He was unable to move his right arm, and both legs were very "hard" to the touch to a point above the midthigh. For the next four weeks he spent much of his time in bed. He had lost about 18 pounds and was slowly regaining it. He developed severe burn-

ing pain along the heel and outer edge of the right foot. This pain was continuous. There were occasional episodes of severe pruritus on the plantar surface of the right foot. There were frequent episodes of sharp, shooting pain in the medial and lateral aspects of the right leg. The left leg had had burning pain since he first regained consciousness, and shooting pains and itching had been present in the left lower extremity from the time of his recovery of consciousness. He complained of two areas of numbness, one just above the knee and another along the lateral aspect of the ankle and foot on the left. He stated that originally the areas had been larger than they were at present. Both legs were noted to swell when he was on his feet. The left arm had been perfectly all right until two weeks prior to his admission, at which time the fourth and fifth digits began to feel numb and there were some burning sensations in this area. He had no complaints referable to the right arm. Sleeping was difficult because of the pain. The patient noticed no deficiency in mental acuity or memory. A sensory examination disclosed an area of hypesthesia on the anterior aspect of the left leg corresponding to the distribution of the left lateral femoral cutaneous nerve. There were also hypesthesia, hypalgesia and impairment of other sensory modalities, with mild motor weakness in the distribution of the left ulnar nerve. There was sensory impairment of all modalities in the distribution of the area supplied by the trunk of the sciatic nerve. These changes went up to the midcalf region in the posterior lateral aspect of the left leg. moderately depressed and some- what hostile to the physicians and other personnel. He felt that the onset of cold weather had caused some mild recurrence of sweating in his feet, and also noted that his feet were sweating more than they had before. He also complained of feeling generally tired and of fatiguing more easily. There was occasional cramping in both hands. [124] +

Four members of a non-English-speaking family presented to a university hospital emergency department, All complaining of headache, lethargy, abdominal cramping, and nausea. Two patients had mild orthostatic blood pressure changes. Several hours later, four other members of the same family presented to the emergency department with similar complaints. Several hours later, four more members of the same family presented to the emergency department with similar complaints. One patient had not ingested the suspected milk, but she suffered from migraineheadache and thought that this was one of her headaches. [125] +

A 35-yr-old woman who underwent accidental CO poisoning; deep coma, hypotensive, cyanotic, and hypoxic; discharged few weeks later with discrete paresis of **peripheral nerves**, discrete ischemic brain lesions on computed tomography (CT) scan, and moderately abnormal electroencephalogram (EEG) without cognitive disturbances. [126] +

A 17-year-old, healthy female adolescent (gravida 1, para 0) at 37 weeks gestational age, headache, nausea and vomiting, chest pain, obtunded, pulse rate of 110 beats per minute, blood pressureof100/60mm Hg, and respirations of 24/min. Fetal pulse rate was 170/m. Uterine contractions five weeks following HBO treatment and was delivered of a healthy 3600-g female newborn with Apgar scores of 9 at one minute and 10 at five minutes. [127] +

Subacute carbon monoxide poisoning: *another great imitator*: Findings in these four patients demonstrate the *protean manifestations* with which both acute and chronic carbon monoxide poisoning can appear. A subject with any degree of coronary arteriosclerosis may be placed in a precarious situation when exposed to carbon monoxide. The heart is called on to increase its work to provide more oxygen for the peripheral tissues, while its own supply has been reduced. It is no wonder that carbon monoxide poisoning can lead to **angina, arrhythmias, ECG changes, and myocardial damage** in patients with underlying cardiovascular disease. The carbon monoxide poisoning probably contributed to the myocardial infarction and **pulmonary emboli** seen in these patients. Vague flu-like illnesses should raise the suspicion of carbon monoxide poisoning. CASE 1—A 67-year-old sought medical evaluation after three days of persistent light-headedness with vertigo, brief stabbing anterior chest pain that worsened with deep inspiration, a dry cough, chills, and a mild headache. On admission to the hospital, he was found to have decreased pinprick and vibratory sensation bilaterally in his hands and feet. Postural hypotension was demonstrated with a blood pressure in a supine position of 140/84 mm Hg and a pulse rate of 70 beats per minute and erect readings of 122/76 mm Hg and 90 beats per minute. His laboratory data included a potassium level of 2.4 mEq/L. An ECG showed first-degree atrioventricular block with nonspecific ST-segment changes. His symptoms were explained with a diagnosis of viral syndrome, hypokalemia of unclear origin. Ten days after discharge he was seen in the emergency room with true vertigo, palpitations, and nausea. Four days later he returned to the emergency room after development of rectal urgency and an explosive incontinent diarrheal stool, followed by a severe crushing anterior chest pain. With the pain he collapsed to the floor; he became diaphoretic and dyspneic. Pulse rate, 112 beats per minute; and respirations, 28/min. He had several flame-shaped and **round hemorrhages** present on both fundi. His hearing seemed grossly intact, though he reported it to be decreased. His chest was clear; his cardiac examination was marked by very soft heart sounds and an audible S4 gallop. He was admitted to the coronary care unit with a diagnosis of **acute myocardial infarction**. The patient had a persistent *tachycardia and dry cough related to inspiration*. This led to a V/Q lung scan that showed findings consistent with **multiple pulmonary emboli.** CASE 2.—A 69-year-old man came to the emergency room after awakening two

days earlier with confusion, nausea, and vomiting. He then passed out and awoke the next day in the bathroom. He crawled to the living room, where he again passed out for an undetermined amount of time, awoke to open his door for fresh air, and then went to bed. He later experienced auditory and visual hallucinations and phoned his neighbor for help because of his extreme weakness. He had shortness of breath but denied chest pain. Pulse rate, 95 beats per minute; and respirations, 40/min. Cardiac examination disclosed an S4 gallop. There were a few dry rales at the base of the left lung and decreased breath sounds over the right upper lung field. His abdomen was distended, and he had decreased bowel sounds. During the night he became confused and *combative*. A chest x-ray film showed a huge bulla in the right upper lobe, slight cardiomegaly, and scattered fibrotic markings. An abdominal x-ray film was consistent with a *mild paralytic ileus*. He was diagnosed with acute inferior mycardial infarction with secondary **mild congestive heart failure** and chronic obstructive pulmonary disease. When the patient's sister and daughter- in-law slept in the same trailer home they woke up early the next morning with throbbing headaches, vomiting, and vertigo. [128] +

A 75-year-old man suffered acute CO poisoning when his car was inadvertently left running in the enclosed garage attached to his home. Two weeks later, he became comatose. When he emerged from the coma, he was almost completely **aphasic**, showed personality changes, and required gastrostomy tube feedings. His neurologic abnormalities persisted for 3.5 months, at which time he was referred for HBOT. His SPECT scan showed diffuse cerebral deficits, especially in the frontal lobe, and also involvement of the basal ganglia. After about 80 sessions of HBOT at 1.5 ATA, his cognitive and social functioning improved substantially, and he could speak coherently. [129] +

A 39-year-old auto mechanic apathetic and confused, with a burn on one shoulder and unable to relate what had happened. After about 10 days his wife found him to be increasingly **absent-minded**, slow in movements and sometimes behaving oddly, e.g., *singing in a strange way and with violent outbursts*. He also began to neglect his personal hygiene. One month after the acute incident he was admitted to the Department of Neurology with tentative diagnoses of tumor cerebri, subdural hematoma or encephalitis. On admission he was well oriented to time and place, answered slowly and with some delay but there were no other abnormal findings in his clinical status. The cerebrospinal fluid, virus tests and CT scan were normal but the EEG showed a generalized slow-wave pattern. After four days his condition deteriorated; he **talked incoherently**, had hallucinations, cogwheel rigidity in his arms and became **incontinent of urine and feces**. He could not walk or eat without assistance and seemed to have impaired vision in spite of normal ocular findings, as

he only avoided colliding with chairs and doors at the last moment. After six weeks in hospital the patient began to improve and continued to make good progress until he was able to walk and eat without assistance, and regained sphincter control. He answered simple questions but was still disoriented. After three months the EEG showed a normal pattern. He was discharged to continue daily rehabilitation. He learned to carry out some uncomplicated household chores but after one year was still very handicapped as regards cognitive functions and had marked impairment of short-term memory. [130] +

A review of the data for the four cases revealed four primary findings: there was no clear relationship between the level of carboxyhemoglobin levels at admission and chronic neuropsychological deficits, all four cases demonstrated declines in estimated intelligence, there were similar declines in memory functioning for the two brothers, but not for the brother/sister, and all four cases experienced consistent and significant late-onset emotional behavioral difficulties. [131] +

The occurrence of **fever** has already been spoken of. It usually comes on during the period of **unconsciousness** and may last till consciousness returns or even longer, while in some cases it appears first several days after the accident. Sometimes the rise of temperature is **very high**, especially if there are convulsions, and this is always an extremely grave sign. The highest thus far noted was in a case of Apfelbach's, which reached 110° F. just before death. 132 +

An unconscious woman in her 70s, a rectal temperature of 29°C. her blood pressure was 170/70 mm Hg; white cell count 16,000, platelet count 311, a left femoral artery embolism (Autopsy of her dead husband showed pulmonary changes consistent with bronchopneumonia.). [133] +

Hypochromic anemia. 134 +

No motion of any muscles except those concerned in respiration, which was chiefly diaphragmatic. 36 +

His appearance was that of a calm and tranquil sleep; countenance was of a pale leaden aspect. 36 +

He appeared like one whose functions and powers of the system were almost extinguished. 36 +

Extraordinary weakness. 36 +

General debility and malaise. 36 +

Felt his strength failed him. 36 +

Every voluntary moment, **even speaking, difficult**. 36 +

Inclination to faint. 36 +

Inclination to faint. 36 +

Sensibility of sight, hearing, smell, and taste also greatly lessened. 36 +

Body all sore. 36 +

Whole body sore to touch. 36 +

Soreness of all the muscles, as after excessive fatigue. 36 +

'Spaced-out', headaches, his handwriting to be irregular during paperwork. Eventually, he became tremulous and nauseated. Drowsy, and reports momentarily losing consciousness during this period. Subsequently, his condition improved, although he had persistent tiredness, headache and gastrointestinal upset over the following 2 weeks. 135 +

Leucocyte count of l8,000/cc or higher. 136 +

Increase in blood sugar, glycosuria. 137 +

It has been shown repeatedly in animal experiments that coal gas causes relaxation of the blood vessels through a sudden weakening of the muscular fibers. This causes slowing of the circulation and the collection of blood in the smaller vessels, and then degenerative changes, especially fatty, in the walls of the arteries. **Hemorrhages into the skin from rupture of vessels on the surface of the body** are not at all unusual, causing **red blotches on the skin and purple spots on the lips and the mucous lining of the mouth**. 138 +

For five days before she had headaches and felt flu-like before becoming stuporous, superficial retinal hemorrhages. 139 +

Sixty-eight men, all steel workers who had either been recently gassed or subject to frequent gassings, were selected, and the blood examined for hemoglobin and red and white cell counts. They found that there was an **increased number of red cells** which ran from 5,500,000 to 9,676.000, more than half, or 66 per cent, being over 6,000,000, but no immature red cells were found. The hemoglobin ran from 95 to 125 per cent, more of them being over than under 100 per cent. No abnormality was found in the white corpuscles. 140 +

The men who had suffered from acute gas poisoning, in describing their experience, said that usually they noticed first a severe headachy dizziness, ringing in the ears, weakness of the legs, nausea and vomiting, and in some cases loss of consciousness. After regaining consciousness a severe frontal headache would persist for from a few hours to a few days. This shows that only 15 per cent of the men had red-cell counts much above the average (between 5,600-6,000/cml) and only 2.2 per cent had counts which would be called strikingly high (over 6,000/cml). The white-cell count showed nothing of interest, nor did the urine analysis or blood-pressure estimations. 141+

What he says is that anemia of profound degree, resembling pernicious anemia in type, may be found as a result of continual breathing of carbon monoxide in coal gas, such as he saw in a woman who did pressing in a tailor shop and in whom this seemed to be the cause. Profound anemia with a red-blood-cell count as low as 1,500,000 as a result of chronic poisoning. On the other hand, there are some observations of a high red-cell count with low color index; that is, a low percentage of hemoglobin. One of these was a man was employed at a gas factory, working at years on a 12-hour shift and exposed to a good deal of gas. After nine months he began to suffer from a sense of weariness and weakness, left-sided headache, **loss of appetite**, *alternating with ravenous appetite*, and **pains in the abdomen**. No carbon monoxide was shown by the spectroscope, but a count of the red cells gave 9,500,000, but only 76 per cent hemoglobin, and later on 11,200,000 with 90 per cent hemoglobin. Another similar case: this man was working for certain periods every day over a burning soldering machine, during which time it was possible to inhale carbon-monoxide fumes. His symptoms pointed to profound anemia. Red-cell count of about 7,000,000. 142 +

Continually dropping levels of hemoglobin in an intubated COVID-19 patient. 908 *

Hypertension in a post-COVID-19. 908 *

A comparison was made of the muscular strength of the hand, using the ordinary hand dynamometer, and, as shown below, it appeared that *the steel workers exposed to carbon monoxide had decidedly less muscular strength than working men of the same age not so exposed*. 143 +

Pneumonia is a very important aftereffect. Cases of post-gassing pneumonia usually develop one to three days after the accident and may be ushered in by a **hemorrhage from the lungs**. The characteristics of this form of pneumonia are that it is lobar in type, caused by the pneumococci, **the right lung is mostly affected**, and

the disease develops rapidly. The temperature is not high, but the pulse is disproportionately **rapid and thready**, **the heart dilates**, and if death occurs it is early in the course of the disease, on the third or fourth day. 144 +

The effect of saturating some of the red corpuscles with carbon monoxide instead of oxygen is to cause a number of fresh corpuscles to be thrown into the circulation in an effort to keep up a normal supply of oxygen. This means that the red-blood-cell count may be high in a man who is continually exposed to carbon monoxide. Blood counts of 6,390,000 per cubic millimeter (the normal number is five to five and one-half million). A stoker in a gas was suffering severely from loss of motor power, pain in his abdomen and in the left side of the head, loss of appetite, and weakness. Three counts of the blood showed, respectively 8,200,000, 9,500,000, and 11,200,000 red cells, but the hemoglobin was only 62, 76, and 90 per cent. 145 +

The 29-year-old woman was disoriented and stuporous at the local hospital, approximately 1 hour after evacuation. Her blood pressure was 85/40 mm Hg, and her pulse rate was 120 beats/min and regular, with spontaneous breathing at a rate of 26 breaths/min and a rectal temperature of 35.7°C (96.3°F). The chest radiograph showed **diffuse bilateral pulmonary congestion**. Anuric, and jugular vein congestion. The creatine kinase level was 2,000 U/L, with a CK-MB fraction of 3%. left ventricular ejection fraction of 15%, right ventricular dysfunction, and moderate mitral valve insufficiency. 146 +

The patient, a 34-year-old male, was found unresponsive and apneic by emergency medical personnel in a burning house. The patient under investigation had carbon monoxide poisoning, and developed **acute respiratory distress syndrome** and **cardiovascular collapse** following smoke inhalation. Anterior–posterior chest radiograph revealed **bilateral infiltrates**. Chest CT demonstrated **diffuse lung disease with dense posterior (dependent) consolidation of both lower lobes and patchy bilateral upper lobe air space disease**. Shortly after arrival, cardiovascular collapse ensued. [147] +

Gangrene following exposure to carbon monoxide has been reported in 5 cases. A further case is described here. A young man, aged 22, who had previously been fit. He was severely gassed and developed gangrene of both legs, requiring amputation. In the case described by Briggs fingers of both hands required amputation, and here also there was no evidence of any disease predisposing to gangrene. In 2 cases gangrene was due partly to anoxemia resulting from the inhalation of carbon monoxide and partly to impairment of the blood-supply by the pressure of the inert body at those points where it was supported. Owing to the positions in which they

were lying, gangrene affected the right side of the neck, occiput, and scapula in Alberti's patient, and the right heel in Lavastine and Alajouanine's patient. In 2 cases, gangrene was due to the combined effects of anoxarmia produced by carbon monoxide and of severe impairment of the blood-supply caused by arteriosclerosis. In the case described by Enzer and Spilberg (1946) there were no symptoms referable to an impaired circulation before asphyxia developed, but in the present case angina cruris had been a severe symptom and the carbon-monoxide gassing precipitated an event which might ultimately have been inevitable. The case in question: He was found unconscious and **cyanosed**. His breathing was **stertorous**. Right foot was cold and blue and his right dorsalis pedis artery just palpable. His temperature was 99°F, pulse-rate 80, and blood-pressure 150/80 mm Hg. Gangrene of the right foot and lower leg developed, and amputation through the lower thigh was done nine days after the patient's exposure to carbon monoxide. There was terminal **blood-clot** loosely attached to the endothelium at the lower end of the popliteal artery. [148] +

Acute stroke-like syndromes. 149 +

A 50-year-old policeman subjected to chronic CO poisoning, highly nervous and **irritable**, increasing fatigability, **tremor** and **sweating**. He experienced only occasional headaches. He had lost 6.8 kg. (15 pounds) in weight—from 72.6 to 65.8 kg. (160 to 145 pounds)—during the month before admission. He **lost his appetite** and frequently felt nauseated. His personality had once again deteriorated markedly, and he was extremely irritable, dizzy and quite **ataxic**, **stumbling** wildly about. On lying down he felt better, and within 2 hours he was "normal" again. At night he experienced insomnia. Excessively **good-humored**, memory lapses and mental dullness; multiplication and division were performed well, but *subtraction poorly*. His speech was slightly slurred, but he was not aphasic. There was moderate **nystagmus** on lateral gaze to the left or right. The *gait was broad-based*, and there was a fine, rapid tremor of the fingers. Two-point discrimination was impaired in the right hand and arm, **staggering gait**, confusion and irritability. [150] +

A 61-year-old white male, **clumsy gait**, **numbness in the extremities**, fatigue, generalized weakness, and **nervousness**. On neurologic exam he was diffusely **hyporeflexic** and had decreased vibratory sensation distal to the ankles bilaterally. Slight tremulousness was noted on finger-to- nose testing. Constant numbness in the feet, hands, and lips; episodes of **loss of balance**; slurred speech; **inability to adequately express his thoughts**; and poorly characterized forgetfulness. He complained of being generally weak but no focal weakness was noted. He described himself as nervous and **excitable** and said he frequently had **trouble sleeping**. [151] +

Patients in the following series present **changes in weight**, dizzy spells, **trembling hands**, memory changes, **alterations in sleep patterns**, and headaches. In addition, patients were often viewed as having become much more **emotionally labile** and **irritable**. On neuropsychological testing, they all showed motor slowing and memory problems. Many seemed to lack social skills that would facilitate their treatment; for example, a number of individuals **argued** with the staff about the course of their treatment and were considered problem patients. [152] +

Pain in the calves of the legs, loss of power affecting especially the extensor muscles, tenderness on pressure, and sensations of crawling and pricking. [153] +

Unusual symptoms: Excessive salivation; bleeding from the gums and throat; In the lungs all sorts of inflammatory conditions may be found; **bleeding stroke**; Among the more unusual symptoms which have been attributed to carbon-monoxide poisoning are paralysis of some of the muscles of the eyes, leading to the well-known **miners' nystagmus**. Affections of the optic nerve, leading to partial or total blindness, have occurred after severe gassing, and so have affections of the nerve of hearing. 154 +

Among the nervous after effects of severe gassing paralyses of all kinds, but most often of the legs, are found. Such paralysis may come *on* at the time of the accident or develop some days later. Almost all the motor nerves have been involved in different cases, and besides this form of paralysis there may be injury to the vasomotor nerves or to the optic nerve. Two cases of this kind have been described as cases of **multiple sclerosis** due to repeated inhalation of carbon monoxide by men working over hot-water heaters with leakage of gas. The first had an acute attack of gassing with a terrible headache, dizziness, vomiting for several hours, bluish lips, and **unsteady walk**. Four days later he complained of numbness in the legs and difficulty in walking. Two weeks later he had double vision. All this time he continued working at the leaky furnace. His condition grew progressively worse, and at the end of two years he was unable to walk, there was atrophy of the muscles of the back, legs and arms, there were wide areas of anesthesia, and his sight was failing; and his mind was unimpaired, but he had lost the sense of position of his limbs. He died six years later, *emaciated and helpless from muscular contractures*. 155 +

The second case, also of a man exposed for some time to gas from a leaky furnace, began in very much the same way, only the symptoms were even severer, for he was delirious, with rapid, labored respiration, wide pupils not reacting to the light, and for several minutes clonic convulsions. There was loss of strength in the right hand, loss of sensation, and diminished vision, especially in the right eye.

These acute symptoms cleared up and, though his vision was impaired, he went back to work. He quickly became much worse, **lost strength and weight**, his gait was spastic, he could not coordinate his muscles, and he had mental depression, loss of memory, and some defect of speech. Finally he could not stand alone or lift a foot from the floor, or feed himself. After 16 months in the country, he recovered the use of his limbs, but typical symptoms of disseminated sclerosis, **an intention tremor**, a loss of sense of position, and **atrophy of muscles developed**. 156 +

A man who recovers from a fairly severe gassing has a throbbing headache, a sensation of constriction in his temples, *a feeling as if his head would split*, he is dizzy, his legs and sometimes his arms, back and neck feel powerless, and he may even not be able to hold up his head. He is numb, and there are instances in which a man who has been burned while unconscious does not feel the pain of the burn after he regains consciousness. He shivers and cannot get warm, he has a feeling of oppression in the chest and palpitation of the heart, and if he attempts to walk home he may bring on a cardiac weakness. 157 +

Disorientated for time and place with marked memory impairment and a **mask-like facies**. There were no extrapyramidal signs. **Generalized brisk reflexes** were present. Computed tomography showed bilateral symmetrical low-density areas in the globus pallidus. One year later he again underwent scanning owing to persistent poor memory and inability to concentrate. CT showed a marked diminution in the degree of low density in the globus pallidus compared with the original scan. 158 +

After having fainted he felt nausea, headache, diffuse substernal chest pressure, generalized weakness, and paresthesia of bilateral shoulders upon waking. 159 +

85 patients conscious but with abnormal neurological signs (hypertonia, hyperreflexia, extensor plantar responses). 160 +

3,000 cases have been reported. In a certain percentage of cases, varying with the author, neurological sequels such as **parkinsonism**, **chorea**, **hemiplegia**, **cortical blindness**, **agnosia**, **apraxia, amnesia syndromes**, **peripheral neuropathies** and **convulsive seizures** are reported. 161 +

Central **diabetes insipidus** is an extremely rare complication of carbon monoxide poisoning we report the case of a young lady, who developed this complication and severe **hypernatremia** after accidental carbon monoxide poisoning. Her serum sodium increased to 205 mEq/L (N: 135-145 meq/L). The urine output was 6,400 in the past 24 hours. At this point, the patient had signs of volume depletion; she had

a dry tongue and mucous membranes, heart rate of 110/minute and a blood pressure of 90/40 mm Hg. In the morning, the next day, a random blood sugar level of 1399 mg/dL was reported. This was managed with an insulin infusion. The blood sugar normalized on the same day, however, the polyuria persisted. [162] +

We report on a woman and her daughter who were noted to have polyuria and hypernatremia after exposure to carbon monoxide. Stuporous, phosphate 3.0 mg/dl, sodium 141 mEq/l, and potassium 3.8 mEq/l. The white blood cell count was 20,700/ mm3. On the 2nd hospital day, the urine output increased to 6,400 ml/day, and the fluid balance was negative at 4,782 ml. At the same time, the patient became comatose and with no response to pain. On the 3rd hospital day, the serum sodium level was 159 mEq/l; serum osmolarity 320 mosm/kg, urine osmolarity 53 mosm/kg, urine sodium was less than 10 mEq/l, and urine specific gravity 1.000. On the 4th hospital day, serum sodium rose to 180 mEq/l. A nephrologist was consulted, and the diagnosis of diabetes insipidus with severe hypernatremia was made. [163] +

Respiratory insufficiency and his body emitted an odor of smoke. Elevated white blood cell count and levels of troponin I, creatine kinase-MB. loss of vision (i.e. right temporal hemianopsia). MR imaging of the brain on day 4 showed diffusion restriction in the left occipital lobe. The loss of vision resolved in three weeks after the carbon monoxide exposure. [164] +

Notable neuropsychologic symptoms persist in a 9-year-old girl one year after an acute exposure to CO. Perceptual deficits, personality alterations, and computerized tomographic evidence of leukoencephalopathy persist. Three of five children admitted between 1973 and 1978 with the diagnosis of acute CO intoxication also have residual behavioral and/or school difficulties. An 8-year-old girl. The family had just completed an automobile trip that lasted 14 hours. The patient spent the entire trip in the back seat mostly "asleep," but she did vomit two hours prior to arrival home. COHb saturation level was 60%. Urinalysis showed 4+occult blood and 3+**protein**. Infrequent generalized convulsions. An ECG obtained on admission showed depressed T waves in the anterior chest leads, suggestive of diffuse myocardial ischemia. Two weeks later she was found to have absent light perception with intact pupils and normal optic fundi, suggesting a cortical blindness. The patient was discharged from the hospital on the 31st day, at which time she was described as clumsy, having a mild weakness of the left arm and leg, with frequent lapses of memory and notable visual impairment. Personality change, emotional lability, **inconsistency of reading ability**, **poor writing skills**, and complaints of blurred vision on lateral gaze. visual acuity of 20/100 in both eyes, mild ataxia of the left arm and leg; and symmetric, normal reflexes, **with Babinski's sign pre-**

sent bilaterally. She could copy words but had infantile writing. In reading a list of four words, she would consistently omit one or two. She was able to remember only one of three simple objects after five minutes. [165] +

A twenty-one-year-old man was comatose and his whole left upper extremity from axilla to wrist was swollen and of woody consistency. The hand was cool and cyanotic, but the nail beds were pink. The left radial pulse was unaltered. The skin of the left shoulder and antecubital fossa was **bullous and blistered**. There was complete motor and sensory paralysis of the left upper extremity. In April 1960, the appearance of his left forearm was that of a classic Volkmann's contracture. The sensory areas supplied by the radial, ulnar, and median nerves were anesthetic, and there was weakness with atrophy of the muscles of forearm and hand. The hand was kept in a position of slight flexion at the wrist, extension at the metacarpophalangeal joints and flexion at the interphalangeal joints. The nineteen-year-old female companion of Case 1 had similar involvement of the left upper extremity. In addition, all quadrants of the left, breast were swollen and woody in consistency. When discovered she was lying on her right side but may have turned to this position after lying on her left side until ischemic injury occurred. Five days after admission, a miscarriage in the first trimester of pregnancy occurred. Only a mild Volkmann's contracture developed. [166] +

This 31 year old patient was admitted in a state of stupor but soon became restless, agitated, and uncooperative. The skin and mucous membranes were pink, the pulse 120 per minute. He seemed to be completely blind in the left eye with only perception of light in the right, During the first two weeks he made almost no attempt at spontaneous speech, but answered questions appropriately. Vaguely absent-minded. **His plantar responses were now certainly extensor**. [167] +

Damage to cerebellum in patients with CO poisoning is rare. In this report, we share a less widely known magnetic resonance imaging pattern in the acute setting of CO intoxication with *damage to cerebellum*. A 11-year-old boy was brought to the emergency room by his mother. The child was in a comatose and lethargic state due to possible CO poisoning. [168] +

A 19-year-old male pre-law student became confused, then stuporous. An anoxic encephalopathy was diagnosed. With time he improved, but his intelligence had regressed to that of a 12-year-old child. He was referred to our center for HBOT 14 months after the incident. CT scanning showed rather diffuse white matter lesions and a mild degree of cortical atrophy. SPECT scan showed markedly decreased localization in both cerebral hemispheres, worse on the right, and in the right basal ganglia. The patient had spasticity in all extremities and the masseters. Although

formal psychological testing was not performed, it was apparent that his responses were *childlike and immature*, and that he was not able to speak in complete sentences. [169] +

A 21-year old, dilated unreactive pupils, reduced muscle tone and acidosis, **irritable**, **listless** and had amnesia for the incident; home for three days because of slight headache and transient giddiness; of odd behavior there he was sent back home. Five days after CO poisoning the patient visited another hospital because his headache had become worse and he complained of a fluctuating sensation of being blind. No physical abnormality was found. The pulse rate was 66/min, BP 140/88 mm Hg and the leucocyte count was 5,400. He was admitted for further observation. During the night his condition rapidly deteriorated, he started to vomit, complained of severe headache and showed *nuchal rigidity*. Lumbar puncture revealed clear fluid with 28 white cells (20 monocytes). The leucocyte count in the blood had risen to 12,900 and he had a temperature of 38.6 C° (which normalized in 48 hours). A CT scan and an ECHO-encephalogram were normal but the EEG showed diffuse pathological changes with pronounced paroxysmal activity over the right fronto-temporal region. The eye grounds were normal and the pupils reacted normally but the examiner could not exclude that the patient was "totally blind". [170] +

Tachypnea, cherry blush and warm extremities. On neurological examination, he had periods of alertness alternating with extreme somnolence and generalized chorea involving mainly the craniocervical region .[171]

Ten days after accidental exposure to carbon monoxide, a 17-year-old youth developed transitory choreoathetosis of both arms, face, and neck, with moderate **dysarthria**. CT revealed symmetric bilateral infarction in the head of the caudate nucleus, the putamen, and the small parts of the anterolateral globus pallidus. [172] +

Delayed encephalopathy due to CO intoxication, suffering from chorea involving a unilateral lower extrapyramidal symptoms. However, by the 20th day after acute insult, restlessness and involuntary movements of the right foot and leg developed but his mental state was alert and language functions, including comprehension and fluency, were normal, according to the statement of his parents. Gait disturbance and cognitive impairment followed a few days later. Confused, and his cognitive functions were impaired. Involuntary, non-rhythmic, brief and rapid movements were evident in the right leg, ankle and foot. He also exhibited a short step gait with decreased arm swing and increased muscle tone. [173] +

63

A 65-year-old woman was referred to our hospital in March due to a low-grade fever lasting for two days with the subsequent development of abnormal behavior (she drank her husband's alcohol, despite not having a drinking habit), dysarthria, **lack of initiative** (she stopped doing household chores) and a stooped posture while walking for over one month. On the first examination, the patient was drowsy and poorly responsive and showed forced grasping with chorea of all four limbs. She did not obey one-step commands. Her lower limbs were spastic, with an increased left Achilles tendon reflex. She was diagnosed with leukoencephalopathy of an unknown etiology. The chorea worsened, and she was restless in the evening. The patient exhibited **echolalia** in response to a doctor's questions, as well as environmental dependency behavior, e.g., trying to place a toothbrush in her mouth upon seeing it. **Euphoric**, *persisting in fiddling with a blanket continuously with her right hand* (frontal alien hand sign) despite a doctor talking to her (attentional disorder), stirring an empty cup with chopsticks when presented with the cup and chopsticks (compulsive manipulation of tools) and continuing to show forced grasping. She was able to read aloud her kana (Japanese phonograms) name written on a sheet of paper; however, when asked to write her name, she wrote lines repeatedly (motor perseveration). [174] +

The clinical cases of six patients suffering with **chorea** after acute carbon monoxide. She completely recovered 8 hours later, but 28 days after the acute insult, choreic movements of her extremities developed and gait disturbances followed 3 days later. Restless. Her memory and calculation were mildly impaired. Involuntary, nonrhythmic, brief, and rapid movements were evident in the extremities, face and tongue. Her speech was clear, but she had difficulty modulating the volume. When performing finger to nose testing, she grasped the examiner's finger. She also had short-step gait without retropulsion. [175] +

Ten days after an acute exposure to CO, a 33-year-old woman exhibited severe chorea. CT scan revealed bilateral lucencies of the pallidum and anterior arm of the internal capsule. On the second hospital day, she complained of imbalance and dysarthria but was discharged. The fifth day after carbon monoxide exposure, she was referred to an other hospital with **intention tremor** and **ataxia** but she was not admitted. Abnormal movements appeared ten days after poisoning. On admission, 12 days after carbon monoxide exposure, temperature was 36,8C, blood pressure 150/90mm Hg, respiration 24 per minute. The patient was fully conscious. She was severely dysarthric and swallowing was impaired. Abnormal involuntary movements were extremely violent. They were brisk, arhythmic and of great amplitude. They involved the trunk, the neck, the four limbs and spared the face. They consisted of flexion-extension of the distal muscles of the legs and of the forearms.

The proximal muscles of the legs were involved in a rotational movement. The trunk was arhythmically pushed forward and the head was moving from side to side. Attitude fixing, stress and movement induced bursts of choreic movements. More distally, the hands were involved by choreoathetoid movements. The patientcould not stand up or walk. There was diffuse hypotonia and normal strength of all muscles. [176] +

One day later, she completely recovered, but 28 days after acute anoxia, restlessness and involuntary movements of her extremities developed. Four days later, urinary incontinence and gait disturbance followed. she was alert, but confused. Her cognitive functions such as memory and calculation were mildly impaired. Involuntary, nonrhythmic, and rapid movements were evident in the face, tongue and extremities. She also had the grasping reflex and **short-step gait** with retropulsion. Her finger to nose testing showed a **coarse tremulousness** of both upper extremities. The tendon **reflexes were very brisk**, and there was no overt sensory deficit. [177] +

Ms. A, a 57-year-old woman with a history notable for alcoholism and depression, attempted suicide; profound psychomotor retardation, hypophonia, and prolonged speech latency. While lying on the bed (hugging a pillow and curled into a fetal position), she exhibited athetoid movements of her trunk, feet, and hands. Her eye contact was poor, **her affect was flat**, and she manifested **word-finding difficulty**. She endorsed poor sleep, **a lack of interests**, *feelings of guilt*, low energy, poor concentration, lethargy, and a continued desire to end her life. Three days after discharge, Ms. A grew more withdrawn, became less communicative, and developed odd, stereotyped movements (such as holding a sock in the air for minutes at a time and slowly crossing and uncrossing her legs). She displayed bizarre posturing and waxy flexibility, **frequently grimaced**, was mute, had **urinary and fecal incontinence**, and was resistant to passive movement. She also manifested periodic non-purposeful athetotic movements in her limbs (e.g., repetitive fanning of the fingers). She was abulic and spoke in short phrases (e.g., “This is torture”). [178] +

We report a 12-year-old male patient who developed transient motor and **vocal tics** twelve days after CO poisoning. Cranial magnetic resonance image of the patient showed bilateral symmetric hyperintensity in the caudate nucleus and putamen. One week after discharge, he developed involuntary movements of the left arm and leg. In the ensuing week, smiling, speech arrest, throat clearing and humming appeared. Neurological examination was notable for smiling, lip smacking, humming, speech arrest and bilateral shoulder shrugging, arm jerking and foot dorsiflexion. MRI study of Tourette’s syndrome patients showed that mean volumes of

the caudate, lenticular and globus pallidus nuclei were smaller than those of controls. Also, bilateral globus pallidus lesions on MRI in a patient with Tourette's syndrome was reported. [179] +

A previously healthy 58-year-old man had severe carbon monoxide poisoning. Following a comatose state, tics of the head, *coprolalia*, fits of shouting, and abnormal vocal utterances developed. In addition to the signs of diffuse encephalopathy, he had some of the features associated with idiopathic Gilles de la Tourette's syndrome. The computed tomographic scan showed ventricular enlargement and low-density areas in the basal ganglia. Unconscious and unresponsive **red** as though he had a sunburn. Both toes went up, labored breathing (32/min) There was a repetitive, stereotyped, irregular movement of the head that he could voluntarily abolish, but when he was distracted, the head movement reappeared. There were other brief to-and-fro head movements that were less frequent. His facial expression changed at times, and he broke into a peculiar laugh that was a mixture of laughing and crying. There was no jaw jerk, dysphagia, or sign of a bulbar palsy. His speech was clear and there was no dysarthria, but he had difficulty modulating the volume. He was able to name objects appropriately, although he misnamed parts of some objects. There was no aphasia. The visual fields were full to confrontation. The extraocular movements were full vertically and horizontally without nystagmus. He made guttural sounds, and continued to repeat the sounds in a crescendo until he was shouting. At other times he made a whinnying sound. He began to repeat obscenities, which he said were out of his control to avoid. He also had echolalia and **palilalia**. There were compulsive fits of shouting that were easily provoked. His verbal outbursts had a sexual content and he exhibited uninhibited sexual behavior toward the nursing staff and at home with his wife. This behavior was quite different from his premorbid condition. Motor examination showed a slightly weak right upper extremity and bilateral spasticity in the lower extremities, with extensor plantar responses. There was useful motor power in both arms, better on the left than the right. The right arm was contracted at the elbow. There was a dystonic posture of the right hand, but he was able to open and close his hand and to make a fist. The lower extremities were also contracted at hip and knee so he was unable to stand and walk. He sat in the chair with his hands clasped, the left hand holding the right hand. He could move his hands spontaneously when asked. He could imitate motor movements, but there was no echopraxia. *When using one extremity, he had mirror movements in the other.* For example, when saluting with the left hand, the right hand was compulsively brought into a mirror movement. When performing finger-to-nose testing he grasped the examiner's finger and was unable to inhibit the grasp without much encouragement. He also had a compulsive motor activity that seemed to force him to grab and grasp without control. When asked not to do this,

he could inhibit it. His finger-to-nose testing showed a coarse tremulousness of both upper extremities. The tendon **reflexes were very brisk** and slightly greater on the right than the left. There was no overt sensory deficit, although he was an unreliable witness. The computed tomographic scan showed cortical atrophy, widening of the sulci, and enlargement of the ventri- cles, with lesions of the basal ganglia. 180 +

On day three of hospitalization the two-year old child was noted to be mute, with masked facies, gait apraxia and a sad affect with very little spontaneous interaction with her environment. A MR study demonstrated bilateral increased signal in the anterior thalamus. 181 +

In a 33-year-old man six month after CO poisoning, a CT-scan of the brain revealed a diffuse cortical atrophy with enlarged ventricles and hypodensities in both pallidal areas. The neurological examination on admission in the psychiatric department demonstrated an akinetic mutistic state. Hyperreflexia, ankle clonus, and **contractures** of the four limbs were seen. Extrapyramidal signs were also present: bradykinesia, severe rigidity of the four limbs and neck with **cog-wheel phenomenon**, **festinating** and **shuffling gait**, a slight tremor and poor facial mimic. [182] +

A 62-year-old female patient, awake, in a mutism-like state, responding just using single words with a delay, obeying partially just single, simple commands. The patient had swallowing difficulties due to executional disturbances, which demanded a feeding tube insertion. At this time **hypomimia** appeared together with bilateral limb stiffness, **increased deep tendon reflexes**, **bilateral Babinski sign** and **sphincter incontinence**. Spontaneous speech was abolished. Usually, there were no answers to open questions. Other answers were very short, usually one or two-word answers with intact articulation and comprehension. Eighteen days after the termination of the hospital stay the patient died due to acute renal failure. [183] +

A 51-year-old male, an MRI performed 7 days after discharge showed bilateral globus pallidus changes and moderate diffuse high intensity of white matter. Three weeks after the poisoning, the patient developed progressive neurological deterioration. Cognitive dysfunction included impaired executive attention and memory of recent events, dyscalculia, apathy, alexia, motor aphasia in the form of **slow speech**, **agraphia** and **mutism**. Motor dysfunction included bradykinesis, generalized hypertonia, axial and limb paratonia, **loss of facial expression**, decreased blinking with preserved although slow ocular movements, abasia, forced dorsal decubitus, hyperreflexia without Babinsky sign, bilateral grasp reflex more marked on the right side, right Marinesco sign and right of symptoms and signs suggested a lesion localized in the frontal lobes, predominantly in the right. After the develop-

ment of delayed neurologic symptoms, the patient was treated with hyperbaric oxygen therapy. Commencing during the HBO treatment period, the patient experienced progressive improvement in symptomatology and three months after the development of DNS, neurological examination revealed strictly frontal signs: Marinesco sign and right grasp reflex, axial paratonia, and cognitive frontal-subcortical impairment. Five months after onset, the patient was asymptomatic. At this time, his neurological examination was normal, except for minimal frontal dysfunction with some difficulty in task planning and minimal loss of memory. Conventional scores of tests of cognitive function normalized. [184] +

Parkinsonian's syndrome. 185 +

Symmetrically increased reflexes of both arms, absent reflexes in the lower limbs, a right extensor plantar response, and an intention tremor and a resting tremor of all limbs. **Pyrexia of 103-105°F**, his pyramidal signs abated and a full parkinsonism picture developed with gross tremor and cogwheel rigidity. By the 4th week his pyrexia and delirium cleared leaving a severe dementia and persistent parkin- sonism. 186 +

A 43 year old housewife was admitted with history of progressive decline in cognitive and executive functions, alterations in behavior and slowness of movements of both upper and lower limbs and tremors in the right hand over the past 2 months. Two weeks later she noticed progressive forgetfulness and difficulty in concentration. Soon she developed urinary incontinence. Subsequently she continued to have decline in cognitive functions and eventually became bed bound within one month, requiring assistance in almost all her self-care activities. Motor system examination revealed hypertonia in both upper and lower limbs with **cogwheel rigidity** in the right upper limb and clasp knife spasticity in the left upper and lower limbs. There was also significant truncal and nuchal rigidity. All deep tendon reflexes were brisk. [187] +

Opisthotonos. Creatinine kinase, 27,368 units/L cogwheellike, resembling a series of saccades rather than smooth pursuit. 188 +

A 24-year-old man disorientation in time, place and persons, a visual agnosia, a prosopagnosia, memory disturbances, and a serious global dyspraxia. The CT-scan of the brain, performed on the 13th hospital day, was normal, but one month later enlargement of the left lateral ventricle was noted. Extra-pyramidal signs were first observed after 14 days with especially cogwheel rigidity, hypersalivation, poor mimic of the face and hypokinesia. [189] +

"Marche à petits pas", **a resting tremor**, an extrapyramidal rigidity predominant on the left side. 190 +

A case of major depressive disorder complicated by CO-induced Parkinson's syndrome is reported. Computerized axial tomography revealed bilateral globus pallidus necrosis. Both the mood and movement disorders responded fully to L-dopa. A 40-year-old Caucasian male was found in an obtunded state in his home following CO intoxication and was taken to a local hospital, where a blood carboxyhemoglobin level, drawn 1 hour later, was 30%. The furnace in his home was found to be emitting 0.02% CO because of improper ventilation. The patient reported a 5-month history of deterioration, characterized by depressed mood, with diurnal variation and distinct quality; **anergy**; **pervasive anhedonia**; **social withdrawal**; loss of sexual interest; early, middle, and terminal insomnia; **anorexia**, with a 20-lb weight loss; constipation; difficulties with concentration; preoccupation with failures in his marriage and job; **excessive guilt**; and suicidal ideation. At the onset of the depressive episode, he had lost his job, and his wife had separated from him, taking their children with her. According to several relatives and neighbors who had frequent contact with him and who verified the details of the above history, he had been living alone since that separation, without adequate heat, food, or **concern for personal hygiene**. There has been no history of movement disorders during the depressive episode. On admission, the patient was given a mental status examination and was found to be unkempt, withdrawn, and apathetic and had objective behavioral evidence of depressed mood (**sad appearance** and huddled posture), **retardation of movement and speech** with prolonged speech pauses, poverty of speech content, lack of spontaneity, and loss of affective reactivity. A neurological examination revealed moderately severe Parkinson's syndrome with bradykinesia; difficulty in turning or initiating movements; **cogwheel rigidity** in all four extremities; **resting tremor**; **masked facies**; **shuffling gait**, **with stooped posture** and decreased arm swing; micrographia; and a slow, soft, monotonic voice. None of these features were noted in the records from the previous hospital. many of motoric and behavioral disturbances (retardation of speech and movement, huddled posture, absence of facial expression of emotion, lack of spontaneity) the presence of apathy, rather than dysphoria, and an indifference to illness along with a denial of currently depressed mood. A diagnosis of parkinsonism secondary to CO intoxication was confirmed by an unenhanced CAT scan, obtained 4 weeks after the CO poisoning, that showed large, bilaterally symmetric areas of hyperlucency with the density of cerebrospinal fluid in the globus pallidi. This scan also demonstrated smaller, bilaterally symmetric hyperlucencies in the white matter of the posterior limbs of both internal capsules. No other structural brain abnormalities were noted on this scan, including caudate, putamen, or substantia nigra lesions,

multifocal or diffuse leukoencephalopathy, ventricular enlargement, or cortical atrophy. The globus pallidus and internal capsule lesions did not enhance with contrast, and no additional abnormalities were seen on the enhanced scan. Orthostatic hypotension. After L-dopa medication, he has remained in remission from the mood and movement disorders, When last seen 1 year after CO poisoning (17 months after the onset of MDD), the patient was in full clinical remission and had regained employment as a carpenter, an occupation requiring a high degree of manual dexterity. Repeat unenhanced and enhanced CAT scans given at both 12 weeks and 6 months after CO intoxication were unchanged from the scan given 4 weeks after poisoning, with the exception that the internal capsule lesions were less well visualized in the repeat scans We believe that our patient developed parkinsonism secondary to CO-induced bilateral globus pallidus necrosis. The onset of parkinsonism was temporally related to CO poisoning and was confirmed by a CAT finding (bilateral globus pallidus necrosis) that is characteristic of CO-induced parkinsonism. Finally, the age of onset and rapid progression of parkinsonism were atypical for idiopathic Parkinson's disease. [191] +

A man, aged 29, his face and hands were purplish. There was a moderately firm, non-fluctuant swelling just below the right ear the size of a small orange, and another firm swelling 4 cm in diameter on the right buttock. The rectal temperature was 101˚F. In addition there were large bright red blotches covered with blebs about the mass below the right ear, and over a large area of the right thigh and buttock where the skin appeared eroded. Urinalysis showed 3 plus **albumin**, and occasional hyaline casts and pus cells. Five days later, April 25, a neurologic examination revealed that the patient was conscious but mentally confused and disoriented as to time and place. His speech was drawling, monotonous and slow. A tentative diagnosis of encephalitis due to carbon monoxide was made, and the patient was kept for further observation and treatment. Somewhat sluggish mentally and presented a typical picture of parkinsonism, with immobile, expressionless face and husky, propulsive, monotonous speech. The motor power and all forms of sensation were normal, but all movements were reduced in speed and rapid finger movements showed the irregular interruptions and decreased amplitude so often seen in parkinsonism. There was a slight movement tremor in the finger to nose test. The handwriting was small (**micrographia**) and showed an irregular tremor. Shortly after this examination in July he began to have periodic attacks of rhythmic lateral tremor of the head, with similar pronation-supination tremor of both hands. These attacks came on about three or four times a day. The skin of the fingers had begun to lose its usual markings, to appear stretched and tight, and to present in general the appearance of scleroderma. The edema of the ankles persisted and the face appeared also more puffy than at any previous examination. Another spi-

nal puncture revealed a slightly blood-tinged fluid in which a count of seven cells was obtained. [192] +

30 yo man agitated and said that he was unable to see or to understand what was being said to him. The skin and mucous membranes were pink, the pulse 40 per minute. **Both plantar responses were sharply extensor**. Throughout this period there was marked suppression in the left homonymous visual fields. A remarkable **apraxia** for dressing. He was quite unable to put on his slippers, dressing gown, or pyjamas and had no idea what he should do with his jacket. He was unable to tie a simple knot in his dressing-gown cord. Complained that his hands appeared to be swollen and that all his fingers seemed to be fused together like a bunch of bananas. He had marked finger **agnosia**. Quite unable to find his way round the ward or to identify his own bed, suggesting that there was some spatial disorientation. He was unable to write, draw, or copy simple shapes for a month. Total acalculia, curious withdrawn attitude and gave the impression of being depressed. He had no Parkinsonian signs at first but these appeared about the tenth day and gradually disappeared in the course of the next week or two, during which time the plantar responses became flexor. Vague feeling of loss of confidence. [193] +

A 57-year-old woman was found unconscious. Brain computed tomography performed immediately revealed bilateral globus pallidus hyperdense lesions and equivocal hypodense lesions found in the periventricular regions, Severe akinesia and **cogwheel rigidity** was found. Twenty-eight days after CO intoxication, she was still not able to answer questions using complete sentences, although she could communicate by saying simple phrases or sentences. We started levodopa treatment, initiated at 50 mg three times a day for one month and a maintenance dosage of 100 mg three times a day for six months, for her parkinsonian features. Evaluation 7 months after CO intoxication showed improvement in rigidity and aspontaneity after medication but she still had problems with festinating and small-step gait. This case, as well as four other cases found in the literature, highlight the delayed development of pallidoreticular damages and its linkage in modulating prefrontal-subcortical neuronal circuits. [194] +

Of 242 patients with CO poisoning examined between 1986 and 1996, parkinsonism was diagnosed in 23 (9.5%). There were 11 men and 12 women. The age at onset ranged from 16 to 69 (mean 45.8) years, with the peak incidence during the 6th decade. The latency before the appearance of parkinsonism varied from 2 to 26 (median 4) weeks, but parkinsonism developed within 1 month after an acute insult in the majority of the patients. All showed encephalopathy with mildly to severely impaired cognitive functions during or immediately after delayed CO sequelae. The common symptoms were gait disturbance, impaired mentality, urinary incontinence,

and mutism. The most frequent signs were **short-step gait**, hypokinesia, **masked face**, **increased muscle tone (rigidity),** glabella sign, grasp reflex, and **retropulsion**. **Intentional tremor** was occasionally found, but resting tremor could not be seen. There was no correlation between the neuroimaging findings and the development of parkinsonism . Levodopa and anticholinergic drugs were not effective. Of 16 patients followed up for 1 year, 13 (81.3%) recovered spontaneously within 6 months. In conclusion, parkinsonism after CO poisoning is not rare and usually appears as a part of delayed CO encephalopathy. Any drug is not effective, but the prognosis is good. [195] +

A case of slow chronic intoxication; apathetic: a 49-year-old healthy man developed unconsciousness in a poorly ventilated room next of the kitchen. Muscle stretch reflexes were increased in the lower extremities with Babinski signs. There were masked face, bradykinesia, Parkinsonian gait, and abnormal righting reflexes. Fluid-attenuated inversion recovery imaging disclosed symmetric hypointense lesions in the thalamus and the globus pallidus. Hyperintense lesions were found in the cerebral white matter. [196] +

Cyanotic and unresponsive. Stuporous but not comatose; depressed and could not concentrate. Increasingly confused and withdrawn, *refused to eat*, and developed stereotyped picking movements. Mute, uncooperative patient with intermittent posturing. She responded with body movements to some commands but would not open her eyes or talk. The only positive physical or neurological finding was a bizarre gait; the patient would stand in place and make *small shuffling movements with her feet while moving her body in a clockwise rotation.* The patient spent most of her 4 months in the hospital on the floor, sitting, rocking, and **staring blankly**. She occasionally responded by giving her name in a faint whisper but otherwise would not talk to the nurses or her physicians. She often defecated in bed, and was sometimes **physically aggressive**. Felt like "something out of a book." mute, but when she does speak she confabulates. It became clear to the staff that she had considerable difficulty remembering recent events. Repeated neurological examination revealed bilateral tendon hyperreflexia, mask-like facies, and a parkin- sonian, gait. 197 +

Deeply comatose, breathing stertorously, and sweating profusely, with a generalized pink appearance. His pulse was rapid and weak and blood-pressure was 90/70 mm Hg. Lack of concentration and soon began to exhibit inappropriate behavior, such as drinking out of an empty cup and walking out of the house in a shirt. He also had difficulty in dressing, trying to put his waistcoat on as a pair of trousers, and so on. He walked with a Parkinsonian gait, was imperfectly orientated in time and place, required help with feeding and dressing, and soon exhibited inconti-

nence of urine. Neurological examination at this stage showed coarse tremor of the hands, hypertonus and weakness in the limbs, and occasional touching of the head in an aimless manner. Increasingly retarded and lacking in volition. He presented the picture of a kinetic mutism and neurological signs developed further. There was Parkinsonian tremor and rigidity, tendon reflexes became brisk, strongly positive grasp reflexes-more marked on the right side-were obtainable, and the plantar responses were now extensor. 198 +

A 50-year-old woman with carbon monoxide (CO)-induced parkinsonism was found to have bilateral lucencies of the globus pallidus on computed tomographic (CT) scan consistent with old necrotic lesions. She showed no clinical response to levodopa therapy, although she did improve with anticholinergictherapy. It is suggested that the parkinsonism in this patient is due to the pallidal lesions demonstrated on CT scan, and that such pallidal-related parkinsonism may not respond to dopaminergic therapy. Motor and coordination testing revealed mild bradykinesia, a resting tremor in lower extremities, moderate lingual-facial-buccal dyskinesia, and moderate impairment of postural reflexes, **micrography**, and slight masking of facies. The posture was stooped and the patient took small shuffling steps but tolerated a narrow base. There was mild cogwheel rigidity throughout. The strength was normal and the reflexes were symmetric without hyperreflexia. There was no Babinski's sign. A glabellar sign was present. The sensory system was normal. Computed tomography of the brain showed bilateral nonenhancing areas of lucency in both globus pallidi. Anticholinergic drugs alleviate Parkinson's disease and frequently worsen levodopa-induced dyskinesia, while levodopa improves Parkinson's disease but worsens tardive dyskinesia. 199 +

His speech was becoming slow, monotonous and low volume. Over the next 6 months, his symptoms progressed rapidly. His walking became slower, short, shuffling steps. He would frequently *freeze* when turning or walking through doorways, when rising from a chair or when getting in and out of bed. His speech had become almost inaudible. He complained of **constant, profuse sweating** and **sialorrhea**. He was unable to feed and bathe himself. Lying almost motionless in bed with a masked face and stooped posture and perspiring profusely. Marked rigidity of all extremities and at the neck. Finger dexterity was poor bilaterally. There was a slight coarse resting tremor in both upper extremities. He had a dramatic improvement with L-dopa. 200 +

Tremor of jaw and extremities, progressive dysphagia, sialorrhea, monotonous speech and bradykinesia, paresthesiae of extremities, demented. 201 +

Tremor of left arm and leg, thick mumbling speech, rigid unexpressive face "gradually over years" demented, hyperreflexia. 202 +

Progressive course of hesitancy in starting and stopping, flexed posture, progressive loss of speech, mentally retarded. 203 +

Evolved a more static picture with generalized muscular rigidity, but little tremor, **demented**. 204 +

Immobile face, monotonous speech, reduced movements, cogwheel rigidity and **micrographia**. 205 +

A 40-year-old male presented with acutely oncoming symptoms of **slowness of activities**, decreased speech output, disorientation, confusion, emotional lability , urinary incontinence, increased appetite and disturbed sleep of two weeks duration. One month prior to these symptoms he had been found in an unconscious state by his friends, in his closed, unventilated room where he had been sleeping overnight with a coal fire burning. He remained well thereafter until two weeks back when the above mentioned neuropsychiatric symptoms developed acutely. He had features of frontal lobe dysfunction in form of apathy, easy distractibility, perseveration and bradyphrenia and prominent extrapyramidal signs (rigidity, stooped posture, decreased arm swing and a positive glabellar tap). [206] +

Slightly increased deep reflexes to well advanced parkinsonism. Skin anesthesia and peripheral motor neuritides. 207 +

Demyelination in the central nervous system. 208 +

Cerebral myelinopathy. 209 +

On autopsy it was found diffuse demyeliaation, with the maintenance of dispersed morphologically intact myelin islets. The demonstrated chemical picture of demyelination in the observed case of CO myelinopathy is *similar to that seen in the demyelinating loci in the cases of multiple sclerosis* and in its diffuse form (Schilders disease), as well as in the demyelination seen in leucoencephalitis. The results may suggest that the catabolism of myelin lipids in the carbon monoxide myelinopathy does not essentially differ from that found in demyelinatiug diseases such as multiple sclerosis, including its diffuse form (Schilder’s disease), and in leucoeneephalitis. [210] +

The characteristic neuropathological findings after CO intoxication are petechial hemorrhages of the white matter, necrosis of the bilateral globi pallidi, spongy change, and progressive demyelination in cerebral cortex, thalamus, and hippo-

campus. Brain imaging, including computed tomography and magnetic resonance imaging, may demonstrate symmetrical globus pallidus and white matter changes in most patients. Temporal, parietal, and occipital lobes are usually affected with asymmetrical cortical and subcortical lesions in CO intoxication. The only damage of cerebellum in patients with CO poisoning is rare and less attention, unless multiple organs are involved. We herein report an 8-year-old girl with a high level of carboxyhemoglobin and an unusual diffuse hypodense lesion in her cerebellum. She made a very good recovery after 10 sessions of hyperbaric oxygen therapy. The damage in the cerebellum only in patients with CO intoxication is rare. In review of literature, Mascalchi et al and Nardizzi reported patients with CO intoxication showing damage in cerebellum and other parts of cerebrum. [211] +

The classical pathological changes of CO poisoning in the brain include diffuse white-matter demyelination and necrosis in basal ganglia, hippocampus, cerebellum, and cerebral cortex. [212] +

Twenty cases of peripheral neuropathy as sequelae of carbon monoxide intoxication have been analyzed clinically. The incidence of pheripheral neuropathy was 0.84% in a total of 2,360 cases and 3.64% in 549 admitted cases of carbon monoxide intoxication. The lower extremity was almost exclusively involved (except one case had only both upper extremities involved), and the left side was more involved than the right. Symptoms were a **burning sensation, tingling sensation, shooting pain and weakness**. Other associated sequelae were local swelling, acute renal failure, delayed neurologic sequelae, and Volkman's contracture in that order. Of 20 cases, 6 showed abnormal findings in the electromyogram only, and 14 were abnormal in both electromyogram and nerve conduction velocity. Nine cases of peripheral neuropathy involved the left side, 4 the right side and 7 both sides. Four showed evidence of involved lumbosacral roots. 213 +

Complications of carbon monoxide poisoning include **peripheral neuropathy**, which is usually confined to the lower extremities. We report a case of carbon monoxide-associated neuropathy resulting in unilateral *diaphragmatic paralysis*. A 56-year-old white man, labored respirations. The white blood cell count was 15,200/cu mm with a normal differential cell count. Chest x-ray film showed an elevated right hemidiaphragm. [214] +

An 18-year-old boy, deeply comatose, marked rapid eye movement, tachypnea of 34/min, pulse 110/min regular. Both hands were cyanosed, blisters on the left thigh and the buttocks had signs of **early epidermal necrolysis** generalized muscular rigidity, with episodes of facial spasm associated with flexion of his upper limbs and extension of both lower limbs. **His pupils became very dilated** but still responded

briskly to light. He was incontinent of urine. *On doing a further venesection the blood was noted to be cherry-red in color*. He lapsed into stupor on many occasions and **Cheyne-Stokes respirations** were observed; a grand mal fit. At 84 days the patient considered that he was entirely normal. However, his mother had noted loss of concentration and less social tolerance than before the incident. There remained slight impairment of rapidly alternating movements in both upper limbs and symmetrical hyperreflexia in both upper and lower limbs. There was impaired touch sensation of the right great toe and the left metatarsophalangeal region. [215] +

A 44-year-old female comatose and **arreflexic**; shortly afterwards she developed **apnea** and severe **hypotension**. After half a day, urine volume had fallen to 5 ml/hr. The urine was dark brown in color, and was initially reported to contain hemoglobin. The diagnosis of **acute renal failure** was made on the basis of continuing severe oliguria (urine volume <300 ml/day), a blood urea of 180 mg/ 100 ml and a urinary urea concentration of 800 mg/100 ml. The usual conservative regime for acute renal failure was established, but despite severe fluid restriction the patient developed gross edema of both legs and feet about 5 days after admission. marked tenderness of the leg muscles, **muscle necrosis** of the leg. Examination of the legs revealed that in addition to edema, her calf muscles were tense, her feet were fixed in plantar flexion, and there were bilateral contractures of the tendo Achillis. She complained of paraesthesia of the feet, and patchy sensory loss was found over the soles and lateral borders of her feet. [216] +

Acutely distressed followed by classic ‘grand mal’ seizure. 217 +

Severe CO toxicity: seizures, syncope, coma, myocardial ischemia, ventricular arrhythmias, pulmonary edema, and profound lactic acidosis. 218 +

Seizures: his head went backwards, and he began to moan and stiffened. Initially lethargic and irritable. 219 +

Tonic-clonic (grand mal) epileptic seizure and complained of blurring of vision. 220 +

Status epilepticus and cardiopulmonary arrest: 25-year-old Saudi woman who presented to our facility with status epilepticus and cardiopulmonary arrest. A regular pulse of 114 beats/minute, blood pressure of 127/98 mmHg and a respiratory rate of 24 breaths/minute. Her leukocyte count was 22,000 with neutrophilia. Her creatinine kinase level was 6,489 U/L (normal range 38 to 174U/L), creatinine kinase-MB isoenzyme level 143.5U/L (normal range 0.20 to 5.00U/L), aspartate aminotransferase 128U/L (normal range 15 to 40U/L), lactate dehydrogenase 4,59U/L (normal range 100 to 190U/L) and troponin I was 4.69ng/L (normal range <0.10ng/L). Se-

vere carbon monoxide poisoning associated with coma, hypoxic rhabdomyolysis and cardiac muscle injury. Generalized tonic-clonic seizure that continued for approximately 50 minutes. Soon after that she developed heart arrest. [221] +

A 37-year-old woman presented with a history of seizures for 11 months. The seizures initially occurred once a month and later increased in frequency. During each episode she experienced feelings of **de-personalization** and de-realization such as daydreaming with unreal thoughts, which were unpleasant. There were no associated olfactory or gustatory hallucinations, epigastric aura, incontinence or tongue biting. These episodes sometimes proceeded to complete loss of consciousness followed by feeling unwell on regaining consciousness. The patient also had symptoms suggestive of **orthostatic hypotension**. The patient gave a history of chronic CO exposure extending over a 7 year period starting from April 1989 during which she had persistent tiredness and headache associated with cognitive symptoms, personality changes and depression. Her house was found to be heavily contaminated with CO due to a faulty heating appliance. MRI of the brain 5 years after the end of chronic non-fatal CO exposure showed a well-defined lesion of the globus pallidus on the left, which was pear-shaped on axial images. Appearances suggest a cystic lesion in left globus pallidus. There was also presence of bilateral mild dilatation of temporal horns suggestive of hippocampal atrophy. [222] +

The patient was a woman fifty-eight years of age, who was overcome by coal gas which escaped into a room from a defective furnace. Vertigo and then fainted, subnormal temperature, shock, cyanosis, depression of the sensorium and inability to speak. Exaggeration of the left knee jerk and shallow slow respirations. Pulse of 96 per minute and respirations of 20 per minute, although there was some mental confusion. Restlessness developed. The following morning, approximately 18 hours after the CO exposure, she stated that her left foot was numb. That day, except that she was mentally sluggish and sleepy, no other abnormalities were noted, but the following morning, approximately 45 hours after exposure to the carbon monoxide her husband reported that she had not been able to sleep because of pain in the left foot. Two days later severe pain in the left foot and an obvious circulatory disturbance in that area. On this occasion the distal half of the left foot was cold and pale. Pulsation of the left dorsalis pedis artery was very feeble but definitely perceptible. On the third day after this admission to the hospital and six days after the CO exposure the skin of the dorsum of the left foot became dark and the following day this had spread considerably. Fever of 101° made its appearance now. The discoloration, an obvious **gangrene**, was rapidly progressive and was associated with pitting edema of the left lower extremity. About three weeks after the onset of the gangrene the left large toe was almost mummified, and the distal half and lat-

eral aspect of the left foot were dark, almost black and sharply demarcated. Amputation was performed at the lower third of the left femur 26 days after exposure to the carbon monoxide. The wound and stump healed well and except for a few days of stormy convalescence immediately after the amputation, recovery was smooth and satisfactory. The left lower extremity, when examined in the department of pathology, exhibited a gangrene conforming to that already described above. The entire arterial and venous systems were carefully dissected down to the toes, and transverse sections were made at intervals of approximately 1 and 2 cm. The gross appearance of the venous system was not significant. There were a few tortuous, occasionally dilated veins, some of them containing recently formed **soft blood coagulum**. There was no evidence of a generalized chronic thrombophlebitis. In the region of the popliteal space and immediately below it the veins and arteries were fused by what appeared to be a long-standing periphlebitis. The gross appearance of the arterial tree was also not remarkable. The larger vessels were obviously thickened with palpable densities made up of calcified plaques distributed at irregular intervals. In the femoral and popliteal arteries there were fragments of recently **formed blood clot**, which were partially and loosely attached to the endothelial lining. Diagnosis: ischemic gangrene of the left foot with a far advanced arteriosclerotic degeneration of the arterial blood supply and a terminal recent and partial thrombosis of the popliteal artery. [223] +

The relapsing form of carbon-monoxide poisoning is a term used to indicate those cases which, having apparently recovered from the first effect of the poison, develop, after a period of fair health, serious symptoms that may lead to death or insanity. 224 +

Delayed sequelae often involve neuropsychiatric symptoms such as impaired judgment, **poor concentration**, **disorientation**, confusion, coma, depression, cogwheel rigidity, **opisthotonic posturing**, extremity flaccidity or spasticity, extensor plantar response, and/or a relative indifference to obvious neurologic deficits. 225 +

A 62-year-old seaman was found unconscious at home drowsy and confused. On the next day, he was asymptomatic, Twenty days later, however, he had to be admitted again due to sub-acute memory impairment, confusion and inappropriate behavior. Now he was alert but completely disoriented; the neuropsychological examination indicated a severe impairment of attention and short-term memory, poor abstraction and categorization, and a marked paucity of spontaneous speech. The mental status suggested a frontal lobe syndrome, with loss of initiative, emotional blunting and some degree of psychomotor retardation associated with **mild euphoria**, *disinhibition* and *inappropriate facetiousness*. On the following days, his cogni-

tive status dramatically worsened with further decrease of verbal fluency, impairment of reading and echolalia; although there were neither focal motor deficits nor extrapyramidal signs, standing and gait had become impossible due to balance impairment and **severe retropulsion**, and he eventually became quite dependent, requiring assistance for daily life activities. EEG showed severe diffuse abnormalities, represented by a nearly continuous high voltage 2–2.5 Hz sharp-wave activity. On day 40, a MRI of the brain indicated multiple and partially confluent T2-hyperintense areas in the white matter of bilateral frontal and parietal lobes, with particular involvement of the periventricular regions. [226] +

A 20-year-old female tourist was found unconscious in a burning building; mildly confused and complaining of blurred vision but without neurological signs at the time of discharge from another hospital 3 days after the fire. She presented to us 3 days later with bifrontal headache, worsening confusion and difficulty in seeing. She was confused with poor memory for recent events and was disoriented in time and place. She was **easily distracted**, was **indifferent to her condition** and disinhibited in her behavior. She was unable to read (in English), identify numbers or colors or describe faces but could recognize friends. She could not recognize objects by sight but could name them using smell or sound (e.g. rattling a bunch of keys). There was no visual field defect. The pupillary responses were normal. She experienced great difficulty in manipulating small objects and, in particular, was unable to untangle knots. Further examination revealed the presence of bilateral palmo-mental responses, a pout response and mild pyramidal weakness of the right leg with ankle clonus and hyperreflexia. Superficial abdominal responses were absent on the right. [227] +

Approximately 1 month later, she became increasingly forgetful, and her family noted tremors in her hands. The patient's mental status deteriorated to the point where she was minimally responsive on physical examination. These clinical findings are compatible with the so-called lucid interval seen in CO toxicity of the CNS. 228 +

The long-term sequelae of non-fatal poisoning have received scanty references, and the prospects for the long-lasting survivors of acute CO intoxication are less clear. We present a case of CO poisoning with progressive neurological and psychological deterioration that began 17 years after recovery from a severe, accidental CO asphyxia. The patient was examined in the neurology out patient clinic 29 years after the initial CO intoxication. A 40-year-old woman was admitted to the Long Island College Hospital in 1997 with the diagnosis of acute gastrointestinal bleed, iron deficiency anemia and MS. She suffered a severe accidental CO poisoning at age 11 in 1968. After an initial comatose state and a prolonged convales-

cence period, physical and mental recovery was complete except for a very mild right spastic hemiparesis was medically stable until 1985 (age 28) when she noticed very slowly progressive walking and **coordination difficulties**. These difficulties worsened over the years to the point where she had to quit her job. She eventually developed urinary urgency, depression, insomnia, and weight gain and was diagnosed with MS. We were able to find only one patient with clinical similarity to our patient, reported by Kowalewski and Kesselring. This was a 15-year-old boy who was left with multiple neuropsychological sequelae after suffering an accidental CO poisoning. Nevertheless, the patient was able to work inside a protective environment and was able to care for himself. He remained stable for 2 years after the CO intoxication and then developed focal and generalized epileptic seizures for the first time. He was followed for 25 years and his neuropsychological status progressively declined to the point that he became **disoriented to time and place**, and became unable to walk. CT and MRI scan revealed **brain atrophy** with bifrontal gliosis. The clinical and MRI similarities between this patient and our patient provide strong evidence that these two patients represent a very late neurological complication of CO intoxication which *seems to be similar to the post-radiation motor neuron syndromes and post-poliomyelitis syndrome rather than the interval form of CO poisoning*. Our patient's initial diagnosis of MS, which was based on the MRI findings, could not be supported. MRI has come to be the standard technique for visualization of cerebral white matter disorders in MS, but MRI may portray similar appearing white matter changes in clinically diverse white matter processes. Therefore, it is essential to correlate the MRI abnormalities with the clinical findings before establishing a definite diagnosis. *The diseases that most commonly mimic MS on MRI* are small vessel ischemic disease in the preventricular location, subcortical arteriosclerotic encephalopathy, lupus erythematosus, neuro-sarcoidosis, migraine and *CO intoxication*. The differentiation between these disorders and MS may be particularly difficult when MRI is interpreted without input from the clinician. Relapsing and remitting MS was excluded in our patient by the absent history of exacerbations or relapses. Primary progressive MS usually starts at a later age (40.2 years) and the most common presentation by far is that of a slowly progressive spastic paraparesis. Visual evoked responses may be delayed. Spinal cord MRI may show demyelinating changes. Oligoclonal bands are present in a vast majority of the patients cerebrospinal fluid. Therefore, the patient's clinical history and laboratory data did not uphold the diagnosis of MS and was not compatible with many other disorders that can cause similarly white matter changes such as the leukodystrophies, disorders of lipid metabolism, mucopolysaccharidosis, ischemic arteritis, sarcoidosis, radiation and chemotherapy effects, viral induced de-

myelination, immune-mediated white matter disease or osmotic induced demyelination. [229] +

This study was conducted on 85 cases most of them were males with mean age 27.41 ± 16.67 years. The poisoning was accidental in 88.2% (mostly by coal heater in 58.8%) and suicidal in 11.8%. The mean of the delayed time was 3.88 ± 1.83-60 hrs. Most of the cases (70.6%) were admitted to the ICU with mean duration of stay 8.72 ± 4.08 days, 41.2% of the cases needed ventilatory support and 29.4% of the cases died. The mean level of carboxyhaemoglobin was 19.55 ±1 3.09%. According to the cardiac manifestations 64.8% were **hypotensive**, 29.3% were tachycardiac, 64.8% had abnormal ECG findings, 35.2% had high CK-mb and troponin-I. According to the neurological manifestations; coma was found in 10 cases, GCS ranged from 3 to 13, 29.4% had abnormal MRI/CT and 23.5% had neurological sequelae. Trying to find predictors for the severity of CO poisoning that may affect the fate; a comparison was done between alive and dead cases. There was significant deference between them in 14 parameters from which CO poisoning abnormality score (COPAS) was calculated to find out the probability of death. Cases with COPAS greater than 9 have high probability of death (high risk). 12% had cyanois and dyspnea. 230 +

Many cases with delayed encephalopathy showed **confusion to time, place**, and **person**, **urine and stool incontinence**, rigidity, bradykinesia, **stooped posture**, **mutism**, **masked face**, **resting tremor**, **bad temper**, **small step gait**. [231] +

Delayed postpartum hemorrhage: A rare presentation of carbon monoxide poisoning: Her postpartum convalescence was uncomplicated until 2 to 3 hours before presentation, when she began to have *soaked pads every 10 minutes by the passage of large clots*. She also reported lightheadedness, nausea, blurred vision, headache, and **shaking chills**. Pelvic examination revealed large clots in the vagina; during examination the patient appeared confused. She was **disoriented to time and place** and was unable to perform concentration or short memory tasks. A slight lateral gaze **nystagmus** was noted. [232] +

Four weeks later he was found to be behaving abnormally and had developed **urinary incontinence**. **Akinetic and mute state**. He had bilateral rigidity, release reflexes and brisk deep tendon jerks. **He was refusing oral feeds**. [233] +

Generalized chorea due to delayed encephalopathy: a 60-year-old woman, who had completely recovered from acute CO poisoning, developed mental and behavioral changes, urinary incontinence and generalized chorea 2 weeks thereafter T2-weighted brain magnetic resonance imaging showed extensive hyperintensity of

the bilateral periventricular and subcortical white matter and the globus pallidus. Two weeks later, she began to experience cognitive impairment, behavioral changes, unsteady gait and urinary incontinence. Restlessness and involuntary movements of the limbs were also observed; alert but could not obey or understand orders. She exhibited **akinetic mutism**, **rigidity of the limbs**, and a **short-stepped gait**. Persistent, rapid, irregular, involuntary choreic movements of the whole body, which were *invisible during sleep*. MRI showed bilateral, symmetric, confluent areas of high signal intensity in the periventricular, subcortical white matter, corona radiata, centrum semiovale and globus pallidus; The symptoms of generalized chorea gradually improved and disappeared completely 2 weeks later. The cognitive impairment improved moderately after 20 sessions of HBOT. However, the urinary incontinence, mild rigidity of the limbs, and short-stepped gait remained unchanged after discharge. Follow-up brain MRI 8 months later showed more cortical atrophy and prominent hyperintensity of the bilateral periventricular white matter. [234] +

The 30-year-old male MRI of brain showed hyperintense change of the bilateral globus pallidi. After 10 sessions of HBOT, he was discharged with mild depressive symptom. However, his condition deteriorated quickly 1 month later with symptoms of declined cognitive functioning, **aphasia**, **apraxia**, **dysphagia**, **muscle rigidity, urine and fecal incontinence**. He could not talk, walk, eat, response to any stimulation and even displayed decorticate-like posture with bed-ridden. Therefore, he was admitted to neurology ward again under the impression of delayed neuropsychiatric sequelae of CO intoxication. MRI showed diffuse hyperintensity of the bilateral hemispheric white matters consistent with changes of delayed leukoencephalopathy as a result of prior CO injury. The bilateral globus pallidi had central necrosis with cavity changes. [235] +

A 71-year-old woman, after improvement of her consciousness she could leave the hospital, but was readmitted 3 weeks later because of increasing confusion. The clinical examination showed hypokinesia and **cogwheel rigidity** of both arms. Other significant signs were the **frozen facies**, the absence of speech and the *marked seborrhea*. A CT-scan of the brain after one week and 6 months later showed only a diffuse cortical atrophy, with large ventricles but without hypodensities in the white matter and the basal ganglia. The initial mutism disappeared. Neuropsychological sequelae remained evident; **euphoria**, **perseveration**, disturbances in criticism, judgement, memory and a tendency to **confabulation**. With this clinical picture of a Korsakoff-syndrome, the patient was finally transferred to a revalidation centre. [236] +

Dementia, mental retardation, hallucination, catatonia, manic depressive state, Korsakoff's syndrome, Kluver-Bucy syndrome. 237 +

An 11-year-old boy was one of a family of five that had suffered severe CO intoxication due to a faulty furnace 4 years previously. He later developed seizures, severe headaches, and difficulty concentrating. His school performance deteriorated. [238] +

A 29-year-old woman had been exposed to CO from an exhaust leak in her vehicle for 3 months. She at first complained of memory loss and migraines, poor coordination, confusion, and difficulty in problem solving. [239] +

A 37-year-old woman, due to severe migraine and nausea; symmetrical and sharp tendon reflexes, plantar skin reflexes extending on both sides; hemoglobin oxygen saturation at 50%. The COHb level is 19%. Based on good recovery, the patient went home 12 hours later. After one month of symptom-free recovery, she begins to experience almost **permanent headache** of variable intensity and major depressive symptoms. The subjective complaints of the patient are then an important fatigability which does not diminish even if she sleeps for a long time, a sad thymia, almost permanent headaches of variable intensity, memory loss, attention and concentration disorders and a lack of the word. [240] +

Long-term neurocognitive deficits occur in 15–40% of patients, whereas approximately one-third of moderate to severely poisoned patients exhibit cardiac dysfunction, including **arrhythmia**, left ventricular systolic dysfunction, and myocardial infarction. Studies have reported that 2% to approximately 10% of patients display delayed neurological sequelae. [241] +

A 28-year-old male patient presented five months after CO poisoning with *achromatopsia*. The achromatopsia was unaccompanied by an inability to recognize faces (prosopagnosia) nor was there any disorder of form or depth perception. Magnetic resonance imaging showed bilateral sharply defied areas of hemorrhagic infarction in the globus pallidus with extensive infarction involving temporal and occipital lobes and with apparent partial sparing of the visual cortex, presumably due to arterial insufficiency. During the first ten days he made no effort at spontaneous speech and seemed to be apathetic to his plight and environment. There were no obvious focal neurological signs. **His speech was drastically abbreviated to a reper- toire of one or two attenuated fragments such as 'yes', 'not sure', 'don't know'**. By the third week, although unable to express himself in writing, he could read and could understand most of what was being said to him. By the fifth week there were only minor defects in spoken speech and he could make a fair attempt at writing in script. He could write his name and address and the names of most of the clubs in the rugby football league competition. He had no difficulty in identifying

letters of the alphabet. He could understand what was being said to him and his replies, in the main, were appropriate. The following month was spent in the rehabilitation unit and by the time he was discharged he could manage simple calculations, but made a few mistakes in telling the time and had occasional difficulty in dressing. Five months later he *complained that things about him appeared 'unreal* and lacked sunshine'. He mentioned hesitantly that everything appeared as if it were muddy and grey and that he had considerable difficulty in perceiving colors. At first he was able to realize this difficulty, for example, saying to his wife that their color television set looked like 'one of the old black and white ones' There was a definite disturbance of color perception observed in the sorting of balls of colored wool. He showed no disturbance of color naming. When asked the color of a pineapple, said it was yellow, and the color of a post office letter-box, red, but had most difficulty in distinguishing blue and green colors, which were perceived as dark brown or grey and generally the world around him seemed depressingly drab and greyish. His affect was not incongruous. He appeared genuinely worried by his visual disturbance, and *was at times close to tears*. When shown a piece of blue material he said it looked 'brownish' and shown an orange he said it appeared 'reddish', a yellow banana appeared 'pale'. Surprisingly, without much difficulty he could match the various shades of red wool—pink, scarlet and crimson. [242] +

Quantitative MRI analysis allows for the detection of subtle corpus callosum changes that may not otherwise be observed following CO poisoning. The long-term effects of CO on the brain have been historically underestimated; however, we found subtle but significant corpus callosum atrophy and cognitive impairments following CO poisoning. [243] +

Brain damage from CO intoxication has not been extensively studied with magnetic resonance imaging. We report the clinical outcome and brain magnetic resonance imaging in three individuals simultaneously exposed to toxic levels of carbon monoxide four years previously. Lesions are seen in multiple locations and do not correlate well with the clinical condition. [244] +

Loss of memory, especially for recent events, increasing for two years. He had **difficulty with speech** because words were difficult to enunciate in the correct order. His gait became unsteady, causing him to sway and to bump into other people. Left-sided headache was occasionally present. [245] +

Pulse 94, and the respiratory rate 36. He was somnolent without focal neurologic deficits showed a right bundle branch block and nonspecific ST-T wave abnormalities. The white blood cell count was 20,800, creatine kinase was 2,293 IU/1 (normal <220) with an elevated MB fraction. Cranial CT two weeks later showed a hy-

podensity in the left thalamus. Subsequently, severe depression was diagnosed. Neurologic examination four years later showed psychomotor abnormalities. MRI showed lesions in the left thalamus, medial temporal lobe, and cerebellum.
[246] +

Among the patients studied, 22 had been acutely exposed to CO while 8 were chronically exposed. One month after CO poisoning, 12 of the 22 patients with acute intoxication showed magnetic resonance imaging abnormalities: 6 also had neurological sequelae and 6 were asymptomatic. The remaining 10 patients showed neither MRI abnormalities nor neurological sequelae. During the 3-year follow-up, 4 of the patients with both MRI abnormalities and neurological sequelae improved in both clinical features and MRI findings. One of the 6 asymptomatic patients with MRI abnormalities developed a progressive cognitive impairment 2 months after acute intoxication, with a concomitant severe worsening of the MRI lesions. Among the 10 patients with neither MRI abnormalities nor neurological sequelae, only 1 developed neurological sequelae after a clear period of 4 months. In the group of patients who experienced chronic CO intoxication, only 1 presented with a neuropsychiatric syndrome which improved at follow-up. Brain MRI showed white matter lesions which remained unchanged at control scan after 1 year. The group of patients with neurological sequelae had significantly longer periods of unconsciousness (27.0 ± 21.7 h vs. 2.9 ± 4.9 h) and CO exposure (7.2 ± 5.2 h vs. 1.7 ± 1.6 h) compared with the group of asymptomatic individuals. The most common MRI findings were hyperintensities on long TR sequences of both deep and periventricular white matter, found in 10 patients. These abnormalities were rather diffuse, with no preferential sites. In 5 patients white matter abnormalities were associated with basal ganglia lesions and in 4 patients with cortical atrophy. One patient had only basal ganglia lesions. One patient who experienced chronic CO intoxication presented with a neuropsychiatric syndrome characterized by **mutism**, **masked face** and cognitive impairment, already present during the period of exposure, and still present 1 month after the end of CO exposure. MRI demonstrated widespread white matter lesions. All patients with normal neurological examination as well as MRI at baseline evaluation remained asymptomatic during the 3-year follow-up, except for one subject. This patient, at month 4, developed an akinetic-mute state and became bedridden within a few months; MRI disclosed deep and periventricular white matter hyperintensities and bilateral globus pallidus lesions. **Frank parkinsonian** features were present in 4 patients. The remaining 2 cases showed chiefly psychiatric symptoms such as **depression** and **delusions**.[247] +

Parkinsonism, chorea, athetosis, ballism, myoclonus, tremor, dystonia, Gilíes de la Tourette's syndrome; hemiplegia, apraxia, hyperkinetic state, hemianopsia, cortical

blindness, agnosia, anosmia, hearing disturbance, motor or sensory aphasia, anomia, agraphia; convulsion, epilepsy; syringomyelia; polyneuropathy, mononeuropathy, facial palsy; vegetative state, **akinetic mutism**. 248 +

Cerebral cortex showed patchy cortical necrosis with severe edema. The necrosis was particularly distinct in the posterior lobe. The head of the nucleus caudatus was highly edematous and hemorrhagic. The basal ganglia, cerebral white matter and hippocampus were felt to be soft at autopsy, in consistent with the histological findings of edema. The vermis cerebelli was slightly atrophic. 249 +

Unconscious at home with vomit around their mouth, and evidence of both fecal and urinary incontinence. On admission, the patient was tachycardic (130 bpm) and hypertensive (160/90 mmHg). He was resuscitated, intubated and ventilated, and a CT head performed. This revealed bilateral basal ganglia hemorrhages. 250 +

Isolated cerebellar damage caused by CO intoxication in a 5-year-old girl with convulsions. [251] +

Cerebral myelinopathy. 252 +

Mild hydrocephalus. 253 +

Endocranial hypertension with compression of the fourth ventricle as a result of cerebellar edema. Obstructive **hydrocephalus**. **Fine intentional trembling**, hypotonia and hyperreflexia. cerebellar atrophy with cortical and subcortical ischemic lesions. 254 +

Cerebral edema with acute hydrocephalus. Generalized tonic-clonic seizure. Arterial blood gas showed a pH of 6.76 (N: 7.35–7.45). Deep tendon reflexes were 3+bilaterally at the biceps, triceps, and patella. **Bilateral ankle clonus** and Babinski signs were present. **Enlarged, fixed, and dilated pupils**. Bilateral retinal hemorrhages with venous engorgement. Corneal reflexes were absent. 255 +

The pathological changes most commonly seen in CO poisoning are in the region of the globus pallidus bilateraly, and ess commonly in the medial temporal lobes and the cerebral white matter. There was a neutrophilia of 27,7000. More drowsy and developed **Cheyne-Stokes breathing**. Obstructive hydrocephalus with cerebellar swelling and compression of the fourth ventricle. Restriction of external ocular movements in all directions of gaze. Mild cerebellar ataxia. 256 +

Prolonged delirium suggesting that all degrees of functional or structural neurological damage may have occurred. 257 +

Neurological: Coma. In the majority of patients exposure to CO produced impairment of consciousness. **Severe hemiplegia** developed in four patients. Two of these were thought to have suffered cerebro-vascular accidents whilst lighting gas fires, though the possibility that the hemiplegia was the result rather than the cause of exposure to CO could not be refuted. The other two cases are worthy of further mention here. Constricted pupils with sluggish or absent reaction to light. Transient deafness was observed in one patient and incontinence was a feature in six patients, two having combined urinary and fecal incontinence. 258 +

Left hemiplegia and an enlarged right pupil suggested tentorial herniation. Persistent dementing process. From the eighth to the eighteenth day the presence of tremor of the hands and cogwheel rigidity of the limbs indicated extrapyramidal involvement. 259 +

Delayed encephalopathy is caused by demyelination of the white matter, followed by **brain atrophy**. 260 +

Delayed encephalopathy with bilateral pallida and milder diffuse periventricular hyperintensity. On day 20 after exposure, he suddenly developed delirium and marked disorientation. Difficulty with walking, urinary and fecal incontinence and involuntary twitching movements of the knee became apparent. He became bedridden and **mutistic**. The twitching movement of the knee became more pronounced. After delirium for 1 week, disorientation and amnesic aphasia remained. Furthermore, aspontaneity and blunted affect. Most function recovered over the long term, but loss of initiative and the blunted affect remained. 261 +

Headache, faintness and vomiting at 2 am while doing a night shift, and later diarrhea, followed by reduced self-care, a lack of interest of work in which he was interested before the illness. He found it difficult to work as a technical officer due to low energy, lethargy, poor concentration and reduced memory. He was **withdrawn** at times and was less communicative. He endorsed poor sleep and had a low mood. His condition deteriorated over the next 2 weeks and developed episodic inappropriate talking, confusion, generalized rigidity of the body and urinary and fecal incontinence. **He was unable to identify his family members** and developed reduced level of consciousness. Generalized rigidity with hyperreflexia and bilateral extensor plantar response. He gradually developed masklike face, positive glabella sign and primitive reflexes (grasp reflex) seven weeks after CO poisoning. He was diagnosed with delayed encephalopathy associated with exhibited athetoid movements of his feet and hands and went into rigid akinetic mute state. 262 +

In a general way it produces a sensation of warmth and tension in the stomach, it excites the peristaltic action of the stomach and intestines, promoted the appetite and digestion, increases the secretions of the stomach, accelerates the respiration and pulse, and according to some authors, causes slowness of the pulse, and diminishes the temperature. 36 +

The whole organism becomes freshened and energetic, and mental activity is increased. 36 +

Large quantities cause vertigo and congestion of the brain. 36 +

Change of symptoms, varying course, intermittent. de Sonnaville °

Good day, suddenly bad day, or suddenly exhausted. de Sonnaville °

Slow recovery. de Sonnaville °

Also after recovery: suddenly tired again, or suddenly sharp worse after exertion. de Sonnaville °

Worse first movement, better continued movement. de Sonnaville °

Mind

Patient depressed and stupid. 36 +

Wants to die! Reliving forgotten pneumonia when he was 7 years old. Was very very lonely then. Transformation, part of his depression is over now. de Sonnaville *

Intoxicated confused stupid. 1202 *

Answers with difficulty. 1202 *

Confused, lost. de Sonnaville *

It's hard to concentrate. de Sonnaville *

Very forgetful. de Sonnaville *

Significant brain fag. Thoughts felt slow and imprecise. Difficult recalling words. Not clear thinking. (Known symptom, but much worse.) (Day 6). 908.HA.1

Slow and slow, in moving and thinking. de Sonnaville *

Very difficult to think, I am unable to follow my own thoughts in the evening (Day 7). 908.HA.3

Dazed in the head, clear and concentrated thinking not possible. (after 30C). 908.HA.6

Hard to concentrate, like in fog, dizzy, not focused (Day 3 and 4). 908.HA.7

Poor concentration. de Sonnaville *

Hard to collect my thoughts (Day 4, 30C). 908.HA.8

Was only able to read a mindless easy novel, but not able to read a more difficult text or something work-related (Day 3,5, 30C). 908.HA.8

Lazy, postponing sport, unmotivated (Day 2). 908.HA.7

Apathetic. 36 +

Lying down, don't have the energy to do something, apathetic. de Sonnaville *

Resignation with serious complaints. de Sonnaville *

Chilly but almost an indifference or apathy to become warm (immediately). 908.HA.9

Utterly calm; barely breathing and it doesn't bother me at all. Pulse 88. (first hour). 908.HA.9

Feels slightly stupid (first hour). 908.HA.9

Happy to sit still in my rocking chair and disappear into a perfect reverie (day-dreaming), from which I can pull myself out of this state easily (but I don't want to). (first hour). 908.HA.9

Aware that her face was utterly still and relaxed, a pleasant numbness; blinking occasionally, but dreamily (first hour). 908.HA.9

Daughter is speaking to her and she registers that she is talking, but she doesn't pay attention to a single thing she's saying. She can, but she doesn't want to (first hour). 908.HA.9

While eating started to tremble from the cold, yet was unwilling to do anything to warm up (second hour). 908.HA.9

After eating, my mind started to clear and was able to think more linearly and logically (second hour). 908.HA.9

Since yesterday morning, I have been experiencing a kaleidoscope of symptoms that are unremarkable, insipid, and utterly unworthy of remembrance and changing every 5-10 minutes. They have no discernible relationship to each other. I have absolutely no desire to write them down, and have no desire to make any effort whatsoever to dignify them with the tiniest whiff of posterity (second day). 908.HA.9

I am sensitive to the remedy (and further exploration will be necessary), then Carbn-o. is a slow acting, insidious remedy with no discernible characteristic other than its insidious, unremarkable, and forgettable nature that can manifest in any conceivable way with a degree of apathy that would be *very difficult (if not impossible) to quantify and qualify in practice.* 908.HA.9

Poetic feeling (first hour). 908.HA.9

With the lassitude, an unusual apathy, and indisposition for any muscular exertion. 36 +

Mental inactivity. 36 +

Mind sluggish. 36 +

Indolence not for daily activity, but only to write the symptoms of the proving (fourth day). 908.HA.9

Feels as if everything is too much and desires rest. She is a bit dull, has less initiative. de Sonnaville *

Desires to rest, being alone, leave me alone, aversion sound, music. de Sonnaville *

Quiet passivity. de Sonnaville *

Gloomy, quiet. de Sonnaville *

Staring. 1202 *

Felt in a very confused and stupid state. 36 +

Very dull, hard to think, confused, sometimes so tired that a good anamneses was impossible, **difficulty breathing deeply** or through, **being very cold**, **very**

painful and tight muscles, **worse motion** and sometimes **with restlessness**. de Sonnaville *

Confusion and stupefaction of the senses and intellectual faculties, amounting at last to complete unconsciousness. 36 +

Answers only with difficulty. 36 +

Stupor and imbecility. 36 +

Consciousness disappears. 36 +

Complete loss of consciousness. 36 +

Comatose. 36 +

Could not be aroused. 36 +

Looks anxious. 36 +

Excessive anxiety; expression anxious. 1202 *

Anxiety about not recovering (health). de Sonnaville *

Anxious, afraid of dying, worried, tearful. de Sonnaville *

Condition resembling intoxication the whole day. 36 +

A woman was found upon the street half unconscious, talking senselessly, screaming violently, only able to say that several of her family were similarly affected; after she got to the house was taken with a violent shaking chill, followed by decided heat. 36 +

Spasmodic crying. 36 +

Screaming and convulsions. 36 +

Patient depressed and stupid. 36 +

Sadness and despondency. 36 +

Restless. Despair of recovery. de Sonnaville *

Frightful anxiety, and instinctive impulse to seek change of air, while he felt powerless to overcome the paralysis of his muscles and move from where he sat. 36 +

Great anguish. 36 +

Apathetic. 36 +

Lack of initiative. de Sonnaville *

After supper, felt buoyant and in good humor, an effect never produced by tea; this mental sensation soon passed over into an irritable and sarcastic one, so that I sharply criticized an article in a medical journal, which a few hours ago I hardly though worth that trouble, and threw away in disgust a book of reference, as flat and superficial, which I highly value at other times. 36 +

Intellectual. 36 +

Very contracted range of ideas. 36 +

Incapacity to draw inferences, or to compare ideas. 36 +

Confused ideas. 36 +

Felt in a very confused and stupid state. 36 +

Confusion and stupefaction of the senses and intellectual faculties, amounting at last to complete unconsciousness. 36 +

Answers only with difficulty. 36 +

On attempting to describe their sensation (according to letters left by suicides) the first few lines are well written, afterwards phases are incomplete, and at last there are only words and letters. 36 +

Dull and ever-changing images passed before my mind, but I felt unable to concentrate my mind on any one. 36 +

Forgot to brush my teeth in the evening before going to bed, had to get up to do so. Forgot to take my eye-drops, had to get up again, unusual for me (Day 1).
908.HA.7

Went in the cellar several times to get things and forgot what it was (Day 4).
908.HA.7

Memory much impaired; remembered nothing of his attack; could not answer questions correctly for two days, and was not able to resume his occupation for a month (after three days). 36 +

Memory completely lost for five days. 36 +

Time passes more slowly during a Qui Gong exercise. (after 30C). 908.HA.6

Day seems very long. de Sonnaville *

Stupor and imbecility. 36 +

Fell to the ground stupefied. 36 +

Unconscious. 36 +

Quite unconscious till the third day. 36 +

Consciousness disappears. 36 +

Complete loss of consciousness. 36 +

Consciousness is sometimes suddenly lost, as if the person had been struck upon the head. 36 +

Unconsciousness and piteous moaning. 36 +

Shortly became insensible. 36 +

Struck senseless to the floor (after one moment). 36 +

Could not be aroused. 36 +

At the time of testing (August 1998), case 4 was a never-married Caucasian female who had last worked as a cook 3 weeks prior to testing. This patient was the sister to case 3. Compared to her functioning levels prior to poisoning, this patient reported having frequent headaches, fatigue, and emotional liability. Significant problems were noted in **obsessive-compulsive behaviors**, **hostility**, and *excessive fears*. Additionally, significant problems with depression and interpersonal relations were noted. This patient explained that her vocational deficits associated with the poisoning included forgetfulness (e.g., forgetting to go to work or going the wrong day), short-term memory problems, and difficulties retrieving previously-learned information. She also described problems with expressive language, inattentiveness, and general mental efficiency (e.g. problem solving). In particular, she explained that it took her longer to solve routine problems. [263] +

Slow, as if she forgets what she thinks. Memory is bad, even forgets what she did. de Sonnaville *

A 27-year-old married who reported having frequent headaches. They occur frequently, typically 3-4 days during the week, *and are alleviated only with sleep*. He also reported decreased short-term memory and difficulty learning new information, both of which were reported to be worsening. Over the past 2 years, this patient reported becoming **increasingly irritable** and **easily angered** and having a *decreased tolerance for frustration*. Comparable to the two previous cases, emotional behavioral questionnaires revealed significant complaints of cognitive difficulties, anxiety, anger, and obsessive-compulsive tendencies Testing indicated that this patient had low average intelligence, which represented a likely decline from premorbid level. Measures of verbal and visual memory, language, abstract thinking, and perceptual-motor skills were commensurate with his measured intelligence. His immediate visual memory was impaired, although this finding was not consistently found across all measures of memory functioning. [264] +

Following an increase in nausea, tinnitus, dizziness, and headaches which had persisted for 3 days, the four subjects self-admitted to the emergency room and were diagnosed with acute CO poisoning: case 1 was a 25-year-old never-married Caucasian male; at the time of testing, this patient reported significant memory and sleep difficulties. He reported having frequent headaches, fatigue, and anxiety, as well as decreased interest in usual activities **depression**, **anxiety**, and cognitive difficulties (e.g. *distorted*, *irrational thinking*). Obsessive-compulsive tendencies were noted, as well as significant difficulties in relating to and interacting with others. These symptoms began approximately 5-6 years post-injury. He had complaints of being unable to remember instructions, being slow and forgetful, and having difficulty with attention and concentration. At the time of testing, this patient complained of difficulty remembering and comprehending complex verbal instructions on his current job, which contributed to decreased work efficiency. [265] +

This patient was a 33-year-old: At testing, this patient also reported significant residual effects of his poisoning, including frequent headaches, short-term memory problems, and **decreased attention and concentration**. He explained that he had *difficulty waking in the morning*, was often irritable, and had a lowered frustration tolerance. Although the memory and learning problems began shortly after the injury, feelings of depression and anxiety had reportedly increased over the previous 2 years **obsessive-compulsiveness** and cognitive difficulties. Significant difficulties with interpersonal relations, depression, anxiety, and anger were noted. Since this poisoning, he has held six jobs, primarily in food service, but lost those jobs due to slow, inefficient performance. On the job, he noted having significant problems with memory and cognitive tracking, typically having difficulty with sustained concentra-

tion. This subject reported that it takes him longer to learn new jobs and that he is irritable and short-tempered. [266] +

Case 3, aged 21, on admission to hospital this patient had recovered consciousness but was *delirious with outbursts of violence* and purposeless activity, and that he was slightly deaf. Headache, vomiting, and a serious disturbance of vision, so that he was only able to see hand movements. He now had difficulty in naming objects, **disorientated in time and space** and on the third day had two major epileptic fits. [267] +

Case 4, aged 18, was still unconscious on admission to hospital with no response to painful stimulation. He later became very restless and noisy, ***using obscene language to the embarrassment of one and all.*** The pulse rate was 120 per minute. On the following morning he was confused and disorientated, and moderately deaf in both ears. He failed to recognize his father's car and had difficulty in undressing himself; *he began to swear* and **behave in an odd fashion** and in the early hours of the next morning he almost certainly had a major epileptic fit. now *fluctuated between delirium and stupor*. *He continued to swear profusely and groan and howl like a caged wild animal*. **He was incontinent of urine and feces**. During the second week, *he continued to have violent and purposeless movements of the limbs with groaning, howling, and incontinence*; no intelligible words were heard and he was clearly out of contact with his surroundings and unable to recognize his parents. Normal words were used inappropriately on the twenty-fourth day and speech returned at the end of the fourth week, though he was still disorientated in all respects and continued to show attacks of violence and aggression (psychotic behavior). Simple arithmetic stopped at 2 x 2. He was quite unable to put on any of his clothes. He was still quite unable to dress himself or fasten his shoes, nor could he undress himself, and buttons presented the greatest problem. According to his parents he had lost his sense of money and was never able to pay for any thing except with a note. His parents said he was **extremely irritable** and **short-tempered**. [268] +

A 28 year-old woman had a toxic exposure to CO when her furnace malfunctioned. Six weeks later, she was rehospitalized with "confusion". Her family described a personality change which began 4 weeks after recovery from her acute CO intoxication. *The patient stopped engaging in her usual activities and would lie in bed unclothed*. She would allow her small son to wander unattended and would greet strangers in an undressed state. Previously, she was "strong-willed", "uptight", and "analytical". Subsequently, she became "nonchalant" and "emotionally labile". Furthermore, the patient had trouble finding her way in her surroundings. Her interview was marked by confabulation and a *lack of concern for her hospitalized condition or*

for her son's status. This patient developed a persistent frontal lobe syndrome, visuoperceptual impairment, and white matter lesions following recovery from acute CO intoxication. Her behavioral change included apathy, disinhibition, confabulation, perseveration, poor judgement, and a dysexecutive syndrome. [269] +

Severe paranoia and increased irritability; inability to do simple calculations, such as addition and subtraction of two-digit numbers; dysnomia; loss of short-term memory; nausea; vomiting; worsening of her preexistent headaches; and disorganized thought processes. [270] +

A 32-year-old woman was admitted for treatment of a conversion disorder. 4 years ago, she accidentally suffered from CO poisoning. She was comatose for 3 days, and brain magnetic resonance imaging performed during that period showed bilateral globus pallidus necrosis. She gradually recovered and maintained good social and occupational functioning. Two years ago, she began experiencing unremitting *diurnal bruxism*, *which manifested as clenching, grinding, grating and gnashing of the teeth*. The bruxism occurred throughout the day and resolved during sleep. Additionally, she developed a gradual reduction in spontaneous activities and speech and became depressed. After admission, she exhibited constant, unremitting, forceful teeth grinding and **jaw clenching**. Her thinking, verbal fluency and intellectual processes were predominantly slow. She usually took at least two hours to complete a meal and displayed a marked reduction in spontaneous activities and speech. She could stand or sit in the same place without doing anything for many hours, while this condition could be immediately reversed with external solicitation. [271] +

Urinary retention, a spastic paraparesis and a T12 sensory level; withdrawn, uncooperative and appeared depressed. She appeared unable to catheterize herself or **find her way about the ward**. She remained apathetic and **refused to eat or drink**. She remained mute with some catatonic features and negativistic behavior. Cerebral CT and MRI performed at this time, however, showed extensive lesions of white matter bilaterally. [272] +

An engineer, 20 years of age, described as being a most intelligent workman, was sent into an exhauster, a space just large enough for one man, to make some repairs about the fans. He came out dazed and stupid, **talked incoherently**, and was very “heady” till 5 or 6 in the evening, when he began to talk quite rationally and could write his name. Except for these first few hours he had little apparently wrong with him for nearly three days, when suddenly his mind became a complete blank. If asked to look through a window at a horse he would repeat the word "horse," but if questioned further his *answers would be silly and irrelevant*. He had to be tended

like a baby. This went on for a month or two, then there was a slight improvement, and he is described *as being at this stage like a big simple child*, who was very easily managed. After a time he was given lessons by the village school teacher, beginning like a child with his alphabet, and soon after he started work, but here also he had to begin as an apprentice, as he had forgotten everything, even how to handle his tools. At the end of ten years he was in the best of health, talking rationally and intelligently, and seeming to an ordinary observer quite normal, but those who knew him before his illness said that mentally be was only a shadow of his former self. He had lost all initiative, always had to be told what to do, and if a responsible job was given to him **he was sure to get excited**. His memory was poor, he was **very easily irritated**, losing command of himself, and then was hardly responsible for what he did. After an attack of anger or excitement his memory became much worse. He was, however, constantly improving. [273] +

Every thing seemed to be in a whirl; there seemed to be a dense white fog. We all sat without trying to escape. The foot of the ladder was quite near, but none of us made an effort to reach it. When he found himself being overcome, he took out his notebook and wrote a farewell letter to his wife and children. In this letter he would repeat certain words, even whole sentences, again and again. He remarked, “I had absorbed enough of the poison to paralyze me to a certain extent, but at the same time my reason had not left me. The general sensation was like a bad dream, and yet I was able to reason properly and write intelligently, though in a disjointed fashion." [274] +

A truck driver experienced severe mental deterioration, behavioral changes and delirium after acute CO intoxication and committed suicide 15 months later. Confused. **Despite being incontinent of urine and feces**, there was no focal neurological deficit. The left side of the body was widely erythematous as in 'initial signs of frostbite'. He was tachycardic and the pupils were equally reactive to light. Neurological examination revealed hypostenia and denervation of the lower limbs, that appeared more marked on the left-hand side. Babinski's sign was negative. The EEG was normal. Myoglobinuria and was present with raised of muscle enzyme levels (CPK 12,900 UI/L, ALT 163 UI/L, AST 432 UI/L, LDH 974 UI/L). He was diagnosed with acute renal failure caused by rhabdomyolysis. An EMG identified the partial denervation of the sciatic left nerve. Six to 7 months after, admission tests revealed good recovery of dorsal flexion in the feet, but also showed deep depression, 'the psychological situation was deteriorating steadily' and diagnosed 'major depression', symptomized by insomnia, lack of appetite and melancholy, with considerable reduction in facial expression and gestures. He referred to notions of ruin, accompanied by olfactory hallucinations. persistent association with delirious ideas

of self-destruction, was helped little by treatment. Two months later an MRI showed hyper-bright focus on both the basal nuclei, but greatest on the right. Fifteen months after the initial episode he escaped the watchful eye of his family and hung himself in the garage of his home. [275] +

Apathetic mask-like facial expression, symptoms of dementia, such as amnesia and disorientation, urinary incontinence, and hypokinesia. Other relatively frequent symptoms were irritability, distractibility, apraxia and gait disturbance. Bizarre behaviors, such as foolish smiles or frowning, sometimes not easily distinguishable from simple manneristic behaviors, were the interesting findings. [276] +

Difficulty recognizing new words and mathematics (slow in calculation) at school. [277] +

Chronic exposure: A 2-year old grandson, who lived with the couple that winter, was diagnosed with a developmental speech disorder called aphasia during the summer of 1995. [278] +

Aphasia with apoplexy. 1202 *

A 34-year-old Hispanic man with a 2-year history of untreated major depression attempted suicide by carbon monoxide poisoning 6 months prior to admission to our inpatient psychiatric unit. *He was unable to resist continuous urges to spit*, even indoors, and to pull the hair on his legs. At about the same time, *he developed obsessional ideas such as thoughts of harming his wife and friends, intrusive sexual fantasies*, and *fears of contamination* such as having contracted the AIDS virus even though he had no risk factors. The patient also exhibited compulsive behaviors including *repetitive hand washing*, *checking behaviors, and counting rituals*. He had no history of obsessive-compulsive symptoms prior to the carbon monoxide poisoning. Magnetic resonance imaging of the brain showed bilateral globus pallidus infarction consistent with his history of carbon monoxide poisoning. He was diagnosed with obsessive-compulsive disorder secondary to bilateral globus pallidus infarction and major depression in remission. [279] +

It is not at all uncommon to have a man who has been gassed while at work pass through a stage of violent delirium. Here is described a typical case as follows: The man is overcome by the gas, but does not lose consciousness completely. He is hurried by ambulance to the hospital, reaching there usually about 10 or 15 minutes after the occurrence of the accident. By this time he is often **delirious**, even **maniacal**, so that he has to be restrained in bed. This stage, however, lasts only a few hours; then he gradually becomes rational and complains of intense, headache. [280] +

Lethargy, headache, and occasional vomiting diagnosed as “flu”; low school grades; severe cognitive deficits, especially those involving executive functions; flu-like illness including headache, malaise, and nausea with slowed intellectual processes. [281] +

Mild but definite cerebellar dysfunction including mild tandem gait dysfunction. Having previously served in a police car, he was transferred to desk duty because he lost his way in areas he had frequently previously patrolled. Mild anxiety, apathy, and hopelessness; memory loss, slowed responses, and ataxia on neurological examinations. Scored below expected levels on reading (fourth percentile) and spelling (second percentile). Lost his way and went home. The next morning, he was amnestic for the prior day. [282] +

Tachycardia, dilated pupils, and loss of bladder control. Pulmonary edema. Psychomotor retardation and cerebellar **dysmetria**. **Fine motor dexterity was impaired**. Problems with attention, confrontation naming, attention to detail, nonverbal problem solving, and abstract thinking. [283] +

Severe psychomotor retardation, bradykinesia, masked facies, rigidity without tremor, severe incoordination, and severe intellectual impairment, being unable to recall his address. He had been a semiprofessional soccer player. Neuropsychological testing placed him in the intellectually deficient range of severely impaired in all realms of cognitive functioning including expressive and receptive language, visioperception, visioconstruction, visual memory, attention, working memory, executive functioning, and fine motor control. Brain MRI demonstrated extensive white matter signal abnormality in the internal capsule, mid-brain, and pons more prominent on the left side. MRS showed decreased NAA in the basal ganglia bilaterally. His condition was classified as a Parkinsonism-dementia complex. 284 +

Memory impairments can be mild, moderate, or severe. He remained in the hospital for several weeks and then returned to work. After returning to work, however, he. could not remember “even simple instructions.” After several weeks, he was unable to retain or form new memories and was unable to remember any experience for longer than a few seconds. He could not remember any new information, general knowledge of the world did not increase over time. Even years later, his memory did not improve suggesting significant brain injury, at least to the medial temporal lobe brain regions, which are critical for the formation and storage of new memories. 285 +

She had become incontinent of bowel and stool upon exiting the car, and had a very brief loss of consciousness of no more than several minutes shortly after en-

tering her house. Following return to home, the patient experienced a number of cognitive and emotional difficulties that included *stuttering*, having trouble remembering the words she wanted to say, feeling sad and depressed. She additionally reported symptoms of balance problems, pins and needles in her feet and arms, memory problems, trouble thinking as quickly as before, difficulty with writing, increased sadness, irritability, and a lack of interest and general feelings of **hopelessness**. These symptoms persisted at a consistent level over the next 2 years, and up until the time of evaluation. During the testing session, the patient was found to be moderately labile and socially inappropriate. She became tearful at times, and *wept intensely* at several points of the testing when she had difficulty completing the task. At other times she was noticeably **euphoric** and **overly cheerful**. Her speech intermittently was halting and mildly disfluent, and accompanied by a mild dysarthria. She had occasional word-finding problems, and demonstrated occasional literal paraphasias. 286 +

A case study of CO poisoning due to burning of charcoal briquettes found bilateral lesions in the hippocampus that were associated with severe impairments in memory. A second case study found bilateral hippocampal damage and temporoparietal atrophy following CO poisoning that was associated with impaired verbal and visual recognition memory. A third case study found severe memory impairments and bilateral hippocampal atrophy six months after CO poisoning. A study of 20 patients with moderate-to-severe CO poisoning found hippocampal atrophy and associated memory impairments as well as generalized brain atrophy measured by the ventricle-to-brain ratio. 287 +

Irritable, *did not care to dress or wash himself*, refused to sit down for his meals, but ate them standing before the mantel-piece. He finally became confused, *abusive*, and **violent**, very clouded sensorium, tremors, athetoid movements, exaggerated deep reflexes, generalized muscular spasticity with fibrillary twitching, polyneuritis, incontinence of urine and feces, and three healed areas each two centimeters in diameter on the back of the lower right leg. Impairment of temperature perception over the lower extremities, weakness, ataxia, and adiadochokinesia. When asked to shake hands he would not perform the action requested despite his avowed understanding of the question asked. Instead, he would touch his nose, rub his hands together or perform some other foreign movement. His knee jerks were still exaggerated, there were decreased power in the left upper extremity, a left digital tremor, and hypoesthesia over the fifth finger of the left hand. Mentally, he seemed somewhat excited and there was memory defect with impaired judgment. The patient lost interest in his personal appearance, lacked ambition, and had **outbursts of temper** during which he would **swear violently** contrary to his

former habits. He performed silly actions, indulged in alcoholic beverages, and was unable to hold a position despite his good physical condition. 288 +

Psychic akinesia: apathetic, inert and, left alone, would sit in bed all day long doing nothing; loss of initiative, which led the patients to lose all their previous interests. He spoke rarely, only when addressed, and answers were simple and short, though to the point. He lost all interest in work, self-care, and trivial daily activities, became withdrawn and unable to show emotions. When coaxed, however, he would execute correctly any order or task. 289 +

Stuporous: confused, weakness of the tendon jerks, right arm weakness. (brain CT, performed 3 days later, displayed bilateral calcification of the globi pallidi and mild cortical atrophy.) Mental slowness. 290 +

Ten months later, showed a hypomimic (masked face) and hypokinetic man, weak deep reflexes. Obsessive features, for instance, *continuously buttoning and unbuttoning his shirt while watching TV*. 291 +

Abulia [lack of will or initiative), emotional indifference, loss of interest in the environment and memory and attention deficits. He gave up his university studies and led a solitary life. 292 +

Confused with anterograde memory impairment. Twenty days later the patient showed a change in personality: he had become apathetic and emotionally dull. A second CT scan showed bilateral hypodensity of the globi pallidi. Retro-anterograde memory loss (retrograde amnesia is the inability to recall past memories and anterograde amnesia is the inability to create new memories). slowing of thought processes consistent with a pattern of subcortical dementia. 293 +

Confused state and unable to recognize her family members with acute confusion and partial retrograde and total anterograde memory loss. 294 +

Severe impairment of memory and verbal fluency. In the absence of external stimulation *he lay for hours, eyes open, doing nothing*. The contrast between his intellectual capacities and this inactivity was obvious in all aspects of his life. He talked only if asked, he took part in sport (he was a sports coach) only if stimulated by his wife, he went to visit friends only if invited by a phone call, and so on. His only spontaneous activity was of a routine nature like going out and getting the newspaper. His affect was impaired in the same way. If asked about the recent death of someone he cared about he cried sincerely, but if asked about recent events of his life he forgot the death and talked only about some political news. Stereotyped activities were not reported spontaneously by the patient, or by his wife. But when

questioned on this point, *he admitted counting when he was alone with nothing to do; he counted from 1 to 20 again*. To stop it he had to go out, or watch TV. This purely mental activity did not give him anxiety or did its withdrawal. 295 +

Akinesia was obvious and verbal fluency was markedly reduced. Intellectual processes were slow and the whole picture tended to give the impression of mental deterioration. Dramatic passivity, **his lack of initiative** despite the fact that his motor and mental capacities were largely preserved. He was able to perform artistic paintings; but for years he painted the same landscape of moors and fens, and this several dozens of times. 296 +

Expressionless and more silent. 297 +

Increasing loss of spontaneity to such an extent that she ate only when she was spoon-fed; she never changed her position on the bed unless otherwise helped, and she showed no verbal response when questioned but gave only slight nodds. 298 +

Anosognosia for blindness with purely visual confabulation (Anosognosia, also called "lack of insight," is a symptom of severe mental illness experienced by some that impairs a person's ability to understand and perceive his or her illness. 299 +

She became mopey, *dragged her feet*, seemed tired and wanted to lie down. She talked, took nourishment, did not have pain and made no complaints. The last lwo days at home were notable because of the loss of control over the bladder and bowels. About three weeks before admission she was overcome by coal-gas while sleeping. She was found unconscious and taken to a hospital where after ten hours she recovered consciousness, and in two days was discharged fully recovered and able to attend to her usual duties. This condition of good health lasted but a few days. She was unconscious, irresponsive to sensory stimuli, her face had the appearance of general muscular contraction, giving to it **a frowning** or stern expression, a sort, of squeezed expression. The corrugatores supereilli, the orbiculares palpebrarum and zvgomatici muscles were apparently spastic. The jaw was tightly closed. The left pupil was slightly larger than the right, which was irregular. The patient was incontinent of urine and feces. The inner segment of each lenticular nucleus shows a well-dellned area of softening. 300 +

He muttered and sang in a low voice; he had no idea of place or time, was incontinent and said he heard and saw the Lord. Later he could not be aroused. The right pupil was very much dilated; the left was contracted and irregular. They did not respond to light. There was rigidity of all the limbs, especially at the elbows. The pa-

tient lay on his hack with head and eyes turned to the right, both forearms, especially the left, were strongly flexed on the arms. Extension of the forearms passively was difficult, and when the left was extended the patient screwed up his face as though in great pain. Pricking the face with a pin caused no change in expression. The lower limbs were slightly flexed at the knees but were more easily extended than the upper. The man moved the right arm and both legs at times quite freely, but did not move the left arm—at least no voluntary movement of this limb was noticed. The biceps and triceps reflexes were exaggerated. The patellar reflexes were not obtained, probably because of the spasticity. Plantar stimulation gave plantar flexion. Symmetrical areas of softening were found in the inner segments of the lenticular nuclei. 301 +

Two four-year old identical twins were irritable and crying and complained of headaches and stomach pains. They suddenly become unresponsive to mild painful stimuli. Their respirations were unlabored, although mildly tachypneic. 302 +

Persisting delirium in both suicidal and accidental patients. In 21.3% of suicidal patients and 18.9% of accidental patients this delirium was prolonged for periods ranging from many hours to 4 weeks, indicating continuing interference with cerebral function. These patients were characteristically **disorientated for time and place**, with poor attention and concentration, impaired grasp and disordered thinking. Visual hallucinations occurred in one patient. **Irritable** and restless and often became violent. A number of patients on regaining consciousness displayed irritable, **aggressive** and **uncooperative** behavior and discharged themselves from hospital before a full psychiatric examination could be carried out. Possibly some of these were delirious. 303 +

CO poisoning results in significant depression and anxiety that persist to at least 12 months. Neurocognitive impairments are common following CO poisoning and include impaired memory, executive function, slow mental processing speed, decreased intellectual function, apraxia, aphasia, and agnosia. An unexpected finding was that the accidentally CO poisoned patients were as likely as patients with suicide attempt to have depression and anxiety at 6 and 12 months. 304 +

Akinetic mute state with unusual motor seizures, probably associated with tentorial herniation and brain-stem compression. Restlessness, irritability, and **belligerence**. Within the next few days his relatives noted that he was lethargic and apathetic, his memory for recent events had deteriorated, and his concentration was impaired. He became moody and depressed, his appetite was poor, he lost weight, complained of loss of libido, and was sleeping excessively. Failure to keep business appointments and other lapses of memory together with increasing inertia were as-

sociated with a catastrophic deterioration in his small motor-parts business. His family became increasingly intolerant of his changed behavior and numerous arguments ensued. Irritable, and aggressive. Violent behavior. Uncooperative. temporospatial disorientation, impaired concentration and attention, labored thinking, and the memory disturbances. Delirium persisted, characteristically showing *nocturnal exacerbation*. By day 7 he did not respond to questions or requests, had ideomotor, constructional, and dressing dyspraxia and became **highly irritable** and **aggressive** if disturbed. A fine tremor of the hands and rigidity of all limbs indicated extrapyramidal involvement. He was incontinent of urine. Leucocyte count 9,300/cmm and ESR 47 mm/hr. Liver function tests revealed raised SGOT (88 units/ml.) and SGPT (45 units/ml.) levels. Less aware of his surroundings, showed only vague signs of recognition of the staff, was mute, and spontaneous. Motor activity was confined to stereotyped *rocking movements*. He swallowed food in an automatic manner and developed fecal incontinence. On the 13th day he suffered a motor seizure lasting 30 seconds with *violent cycling movements of the legs* and incoherent mutterings. On the 14th day he deteriorated to an akinetic mute state. He was mute, displayed no spontaneous movement, and **lay in bed with his eyes open**, occasionally fixing them on an object or person. Food presented was automatically swallowed and painful stimulation produced reflex withdrawal of the limbs. Emotional expression was absent and when spoken to he showed no signs of recognition. Urinary and fecal incontinence persisted. Left hemiplegia, bilateral grasp reflexes, and an **enlarged right pupil** suggested tentorial herniation. Dressing and constructional dyspraxias (inability of patients to dress and copy accurately drawings or three-dimensional constructions). **Euphoric**, overactive, and **talkative**, though he continued to show an expressive dysphasia which caused him considerable frustration. When seen 18 months after the second exposure he was well-groomed, mildly elated, *loquacious*, and somewhat overactive. Remote memory was relatively intact, but there was defective retention of recent events and confabulation was used so effectively as to be barely detectable on casual conversation. The family verified that he had affective flattening with a tendency to *mimic* the emotional responses of others. He was *impulsive*, disinhibited, and had a **volatile temper**. He **lacked initiative**. His general hygiene and care in his appearance were markedly different from his premorbid fastidiousness. He had resumed an active social life, though with little interest in the opposite sex, and he was employed in the family business of managing a boarding-house. 305 +

Depression, anxiety, neurasthenia, insomnia, melancholia, personality and judgment changes, amnesia, astasia-abasia; dementia, mental retardation, hallucination, catatonia, manic depressive state, Korsakoff's syndrome, Kluver-Bucy syndrome. A 52-year-old woman presented to the ER with sporadic complaints of

shortness of breath, nausea, weakness, left-sided paresthesias, and flashbacks. The patient had been painting her house outdoors with Rust-Oleum spray paint 4 days prior to coming to the ER. Since painting, the patient had experienced the aforementioned symptoms approximately 4 times daily. The flashbacks involved vivid recollections of childhood memories. The symptoms were unrelated to any activity, usually lasted a few seconds to minutes, and had never been experienced before. 306 +

Disoriented to time, looked perplexed and was inconsistent in her performance. Her perplexed facial expression was again noted. Her psychomotor activity was clumsy: she had difficulty extending the proper hand for a handshake. Inappropriate mild euphoria was apparent. She did not comprehend commands, or could she perform simple tasks such as raising her right hand or walking to the door and opening it. She was disoriented to time, place and situation. Immediate recall and recent event were impaired. Her general fund of information showed a striking deficit. Judgment, concentration and abstract thinking were severely impaired. Shuffling gait. 307 +

A case with delayed encephalopathy after CO intoxication, which began with neurological symptoms and continued with **obsessive-compulsive disorder**, depression, *kleptomania*, and psychotic disorder. The 41-year-old female patient had no psychiatric or neurological symptoms or disorders prior to CO intoxication. We concluded that the kleptomania seen in this patient was related to concurrent lesions in the temporal lobe and globus pallidus; in other words, her kleptomania may have been related to dysfunction simultaneously seen in both the temporolimbic and frontal-subcortical circuits. On month after the intoxication, she started to experience forgetfulness, nervousness, and disordered speech. An anxious mood and irritability, obsessions, and *interrogation and control compulsions*. After a 4-5-month asymptomatic period, *the patient began to steal various objects from shops*. The patient reported that she was ashamed of her stealing behavior, but could not control the impulse to steal. She also reported that she generally stole objects that she did not need and would not use, and that this situation was not related to financial difficulties. Moreover, the patient started to suspect that her husband was being unfaithful because he began arriving home late at night and she would spend the whole day struggling with suspicious thoughts about him. After a short time she became convinced that her husband was being unfaithful despite evidence to the contrary and her husband’s reassurance. The patient became increasingly irritable and nervous and she *started to fight with family members* and began to threaten suicide. Difficulty concentrating. Mild forgetfulness and distraction. Anxious and depressive mood, difficulty in sleeping, *intense hostile feelings toward her husband*,

irritability, delusions regarding her husbands' infidelity, attempts to confirm the delusional thinking, and impulsive stealing behavior. [308] +

Loss of consciousness for a long time. 36 +

Vertigo

Gloomy confusion of head. 36 +

Inclination to turn in a circle. 36 +

Kept going round in circles. Anand *

Dizziness and tired all day (Day2). 908.HA.7

Strong dizziness, like getting black before eyes, like almost fainting, need to lie down and close my eyes, physical and mental weakness (Day 2). 908.HA.7

Vertigo worse after slight exertion, better eyes closed. de Sonnaville *

Want to lie down and nothing else. de Sonnaville *

Tendency to vertigo, and to turning in a circle. 36 +

Vertigo, to staggering and falling. 36 +

Staggering when walking. de Sonnaville *

Vertigo, with flickering before the eyes. 36 +

Vertigo, and temporary darkness before the eyes. 36 +

Continual vertigo, especially on rising after lying down. 36 +

Giddiness and trembling, so that he fell to the ground. 36 +

On rising up, staggered was obliged to hold fast to something, and sank exhausted into a chair. 36 +

The slightest movement is liable to bring on vertigo and loss of consciousness. This occurred twice in this case, the man saying he felt well and wanted to go home, and **becoming unconscious when he started to get up**. 309 +

Vertigo: disorientation and light-headedness, sudden, *severe sensation of rotation*. faint, bilateral tinnitus although his hearing was unaffected. 310 +

Head

Heaviness of the head. 36 +

Heaviness of the head, without vertigo. 36 +

Excessive heaviness in the head. 36 +

Dull heaviness of the head. 36 +

On rising, heaviness of head, which lasted all day (second day). 36 +

Pressing headaches in the morning on waking (Day 6, 30C). 908.HA.4

Head pressure after getting up (Day 2, 200C). 908.HA.4

Head pressure in the morning (Day 3, 200C). 908.HA.4

Head pressure on the forehead on awakening. (Day 4, 200C). 908.HA.4

Head pressure all day long (Day 4, 200C). 908.HA.4

Pressing in head (Day 5, 200C). 908.HA.4

Head pressure at 2:30 p.m. after physical exertion (32 degrees Celsius) (Day 8, 30C). 908.HA.4

Broad pressure on the vertex at 8 a.m., which disappears in the open air (Day 2). 908.HA.5

Pressing pain on the vertex again around 10 p.m., which goes away with sleep (Day 4). 908.HA.5

Constant, non-descript frontal headache (2/10) with an ever-so slight clenching of the teeth (second day). 908.HA.9

Slight head pressure rising from the occiput directly at the transition of the cervical spine and skull calotte and rising on both sides in a hood-like manner. On the left side a painful spot (this is where I hit the concrete wall when I fell). Head pressure on the forehead and neck better after a cold shower, cold footbath and a cold wet cloth on the forehead. Sensitivity to light, darkness improves, after two hours of sleep in the afternoon between 2 p.m. and 4 p.m. the head pressure is significantly better. Had strong shoulder and neck tension. (after 30C) (Day 17). 908.HA.6

Headaches in the morning when waking up (Day 9, 200C). 908.HA.4

Headaches morning when waking up, throbbing all over the head for several days. (after Day 10, 200C). 908.HA.4

Headaches morning when waking up on the top of the head with sensitivity of the brain, like a mild soreness for several days (after Day 10, 200C). 908.HA.4

Headache and facial pain better after getting up, better by movement (swimming) and better after the first cup of coffee at around 9 a.m. (after Day 10, 200C). 908.HA.4

Headache left temple at 8 p.m., extending to left eye and left jaw, pressing, went to bed at 10 p.m. still with headache, now focused in left orbit. (2 hours after 30C) (Day 1). 908.HA.7

Headache left temple extending to left eye and yaw (again 2 hours after 30C) (Day 3). 908.HA.7

Headache on the left side from forehead to occiput in the afternoon, pretty intense 7/10, but it lasts only a few seconds (Day 4, 200C). 908.HA.3

Headache, band, hairache touch. de Sonnaville *

Headache worse noise, **better pressure, coolness, eyes closed, moving.** de Sonnaville *

Headache worse mental effort, light, cough, noise. de Sonnaville *

Better pressure, heat, massage, **lying.** de Sonnaville *

Very heavy head worse after exertion. de Sonnaville *

Pain head and neck, stiff worse awakening, better hot shower. de Sonnaville °

Severe headache, especially occipital, worse bending, worse bending head back. de Sonnaville °

Pulsating. de Sonnaville *

Stitching. de Sonnaville *

Vertex, forehead, occiput. de Sonnaville *

Worse slightest movement, lifting head impossible. de Sonnaville *

Worse motion. de Sonnaville *

Headache with nausea (Day 2). 908.HA.6

Increasing nausea during the morning (Day 2). 908.HA.6

Around noon for three hours more severe nausea with dryness in the mouth, must lie down, vomited three times. Every movement such as turning around in bed and getting up from bed worsens the nausea; talking, being spoken to, eating, drinking, stronger pressure on the stomach worsens the nausea; laying hand on the stomach improves slightly (Day 2). 908.HA.6

Lie very still, in the dark, do not want any noise, so nausea and headaches are more bearable. Cold and damp cloth on forehead and eyes relieves headaches, a bit also the nausea. With the nausea and headache a feeling of fullness, bloated abdomen, ice-cold dry hands and a slight feeling of inner cold. Short sweats only after lying down (Day 2). 908.HA.6

Slight relief of nausea after vomiting. After the third vomiting with violent choking and only bilious vomiting, the nausea disappears and shortly after that the headache disappears as well. Afterwards very strong weakness and tiredness, face paleness and freezing, wants to continue lying in the quiet, dark room and sleep a little. Sleep about 1 hour. After that I can get up again, I am still a little weak but very clear in my head and can meet friends, long train ride, a lot of talking. Strange, pleasant clarity in the head, feeling of calmness and balance, slow thinking, more sensitive to outside stimuli and altogether more sensitive. Watery, painless diarrhea in the afternoon. (after 30C) (This prover has a history of migraine, but usually during stress, at the time of the proving in vacation, no stress) (Day 2). 908.HA.6

Sudden pain in head. 36 +

Headache and vertigo, for several days. 36 +

Headache, especially in the temples, together with violent pulsation of the temporal arteries. 36 +

Headache, beginning in the morning, and spreading throughout the whole head, but felt chiefly in the occiput, which seems pressed outwards. 36 +

The nape of the neck seems swollen when touched; the whole posterior portion of the head from about the summit of the occipital bone to the base of the neck seems tense, swollen; the head can hardly be moved. 36 +

Headache commenced generally with confusion and heaviness of the head, and dull undefined pressure in the temporal region, it then gradually increased and extended from the temples forward and backwards, encircling the whole head; usually it increased rapidly to an extreme severity. 36 +

The headache, which is the first symptom, is also the last; in a young girl it continued with great severity for more than a month, almost without interruption. 36 +

Violent persistent headache. 36 +

Severe headaches, **especially occipital**, **worse bending forward** and **bending the head backward.** de Sonnaville *

Symptoms commenced with violent headache, which soon became very intense. 36 +

Violent and constant headache, worse in the frontal region and accompanied with a sensation of tightness and constriction towards the temples. 36 +

Dull headache. 36 +

Pressive headache. 36 +

In brain, severe pressure. 36 +

Painful sawing pain through middle of head, which feels congested. 36 +

Intolerable pain in the forehead. 36 +

Frontal headache, extending over the whole head, but chiefly felt in the forehead, which seems pushed out. 36 +

Constant frontal headache (second day). 36 +

Pressing pain at the forehead and parietal bones. 36 +

A severe pressing frontal headache, as if the brain were compressed, and simultaneously severe palpitations of the heart, with which I am never troubled. 36 +

Weight across forehead. 36 +

Throbbing pain in forehead and temples. 36 +

The attack commences with a vague, dull pain in the temporal region, which gradually extends forward and backward, encircling the head; it becomes exceedingly intense. 36 +

Headache characterized by compression of the temples. 36 +

Sticking in temples. 36 +

Carboneum oxygenisatum

110

Throbbing in temples. 36 +

Dullness and oppression in the crown of the head. 36 +

A small red spot (similar to the streak on the arm), on the right temple, near the outer margin of the orbit. 36 +

Headache and confusion. 311 +

Sensation as if the head is full of air. 918 ˚

Headaches with tiredness and dyspnea. 312 +

Headache, dizziness, muscular weakness, disturbance of gait, paresthesia, breathlessness on exertion. 313 +

Violent, mainly frontal, dragging headache. The brain was diffusely edematous with hemorrhage. 314 +

Brain herniation. 1202 ˚

Cerebral edema. 1202 *

Cerebral hemorrhage or cerebrovascular accident or apoplexy with violent headache with tightness and constriction of frontal and temporal area. 1202 *

Hydrocephalus. 1202 *

Cerebral edema. 1202 *

My eyes would not stop aching and the pain would not quit my **temples**, just lying in the sleeping bag quieted the hammering of my heart. **Splitting headaches**, the nausea, the stabbing pains in my body and eyes, the hot and cold rushes of dizziness. 315 +

Throbbing headaches intermittently; shortness of breath, dizziness, weakness, and intermittent chest pain. Mild respiratory distress, with a respiratory rate of 28 breaths per minute, pulse is 92 beats per minute. 316 +

Throbbing headache with faintness for three months before admission whenever she was in the kitchen. Followed by deep coma. With a temperature of 33°C (rectal) and blood pressure 90/60 mmHg. **Her skin was pink**. Leucocyte count 33,000, platelet count 101, concentration of fibrin degradation products raised, prothrombin time 47 seconds (normal: 11-12 seconds), and blood urea concentration 91 mmol/l (56 mg/100 ml). Abdominal pain, which was accompanied by loose **stools con-**

taining blood. Forty-eight hours after admission the pain had worsened and bowel infarction was suspected. At laparotomy the bowel was seen to be blue but viable. She failed to regain consciousness and it was found that the serum aspartate transaminase activity had been 1,137 IU/l and the prothrombin time 55 seconds in a sample taken six hours before operation. Jaundiced with signs of grade 3 hepatic encephalopathy. Acute tubular necrosis of the kidney and she required hemodialysis. Fulminant hepatic failure. 317 +

Head hot, cool damp forehead (4 p.m.) (Day 5, 200C).908.HA.4

Violent pulsation of temporal artery. 1202

Eyes

Heavy feeling in eyes. (Few minutes after 30C for some time) (Day 1). 908.HA.1

Heavy feeling in eyes, can hardly keep eyes open. (Day 6). 908.HA.1

Right eye discomfort, soreness and sensitive to light. Have to keep squinting that eye. Better with closing the eye and putting pressure on it and keeping lights low or off. (Day). 908.HA.1

Eyes painful better close, worse touch eyes and light. de Sonnaville *

Eyes weak and dim, sunken. 36 +

Eyes wild, staring. 36 +

Eyes weary-looking.36 +

Eyes distorted in the orbits. 36 +

Eyes fixed and insensible. 36 +

Eye hyperemic. 36 +

Eyes half-open, staring. 36 +

The eyes are contracted and sunken. 36 +

Eyes string and protruding.36 +

Idiotic staring at one point. 36 +

Lids and lips bluish-red. 36 +

Carboneum oxygenisatum

112

Vessels of conjunctiva injected. 36 +

Conjunctiva dull red. 36 +

Pupils dilated. 36 +

Pupils somewhat dilated. 36 +

Pupils dilated and insensible. 36 +

Pupils insensitive with coma. 1202 *

Eyes wild staring and protruding with coma. 1202 *

Eyes half open with coma. 1202 *

Pupils dilated with coma. 1202 *

Pupils dilated and not reacting with coma. 1202 *

Left hemiplegia with coma. 1202 *

Pin-pick pupils of both eyes (after 3 hours). 908.HA.9

Pupils contracted, insensible, pupils become insensible to the light, and the conjunctiva to foreign substance. 36 +

Sustained horizontal nystagmus and **intention tremor in both persons**. 318 +

His pupils were dilated and very sluggish. 319 +

Light pain behind the eyes while driving, for 30 minutes (Day 11; 200C) 908.HA.3

Retinal hemorrhage, **papilledema**, **retinopathy**, **optic atrophy**, amblyopia, **scotoma**, **hemianopsia**, **blindness**. 320 +

Dim sight. 36 +

There is something wrong with the eyes, but its nature is eluding. It is almost as if their inner light is dimmed—a dampening of intelligence, charm, and wit (3/10) (second day). 908.HA.9

Floaters in the eyes in the morning on awakening (Day 3, 30C). 908.HA.4

Almost constant dimness of sight and vertigo. 36 +

Dimness of vision, with flickering and fluttering before the eyes. 36 +

Vision obscured. 36 +

Flickering before the eyes, with vertigo. 36 +

Visual acuities were 6/60 right eye and 6/36 left eye. Fundoscopy revealed bitemporal disc pallor with the visual fields showing bilateral paracentral scotomata, less dense on the left than the right optic neuropathy. 321 +

Visual fields revealed bilateral central scotomata and peripheral constriction. Fundoscopy showed bilateral temporal disc pallor. Optic neuropathy. 322 +

He woke up complaining of poor vision, which was documented at 6/18 right and left. Fundoscopy revealed bilateral disc cupping (cup:disc ratio 0.7) and pallor. His visual fields showed a slight centrocecal defect to dim targets optic neuropathy. 323 +

Total blindness. 324 +

Two female patients developed visual deterioration after CO poisoning The patient, a 34-year old female, had no visual complaints on the first three days after the accident. On the third day, she started to complain of memory problems and on the same day started also to complain of blurring of vision. Later on, nausea, vomiting, headache and visual problems gradually increased. [325] +

The patient, a 32-year old female was referred to our hospital on the sixth day of the incident with visual complaints. She reported that she could see almost nothing. She also reported not being able to remember the first four days after the incident. [326] +

A 28-year-old man was exposed to CO accidentally. He was rescued rapidly, but was unconscious for 10 minutes. On regaining consciousness **he could not see**. At 20 hours he could read a newspaper and was discharged "well" at 24 hours, with a retrograde amnesia of 10 minutes and a post-accident amnesia of 4 hours. He remained well until 5 days after the accident when, over several hours, he developed bitemporal headache, photophobia and blurring of vision. The next day he was unable to see beyond perception of light. [327] +

A boy of 13 years and 7 months was admitted in August with blindness and mental deterioration. When the boy he was found that he was totally blind and that he could neither hear nor speak. Both fundi showed recent hemorrhages. He had rhythmic twitching of the left side of the face, there was a left-sided Babinski sign

and both hands were in flexion. Within one week he began to hear and to speak. He could walk after two months, but he remained totally blind. During his five months' stay in the hospital he had occasional convulsive seizures with decerebrate rigidity attitudes. In the previous March, he had been observed at another neurologic hospital where it was noted that he had begun to perceive light. it was noted that he had begun to perceive light. The left upper extremity was held in flexion at the elbow and wrist, and there was bilateral loss of associated movements in the arms. The patient had some difficulty in the performance of skilled acts, which was due in part to weakness and in part to apraxia. He showed associated movements (reflexes) in one arm on gripping the examiner's hand with the other. He had no incoordination and no abnormal involuntary movements. All the deep reflexes were hyperactive, the abdominal reflexes were present, and the Babinski toe sign was positive on both sides. The left upper extremity was hypertonic. There were bilateral pes cavus and some equinovarus. The patient could not recognize small objects, such as a coin, key, pen or watch, and large objects, such as a book, newspaper or telephone, he could distinguish with difficulty. Color perception, on the other hand, was quick and accurate. He recognized not only all primary colors, but such shades as brown and pink. He knew at once the colors of small objects which he could neither name nor tell the form of. He picked out colors on command. He groped in trying to grasp objects, and sometimes collided with persons and things, apparently because he could not estimate distance and probably as a result of a defect in visual spatial discrimination. Speech was unintelligible and dysarthric, but there was no definite evidence either of motor or of sensory aphasia. He showed intellectual deterioration, emotional inadequacy and lack of insight. His mental age, on psychometric examination, was given as 7 years and 8 months and his intelligence quotient as 52. [328] +

Cortical blindness. [329] +

Total blindness. 330 +

A 45-year-old woman visited our hospital complaining of poor vision after CO poisoning. We have confirmed the presence of a point mutation at position 11778 in the ND4 gene of mitochondrial DNA. This case suggests that CO poisoning may precipitate the clinical expression of Leber's hereditary optic neuropathy. [331] +

His pupils were dilated, and would not react to light. 332 +

Visual agnosia: inability to perceive the whole, the Gestalt, visually. Only parts of the whole are perceived, and their correct relation is not recognized. This results in an inability to read, to copy letters and geometric figures and to recognize pictures

or objects on short exposure. Writing is unimpaired. Preceding optic impressions superimpose themselves on subsequent ones, so that proper perception and recognition are interfered with. 333 +

Retinal hemorrhages. 334 +

Retinal haemorrhages with apoplexy or coma. 1202 *

A mother with scattered superficial retinal hemorrhages, and her child with multiple large subinternal limiting membrane hemorrhages, peripapillary hemorrhages, severe venous tortuosity, and **disc edema**. On ophthalmoscopy, we observed prominent bilateral disc edema, severe *venous tortuosity* and engorgement, peripapillary hemorrhages, and multiple large subinternai limiting membrane hemorrhages scattered over both posterior poles and extending into the left macula. 335 +

Hemorrhages were found in the nerve fiber layer of the retina in all five of the patients who had been exposed for more than 12 hours. For approximately two weeks before her initial examination, she noted nausea, headaches, and dizziness, which gradually increased in severity. She was treated for flu-like illness by her internist. At this point, her 61-year-old brother returned from an out-of-town visit. While remaining at home for a two-day period, he first noted a **ringing and loss of hearing** in the right ear. This led to increasing dizziness, followed by headaches, nausea, and finally, episodic vomiting. During the same weekend, a daughter visited for the evening and also began having nausea, dizziness, and headaches. The 61-year-old brother, however, had persistence of the hearing loss in his right ear. Therefore, he consulted an ear, nose, and throat specialist, who concluded that he suffered from nerve deafness on the right side. In addition, retinal hemorrhages were noted in his right eye. At this time, examination of the fundus showed a dense, small, retinal hemorrhage just on the nasal side of the fovea on the right side. There was a less well-defined larger area of hemorrhage just temporal to the optic nerve on the left. The patient's sister was examined at the same visit. The abnormal findings included the dense cataract on the right side and a two-disk diameter area of nerve fiber layer hemorrhage in the upper temporal quadrant of the left eye. [336] +

Four persons were involved in this incident: an 80-year-old man, his 79-year-old wife who had been chronically ill with myeloma, their 49- year-old son, and their 44-year-old daughter. Both parents had been suffering from nausea, poor appetite, dizziness, and headaches for approximately four weeks, beginning Oct 1. The father was thought to have a flu-like illness. These symptoms continued on an intermittent basis for four weeks until the son and daughter visited the parents on the night of Oct 29. They were concerned with the deteriorating health of the parents

and decided to remain overnight. In the morning, the daughter awoke with similar symptoms of nausea, dizziness, and headache; she found her brother unconscious on a lower floor bedroom of the home, and her father, as well, unconscious in his bed. An ambulance was called. The father was pronounced dead in the home and the mother and son were admitted to the hospital. The fundus on the left eye showed scattered nerve fiber layer hemorrhages throughout the fundus. His symptoms included headache, nausea, dizziness, tingling weakness, and numbness of the left arm. Results from his physical examination were, however, essentially normal except for an erythema of the skin of the upper arm. [337] +

There were five persons involved in the third CO exposure incident: a husband and wife in their late 50s, their 30-year-old son, and two friends of the parents who were visiting the home on the night of the fatality. Decreased vision, headaches, nausea, three episodes of vomiting, and mild disorientation. There was a reddish rash over the right wrist and a few flame-shaped hemorrhages were noted around the optic nerve on the left. As her symptoms became gradually more severe, her husband began to have similar symptoms of headache, nausea, and dizziness, and together they assumed that they were suffering a viral or flu-like illness. By Nov 24, the son, who lived elsewhere, became concerned over his parents' health and returned home to evaluate the situation. He slept on a couch on the lower floor of the home. Early on the morning of Nov 25, the mother was awakened by a noise in the living room and found the son unconscious. An ambulance was called. The son was pronounced dead at the scene and taken to the medical examiner's office. The examiner noted the cherry-red appearance of the body and immediately suggested CO poisoning. The Father: Two days later a fundus examination was performed at home. Both eyes showed venous congestion and scattered nerve fiber layer hemorrhages. Mother: Results of an ophthalmic examination on the fourth day of hospitalization were normal except for the fundus findings. Flame-shaped superficial retinal hemorrhages were scattered in all quadrants of both retinas. The retinal veins were engorged and slightly tortuous. [338] +

Weakness in the right upper extremity, complete blindness, and a depression of intelligence. Amaurosis. 339 +

Paralysis of several ocular muscles. 340 +

Ears

Suddenly ear ringing (minutes after 30C for 30 minutes, two times after the 30C) (a recurring symptom, but had been better since taking my chronic remedy 2 weeks ago) (Day 2). 908.HA.1

Earache left morning on waking, better by moving the jaw and pulling on the auricle (Day 2, 30C).908.HA.4

Ears very blocked and squeaked and popping (Day 5). 908.HA.1

Ear, stopped.de Sonnaville *

Crackling in ears (Day 7). 908.HA.1

Hearing

Suddenly ear ringing (minutes after 30C for 30 minutes, two times after the 30C) (Day 2). 908.HA.1

Noise in ears. 36 +

After a short time, confused sounds in the ears, which are exceedingly painful; afterwards there is a continuous dull vibrating, similar to the noise of a wagon, mingled with pulsating sounds, which at first seem to be indistinct and distant, but gradually become stronger; whilst lying in deep stupor, there is an incessant humming, which gradually disappears as consciousness returns. 36 +

Ringing in the ears, with various kinds of illusions of hearing. 36 +

Roaring in ears. 36 +

Troublesome roaring and singing in the ears. 36 +

CO poisoning is one of the rare causes of hearing loss, which may cause reversible or irreversible, unilateral or bilateral hearing loss after acute or chronic exposure. Complained of hearing impairment without vertigo or tinnitus.[341] +

12 patients with hearing loss from acute CO poisoning were studied. The majority of these patients showed asymmetrical sensorineural hearing loss that improved gradually with some damage persisting. A 16-year-old female with acute CO poisoning had bilateral sensorineural hearing loss. The U-shaped audiogram in our case report appeared to be a unique manifestation of hearing loss from CO poisoning. Taniewski and Kugler surveyed 3000 audiograms and found only 19 U-shaped patterns not associated with CO intoxication. A bilateral, symmetrical, U-shaped audiometric curve, however, was observed for 31 of 35 patients suffering acute CO-induced hearing loss. As in our case, the most pronounced loss was observed near 2000 Hz. *Speech discrimination was significantly poorer than pure-tone thresholds*. [342] +

The effect of CO poisoning on the threshold sensitivity of the responses from the auditory cortex, inferior colliculus, and cochlea to acoustic stimuli in guinea pigs was studied. The toxicity of CO is believed to be secondary to tissue hypoxia and is partially reversible. Loss of auditory threshold sensitivity in CO poisoning is most prominent at the auditory cortex. The loss of sensitivity at the inferior colliculus is the next most severe. There is no loss of sensitivity at the cochlea. [343] +

A 53-year-old man presented to an urgent care centre with sudden deafness. Lightly disoriented and unable to provide much detail about the onset of his symptoms but recalled driving to the urgent care centre. He complained of dull bifrontal headache and ringing in the ears. He had difficulty in drawing a clock. An MRI of the brain revealed an abnormal signal in the globus pallidus bilaterally. [344] +

An explosion occurred in a Japanese coal mine and over 400 miners died as a result of CO poisoning. The 14 survivors were hospitalized; initially, the patients suffered from severe acute symptoms of CO poisoning, such as unconciousness. Later examination revealed central nervous system manifestations, i.e. amnesia, inability to write complicated Japanese characters, inability to read or count, and clouded sensorium. In addition, one patient demonstrated tremor of the upper extremities.In general, movements were slow and faces were mask-like. Ophthalmological symptoms which had been reported previously were not noted. Vertigo, nausea, vomiting and tinnitus were the presenting signs in one case. In 7 of 14 patients, nystagmus was noted in the sitting position. Nystagmus was significant in 2 of these 7 patients, while the others showed fine nystagmus; tinnitus was found in 5 patients. Neuritis of vestibular and auditory nerves. In a detailed report of 15 cases in another series, it was reported a vestibular lesion in two third of the patients, and a cochlear lesion in one third. [345] +

Eighteen hours following admission, he regained consciousness but was suffering with amnesia, blurred vision, deafness and tinnitus. On day six, he showed a bilateral moderate to severe sensorineural hearing loss. 346 +

Bilateral hearing loss, increasing dizziness and lethargy. As early as 1948, a report by Lumio documented a 78 per cent incidence of sensorineural hearing loss among 700 cases of chronic CO exposure. Since then however, reports have been limited to those patients presenting with hearing loss in association with acute CO poisoning. In 1967 Garland and Pearce reported four cases of accidental CO intoxication with slight to moderate hearing loss, which apparently improved over 24 hours. Morris reported a case in 1969 of severe bilateral sensorineural hearing loss, that partly improved over a period of 11 months. Young *et al*. reported that in an animal

model carbon monoxide exposure potentiated high frequency auditory threshold shifts induced by noise. 347 +

17-year-old male He was found at home and was unresponsive due to CO poisoning. A chest X-ray showed evidence of a small focal consolidation on the lower right lobe. There was evidence of mild bilateral calcification suggesting pulmonary edema. Overall he had generalized weakness and was flaccid on the left with an increase in tone on the right. He was diagnosed at admission with gait dysfunction, dysphasia, and generalized weakness. He was diagnosed at this time with mild mixed aphasia and mild-moderate cognitive deficits. Bilateral hearing loss of sensorineural origin. [348] +

Disturbance of cochlear and vestibular system. 349 +

Nose

Burning dryness in back of the nose, behind the nose and in the throat, disappears in 1-2 hours (Day 7). 908.HA.3

Burning in the back of the nose, behind the nose in the afternoon for aprox. 15 minutes (Day 11). 908.HA.3

Dry felling inside nose, like velvet in the evening in bed, urge to clean, but no discharge (Day 4 and 5). 908.HA.7

Nose like blocked, but can breathe through the nose (Day 4, 200C). 908.HA.4

Blocked left nose morning on waking (Day 5, 200C). 908.HA.4

Blocked nose, dry nasal mucous membranes in the morning for some days. (after 200C) 908.HA.6

Nose obstructed. de Sonnaville *

Crackles occasionally in the nose (Day 4, 200C). 908.HA.4

Occasional sneezing in the forenoon (Day 2, 200C). 908.HA.4

Flow rhinitis 2 times in the forenoon (Day 4, 200C). 908.HA.4

Nasal root every breath felt. (Day 6, 30C). 908.HA.4

Coldness of breath with face cold to touch, coldness of hand and nose. 1202 *

Violent inflammation of the nose and throat, which makes swallowing very difficult (second day). 36 +

Bleeding from the nose. 36 +

Dull sense of smell and taste. 36 +

Loss of smell and taste. de Sonnaville *

Things smell bad. de Sonnaville *

Prickling in the mucous membrane of the nose, sneezing and profuse secretion of mucus. 908 +

Face

Face feels numb, as if had drunk too much wine. It is un-wielding and unresponsive to facial expression (second hour). 908.HA.9

Left-sided headache, pressing pain on the face up to the upper jaw on waking in the morning, feel the left maxillary sinus in the depth (Day 3, 30C). 908.HA.4

Sensitive maxilary sinus, feel the maxilary sinus (Day 2, 200C). 908.HA.4

Sensitive maxillary sinus more left side. I feel the maxilary sinus (Day 6, 30C). 908.HA.4

Pressure on the face in the region of maxilary sinus (Day 4, 200C). 908.HA.4

Pulling pain in the face, forehead, around the eyes and in the maxilary sinus. Daily together with the headache. Pulling from the top of the head over the face. (after Day 10, 200C). 908.HA.4

Headache and facial pain better after getting up, better by movement (swimming) and better after the first cup of coffee at around 9 a.m. (after Day 10, 200C). 908.HA.4

Cracks in corners of the mouth (Day 12 til 16, 200C). 908.HA.3

Looks anxious. 36 +

Face pale. 36 +

Ashen appearance. de Sonnaville *

Face pale. 1202 *

Pale face, warm to the touch. 36 +

Very pale face, continued for several days. 36 +

Face pale, red, cyanotic, and puffy. 1202 *

Distorted face. 1202 *

The lower lip immensely swollen and turned outwards. 36 +

Lower lip would turn outward and swell transiently, in a patient with COVID-19 pneumonia, rapid breathing and shortness of breath with exertion. Sebastian *

Lips bluish. 1202 *

Face livid. 36 +

The complexion had assumed the livid hue of death. 36 +

Cyanotic. 36 +

Face red. 36 +

Face red (four children). 36 +

Face red and puffy. 36 +

Face bluish-red. 36 +

Face tumid. 36 +

Face puffy and reddish-brown. 36 +

Features distorted. 36 +

Convulsions of the facial muscles. 36 +

Lips bluish. 36 +

Lips and tongue rosy-red. 36 +

Tongue rosy red. 1202 *

Tongue red. 1202 *

Clenched my jaws very firmly during nap (Day 4). 908.HA.7

Clenched jaws several times (Day 5). 908.HA.7

Carboneum oxygenisatum

122

Jaws firmly clenched (and others). 36 +

Trismus. 36 +

Trismus with coma. 1202 *

It is of service in **trismus**, when the jaws are firmly closed. 100 *

Tightness of jaw. 1202 *

Trismus, with epileptic convulsions. 36 +

Pale face. 350 +

Face pale. 36 +

Pale face, warm to the touch. 36 +

Very pale face, continued for several days. 36 +

Nausea, generalized weakness, dizziness and a "fuzzy feeling" throughout her body. The feelings of nausea and weakness increased until she collapsed. Observers noted that she was unconscious and shivering; **her face was very flushed.** The redness of her face faded into blotchy red patches. Six months later the girl presented for neurologic consultation. In the intervening months her marks at school had dropped noticeably but her parents thought that social activities and other interests were responsible. She was regarded as normal by her parents and herself. 351 +

Cherry-red lips. 352 +

Cherry-red color of lips. 353 +

Pale and flaccid, with perioral cyanosis and shallow respirations. 354 +

CO coma, the lips should be rosy red and there should be an appearance of perfect health, but in industrial cases this seems to be rarely true. It is much more common to find pallor, with red blotches on the skin, and blue lips. 355 +

Mouth

Coldness of breath with face cold to touch, coldness of hand and nose. 1202 *

Small canker sore inside left cheek, lasted only one day. (Known symptom, but usually more severe and lasted longer) (Day 1). 908.HA.1

Paralysis of the tongue. 36 +

Tongue feels thick and un-wielding (after 3 hours). 908.HA.9

Mouth is dry with no desire to drink (after 3 hours). 908.HA.9.

My mouth is dry, my throat is dry with an aversion to drink (after 6 hours). 908.HA.9

Dry mouth. de Sonnaville *

Mouth drawn. 36 +

Froth from mouth. 36 +

Froth from the mouth. 1202 *

Foaming at the mouth. 36 +

Salivation, then nausea and vomiting. de Sonnaville *

While eating, flow of a slightly acid water into the mouth, which mingles with the food without causing disgust. 36 +

After supper, the mouth is lined with mucus so viscid that, on trying to spit it out, it sticks to the lips. 36 +

Excessive sensitiveness of taste and smell, which lasts four days, an goes on diminishing, until, in six days, these two senses are duller than formerly. 36 +

Appetite as usual at dinner, but bread and all sort of food had a foul taste, even sugared rice-cake (second day). 36 +

Strange taste in my mouth, not clear what kind of taste, just different. (Day 1). 908.HA.1

Dull sense of smell and taste. 36 +

Loss of smell and taste. de Sonnaville *

An elevated amylase level has been an incidental observation among CO victims in perhaps 40% of cases. The salivary gland has been shown to be the source of these observed serum amylase level**.** 356 +

Throat

Dryness of the throat. 36 +

Post-nasal drip and mild cough with slightly throat tickle. Kept clearing my throat frequently. (Short time after 30C for one hour) (Day 1). 908.HA.1

Between 4-5 p.m. a burning sensation in the trachea (Day 8). 908.HA.3

Between 4-5 p.m. a burning sensation in the throat (Day 9). 908.HA.3

Dryness of throat on waking (Day 10, 200C). 908.HA.3

Soreness of throat a few hours in the morning and in the evening, agg. empty swallowing, ameliorated while eating (Day 17, 200C). 908.HA.3

Pain throat: worse swallowing, at night, eat, **talking, better drink,** cool drink. de Sonnaville *

Sensation of a dry, swollen throat with pain extending to the ears on empty swallowing; no pain or discomfort on swallowing food (after 3 hours). 908.HA.9

Pain extending to ears. de Sonnaville *

Slight sore throat both sides equally. Swallowing doesn't seem to influence it. Warm tea makes it better for a while (Day 5, 30C). 908.HA.8

Throat pain worse from warm tea.de Sonnaville *

Pain in the throat, from swallowing saliva; lasted all night. 36 +

The sore throat continues, and extends to the right ear (second day). 36 +

Violent burning pain in the fauces. 36 +

Burning sensation of throat. 357 +

Acute hyperthyroidism 358 +

Stomach

Unquenchable thirst for small sips often (icy cold water) with dry mouth not better by drinking (seventh day). 908.HA.9

In the afternoon, three hours before supper, sudden paroxysms of hunger, which soon ceases without eating (second day). 36 +

Desire for beer (Day 5). 908.HA.3

Desires sour things. de Sonnaville ˚

Desire for carbonated drinks. Anand ˚

No inclination to eat. 36 +

Anorexia. 36 +

No appetite, but food relished well. 36 +

Disgust for everything. 36 +

Felt sick, and retched once or twice. 36 +

Stomach gurgling. 908.HA.2

Repeated belching. 908.HA.2

Discreet nausea with constant feeling of hunger, but eating does not improve the nausea. (after 30C). 908.HA.6

Slight nausea morning on waking which persisted for a few hours then went away. Eating did not affect it. (Day 2,3, C6). 908.HA.8

Slightly nauseous, ate only a small piece of bread for lunch, afterwards I felt a bit stronger and less nauseous (Day 3, 30C). 908.HA.8

I didn't feel like eating, but still had dinner and again felt better afterwards, stronger and not nauseous (Day 3, 30C). 908.HA.8

Nausea in the morning after rising, eating breakfast made it a little better (Day 4, 30C). 908.HA.8

Nausea got worse in the evening, but I ate against my inclination and was surprised to feel much better afterwards (Day 4, 30C). 908.HA.8

Nauseous in the morning a few minutes after rising, better by eating, then returns about 3 hours after the last meal (Day 5, 30C). 908.HA.8

Nausea from smell of cooked sausages (Day 7). 908.HA.3

Nauseous with the heat. de Sonnaville ˚

Slight nausea. 36 +

Nausea and vomiting. 36 +

Nausea and vomiting every now and then; the stomach could bear only liquids in very small quantities. 36 +

Repeated vomiting. 36 +

Vomiting after meals. 36 +

Vomiting, caused by the smallest quantity of food. 36 +

The stomach was so irritable that everything taken was immediately vomited (second day). 36 +

Pressing on abdomen causes vomiting of a yellowish, almost fecal-like-fluid. 36 +

Digestion disturbed. 36 +

Heartburn, worse after eating or drinking. de Sonnaville °

Very severe and obstinate pain in the epigastric region. 36 +

Vomiting, diarrhea, faintness and unsteady gait. 359 +

Frothy vomitus. 360 +

Nausea or vomiting. 361 +

Stomach ulcers. 362 +

Nausea, vomiting, and abdominal pain after their evening meal. 363 +

Abdomen

Pains in the abdomen. 36 +

The abdominal cramping with diarrhea vanished instantly. (curative). 908.HA.9

Rumbling abdomen, and then dark diarrhea. de Sonnaville *

Abdominal pain before stool, with rumbling in abdomen, then diarrhea, better after stool. de Sonnaville °

Abdominal pain, followed by diarrhea. de Sonnaville °

Cramping pain, better pull up legs, better pressure on abdomen. de Sonnaville °

Pain abdomen from coughing. de Sonnaville °

Abdominal pain, nausea. de Sonnaville *

Frequent crying out that they had pains in the abdomen; it, however, was not distended or tense. 36 +

Violent pains in the bowels, **liver enlargement**.364 +

Hepatomegaly. 365 +

Hyperglycemia. 366 +

Elevated liver enzymes. 908 +

A 65-year old patient admitted with CO poisoning developed acute pulmonary edema during treatment with hyperbaric oxygen. He became very short of breath and was immediately removed from the hyperbaric chamber. Severe pulmonary edema was diagnosed and confirmed on chest X-ray. About 12 hours after intubation, his blood pressure and central venous pressure started to drop rapidly while there was progressive distension of the abdomen with *ileus.* After initial recovery he developed extensive intestinal ischemia which rapidly led to death. A plain abdominal X-ray showed widespread small bowel distention with fluid levels. Autopsy showed advanced ischemic necrosis of all abdominal organs including the liver, small and large bowel. There was no significant atheroma or thrombotic occlusion in the mesenteric vessels. It is suggested that intestinal vasoconstriction due to left ventricular failure made the gut much more vulnerable to the hypoxic effects of carbon monoxide than the brain and heart. [367] +

Stool and Rectum

Large soft easy bowel movement (usually very constipated) (Day 2,5,6,7,8). 908.HA.1

Watery, painless diarrhea in the morning. (after 30C). 908.HA.6

Involuntary evacuations. 36 +

Stools thin, painless. 36 +

Fullness in abdomen from constipation, worse at night (Day 10). 908.HA.3

Stool incomplete (Day 10). 908.HA.3

Constipation with no urging for stool (Day 2, 200C) 908.HA.3

Constipation, urinary or fecal incontinence. **368** +

Urinary and fecal incontinence with loss of consciousness. **369** +

Urinary organs

Urinary frequency in early afternoon. I had to urinate every few minutes for a few hours. No dysuria or abdominal pain. Symptoms eased in a couple of hours. (Day 9). 908.HA.1

Sick with fever, backache, abdominal pain and pain when urinating, frequent urging to urinate but no cystitis in pregnant woman. de Sonnaville °

Woke from sleep around 2:30 A.M. with urinary frequency. Had to urinate every 5 to 15 minutes for a couple of hours. Fell back to sleep at 4am and urinary frequency was normal. No dysuria or abdominal pain. 908.HA.1

Burning sensation at the urethral opening, which persists throughout the day. (One hour after taking the third dose 30C) (Day 4). 908.HA.5

Pains in kidneys. 36 +

Paralysis of the bladder. 36 +

Bladder remains paralyzed a long time. 36 +

Paralysis of the bladder lasted nine days after the attack. 36 +

Involuntary evacuation of urine and feces. 36 +

Urinary and/or fecal incontinence. 370 +

Involuntary urine ad stool with coma. 1202 *

Urination from the first became more and more scanty. 36 +

The secretion of urine is increased; carbonated waters are the best diuretics. 36 +

The specific gravity showed a corresponding diminution. 36 +

Kidney

Rhabdomyolysis and myoglobinuria secondary to anoxia was the probable cause of acute renal failure. Tense swelling of legs noticed from day 2 of illness, more proximally than distally, which increased progressively and was suggestive of

rhabdomyolysis. His BUN was 139 mg/dL, total bilirubin 0.7 mg/dL, calcium 7.7 mg/dL, phosphorus 10.5 mgidL, uric acid 8.4 mg/dL, Na 133 mEq/L, K 5.5 mEqiL, CI 100mEq/L, CPK 21,776 IU/L (MB 132IU/L, MM 21644 IU/L), SGOT 810 IUI L, SGPT 590 IU/L, alk phos 104 IUIL, and LDH 2119 IUIL before start of dialysis. 371 +

A 30-year-old male hod-carrier pain in both flanks and numbness in the right leg. Rectal temperature was 34.5°C and blood pressure was unrecordable. The circumference of the right thigh was 7 cm greater than that of the left thigh and knee movements were impaired, with sensory loss over the lateral aspect of the right leg. profound metabolic acidosis; potassium on admission was 8.6 mmol/L with creatinine of 377 umol/L and creatine kinase of 280,000 IU/L (reference range up to 150). He became progressively oliguric with rising creatinine. During his convalescence, he became aggressive and difficult to manage.[372] +

A 40 year-old male cyanotic he became increasingly acidotic. Creatine kinase was elevated at 3,493 IU/L and eventually ended up with acute kidney failure. [373] +

Acute renal failure. 374 +

CO poisoning in a 37-year-old man was complicated by neurologic damage, skin changes, muscle necrosis and nonoliguric renal failure. The relation between nontraumatic rhabdomyolysis and acute renal failure in CO poisoning is reviewed. Numbness and weakness of the right arm and weakness of the right leg. There was slight tenderness and swelling of the entire right arm, and a reddish-purple petechial rash extended along the dorsal and volar surfaces of the right forearm; it was most pronounced over the areas of muscle tenderness. The right arm and leg were extremely weak and areflexic. Sensation of light touch and pinprick was decreased on the right side of the body. Over the next 3 days the flexor and extensor compartments of the right arm swelled greatly and the patient experienced a steady severe frontal headache and steady midabdominal pain. Sensation of pinprick was decreased on the dorsum of the right foot and right hand, and he complained of numbness in the fingers of the left hand. The skin of his arms was **reddish-purple,** mainly over the areas of muscle tenderness, and in the next few days it became brawny. Within 4 days the serum creatinine concentration had climbed to 10.4 mg/dL. On the third and fourth hospital days marked hyperkalemia necessitated the use of a cation exchange resin administered in an enema. 375 +

Severe pain in her back and lower limbs on recovering consciousness and was continually crying out. Persistent severe back pain. The next days he passed no urine. Muscle necrosis was suspected because of the continued back pain and

tenderness. Groaning and shouting because of headaches and pain, with extreme tenderness in the muscles of her back and lower limbs. She was very emotional. Examination revealed acute tenderness of all muscles, particularly over the lower thorax, thighs, and calves. There was a swelling (5 by 4 in.: 12.5 by 10 cm.) of the right paravertebral muscles over the line of the 10th rib. This was exceptionally tender, there being superficial pitting edema. Reflexes were absent in both knees, diminished in the ankles, and equivocal in the plantars. There was gross weakness of the back and lower limb muscles. intermittent generalized epileptiform fits. Muscle necrosis, acute renal failure, mental changes, a radiculomyelitis of L2 to 5, and probably myocardial damage. 376 +

A 20-year-old man was admitted comatose due to CO poisoning and developed massive, progressive edema of both legs within a few hours. He was found comatose in a sitting position where he had been for nearly 9 hours. His urine was red-brown in color. Diminution of lower extremity pulses and laboratory evidence of marked muscle damage (serum creatine phosphokinase of 142,000) prompted bilateral lower extremity fasciotomy. Frank myoglobinuria ensued. Fasciotomy was performed promptly, but return of musculoskeletal function was incomplete. [377] +

An elderly lady was hospitalized because of diffuse myalgia and weakness, and muscle biopsy showed nonspecific rhabdomyolysis. Renal failure also developed on the day of admission but resolved spontaneously, as did her symptoms. The admitting serum creatine phosphokinase was 29,000. Eight hours after admission the urine was noted to be amber in color and positive for hemoglobin. The BUN rose to 40 mg/dl from an initially normal value, and the CPK reached a level exceeding 60,000. We found 13 other cases of myonecrosis associated with CO poisoning in the English language literature. [378] +

Glycosuria, proteinuria, hematuria, myoglobinuria, acute renal failure, abortion, stillbirth, menstrual disturbance, reduction in weight of testes and in number of spermatozoa. 379 +

Acute renal failure is a severe complication of acute CO poisoning which, combined with other organ lesions, may result in lethal outcome. In all vague cases of ARF with nontraumatic rhabdomyoiysis, CO poisoning should be considered as a possible etiologic factor. The diagnosis is made on the basis of several simple laboratory tests: determination of carboxyhemoglobln concentration, demonstration of myoglobin in urine or pigment granulated cylindres in urinary sediment, positive orthotoiuidine test, and high CPK values originating from skeletal muscles. 380 +

Severe muscle necrosis and severe muscle damage leading to acute renal failure. Elevated myoglobin (>1,000.00 ng/mL; normal, 0-110 ng/mL), creatine kinase (CK)-MB (264 ng/mL; normal, 0.0-5.0 ng/mL), CK (99,053 IU/L; normal, 56-244 IU/L), blood urea nitrogen (BUN; 57.5 mg/dL; normal, 8.0-20.0 mg/dL), creatinine (4.8 mg/dL; normal, 0.6-1.2 mg/dL), lactate dehydrogenase (11,200 IU/L; normal, 200-400 IU/L), C-reactive protein (CRP; 44.47 mg/dL; normal, 0-0.5 mg/dL), white blood cells (14.54×103μL; normal 4.0×103-11×103/μL) and erythrocyte sedimentation rate (43 mm/hour; normal 0- 10 mm/hr). On urinalysis, urine appeared dark brown and showed erythrocyte 4+and myoglobinuria. Urine output was less than 50 mL/hour. The patient was unable to urinate, and had sciatic nerve palsy associated with rhabdomyolysis, acute renal failure and compartment syndrome. Severe pain, increasing swelling in the buttock. In a review of 250 cases of cause of rhabdomyolysis, 8 were due CO poisoning. CO-induced muscle necrosis may lead to rhabdomyolysis—one of the most common causes of acute renal failure. Compartment syndrome resulting from rhabdomyolysis associated with non-traumatic causes is a very rare condition. 381 +

The three patients developed severe rhabdomyolysis complicated by compartment syndrome. The patient who died developed compartment syndrome in three extremities. Two patients developed anuric acute renal failure due to acute tubular necrosis as shown by renal biopsy. Two patients developed the full-blown picture of systemic capillary leak syndrome. Gross and microscopic examination of specimens taken at autopsy showed extensive necrosis of all skeletal muscles, myocardial necrosis and acute tubular necrosis affecting both kidneys. Generalized edema and severe muscle swelling of the entire body with compartment syndrome affecting the right arm, right thigh and left calf. The limbs were swollen, very tense, pale and cold, and arterial pulsation was absent. 382 +

His pulse was 112 bpm and regular, BP was 90/60 mm Hg, generalized edema and severe muscle swelling over all the body. Swelling in the left leg muscles was severe enough to cause compartment syndrome. The leg was painful, swollen, very tense, pale and cold. Arterial pulsation was absent, sensations were impaired and muscle power was diminished below the knee. Acute renal failure with severe oliguria. Extensive skeletal muscle necrosis. Myocardial specimens showed extensive myocardial necrosis acute tubular necrosis affecting both kidneys. 383 +

A case of CO poisoning with severe myonecrosis and acute renal failure (ARF) is presented with extremely high values of CPK but with preservation of myocardial tissue. The blood pressure on admission was 50 mm Hg systolic by doppler, but increased to 90/50mm Hg. On admission, the serum sodium level was 141, potassium level was 6.1, chloride 109, and bicarbonate 15 with an arterial pH of 7.056.

The blood urea nitrogen was 39 and the creatinine 2.8. The creatinine phosphokinase (CPK) was 9,000. Tachycardic and tachypneic. Creatinine and BUN steadily increased and nonoliguric renal failure progressed. Gradually progressive and severe lower extremity and scrotal edema and severe lower extremity muscle weakness. Severe myonecrosis was clearly evident scans. Laboratory data demonstrated the CPK to be elevated initially, but reached extraordinarily high levels, peaking at 318,000 with simultaneous elevations of serum glutamic oxaloacetic transaminase (SGOT) and lactic dehydrogenase (LDH). The BUN peaked at 103, and the creatinine peaked at 7.8. The urine turned red with no red blood cells seen microscopically, and the urine myoglobin was measured at 480,000 units. The renal failure was felt to be secondary to myonecrosis and myoglobinuria; it gradually resolved without dialysis. Intervening hypoalbuminemia and anasarca. The patient persisted with bilateral lower extremity weakness, and he was discharged to a rehabilitation facility. The patient regained some use of his lower extremities, and was able to ambulate with a cane, but remained with severe bilateral lower extremity weakness and severe lower extremity muscle wasting. Skeletal muscle is particularly sensitive to CO poisoning, which may result in a severe, disabling myonecrosis. This toxicity stems primarily from two CO effects apart from the decreased hemoglobin carrying capacity of oxygen. First, CO may inactivate cytochrome oxidases, which are vital to maintain aerobic respiration within working muscle cells. Second, CO binds with myoglobin thus interfering with the normal binding of oxygen to myoglobin, which serves as an important oxygen reservoir within the muscle tissue itself . 384 +

Renal failure may occur due to rhabdomyolysis and hypoxia. Serum creatinine to 842.4 μmol. The CPK activity was continuously monitored; its highest value was 2500. 385 +

Acute renal failure due to muscle necrosis. Skeletal muscle necrosis leading to acute tubular necrosis of the kidneys an abnormal pigment, described as hematoporphyrin, was found in the urine reviewed of 30 cases of muscle necrosis in CO poisoning; the affected muscles were painful and swollen, gangrene in a lower limbs; brawny edema of the right shoulder, upper arm, and thorax, and muscle necrosis. Sacral and scapular lesions were superficial pressure sores. An extensive soft-tissue swelling, with edema of the overlying skin, was observed to involve the inner side of the right thigh and to extend above the inguinal ligament. There was extreme local tenderness. After the onset of oliguria, acidotic breathing (deep and lobored). Both kidneys enlarged and tender. The blood-urea level had risen to 325 mg/dl. Oliguria vomiting was still frequent. On autopsy muscle had “fish-flesh” appearance. Meninges and cerebral hemispheres were congested, and the brain was

edematous. Bilateral terminal congestive changes in the lungs. Acute congestion of the liver was present. The kidneys were pale and swollen. 386 +

Urine

Increased urinary elimination of iron. 387 +

Urine strong odor and darker than usual. (Day 4, Day 5). 908.HA.1

Urine contained sugar. 36 +

Sugar is found in the urine. 36 +

Urine contains a trace of sugar. 36 +

Male

Scrotal edema. 388 +

Female

Burning entire perineum including anus and labia when showering and soaping (Day 2). 908.HA.5

It has long been believed, since Roman days in fact, that the smoke from extinguished candles would bring about premature labor, and Lewin believed that there is evidence to show that while slight CO poisoning in the mother may produce no symptoms striking enough to arouse attention, but it may be quite enough to kill the child in utero, and that such poisoning is probably fairly common in industries where women are employed and where the air is vitiated with CO. He has known of an instance of this sort, a pregnant woman working on a printing press which was driven by a gas motor. Some accident allowed gas to escape, she was seized with symptoms of CO poisoning, and aborted. 389 +

Severe acute exposures to CO caused fetal death or toxic effects, including anatomical malformations and functional alterations in psychomotor and mental development have been reported. A 28- year-old woman who was 20 weeks pregnant developed a severe headache, nausea and dizziness during a 6-h exposure in the restaurant where she worked. She received medical attention (including administration of oxygen) and delivered a normal infant 4 months later. A carbon monoxide leak, from the furnace, was confirmed by public health authorities. It was noticeable that many early reports came from Europe where coal gas was introduced into households in the mid-1800s. Reports of CO exposure due to coal gas in households were found in the literature through to the mid-1940s. More recent exposures

are largely attributed to furnace and stove leaks. It is clear from the summarized case studies that severe exposures to CO during pregnancy can cause fetal death or serious toxic effects. Toxic effects included anatomical malformations and functional alterations in psychomotor and mental development. Autopsies of stillbirths showed that the central nervous system was a target for CO induced damage at any time during pregnancy. Although it is conceivable that the reported adverse fetal outcomes were related to factors other than CO exposure (e.g. maternal health, infectious agents), this is unlikely as many authors noted that the pregnancies had been unremarkable, except for the CO exposures. [390] +

We reviewed the cases of all patients with CO poisoning during pregnancy admitted to two teaching hospitals in Salt Lake City during a two-year period. Patients 1, 2, and 3 were associated with maternal and fetal survival, and patients 4, 5, and 6 were associated with maternal survival but fetal death. Two fetuses were delivered stillborn within 36 hours after exposure. One fetus remained alive in utero for 20 weeks and was delivered nonviable at 33 weeks gestation *with multiple mor- phologic anomalies*. All of the women recovered fully and were neurologically normal at the time of discharge. [391] +

Cerebral palsy in a baby born of mother who died at 32-second week of pregnancy of CO poisoning. [392] +

Respiratory organs

Respiration

Compelled to take deep breaths for no reason. Was not short of breath, but found I kept taking very full and deep breaths throughout the day. (Day 6). 908.HA.1

Compelled to breath deeply spontaneously in morning when first woke up and throughout the day. I am able to take deeper breaths now than before the remedy. I can expand my chest in all directions, pleasant feeling. (Day 8). 908.HA.1

Need to take a deep breath, a couple of deep breaths.908.HA.2

Inclination to breath deeply (immediately after first and second dose) (Day 1 and 2). 908.HA.5

Respiration is noticeably slower; don’t seem to need to take a breath; when breathing, it’s slow and shallow; utterly calm; pulse 108 (first hour). 908.HA.9

Respiration slow. 1202 *

Breathing is a somewhat shallow 10 bpm and feels like not getting enough air (2/10). Want to sit outside in the shade in the wind, which does not help this air hunger. Taking a deep breath does not help (second day). 908.HA.9

Difficult to breathe deeply, irritation of cough after deep inhalation. (Healing symptom). 908.HA.5

Difficult respiration on ascending. (Healing symptom, permanent better). 908.HA.5

Difficult respiration on exertion. (Healing symptom, permanent better). 908.HA.5

Shortness of breath, **cough worse breathing** and **talking**, **pain in lungs**, **not being able to breathe properly**, **severe headaches**, **especially occipital**, **worse bending forward** and **bending the head backward.** de Sonnaville *

Shortness of breath, cough, worse breathing, talking, lying down. de Sonnaville *

Out of breath very quickly. de Sonnaville *

Difficulty breathing deeply. de Sonnaville *

Shortness of breath from little exertion, with high heart rate. de Sonnaville *

Difficult breathing better fresh air, fanned. de Sonnaville °

Worse bending forward, lying on side. de Sonnaville *

Worse moisture and warm. de Sonnaville *

Worse after dinner. de Sonnaville *

Worse pressure of clothing (bra). de Sonnaville °

Respiration difficult worse cold air, talking, dress up. de Sonnaville °

Respiration difficult with pain back, dorsal. de Sonnaville °

Mild cough, little mucus. de Sonnaville °

Cough worse talking. de Sonnaville °

Need to take a deep breath. de Sonnaville *

Band around chest. de Sonnaville *

Cough worse exertion, talking. de Sonnaville *

Cough worse geting out of bed, turning in bed, moving, taking a deep breath, inhaling. de Sonnaville °

Expectoration sweet. de Sonnaville °

Tickling cough better after expectoration. de Sonnaville °

Worse tension, excitement. de Sonnaville °

Worse lying flat. de Sonnaville °

Great weakness, extreme shortness of breath and could not talk or take 3 steps without being out of breath. Borg *

Suddenly developed extreme shortness of breath. Struggling to breathe. Borg *

Rattling of mucus in air-passages. 36 +

Bloody mucus is raised from the bronchi. 36 +

Respiration audible, almost rattling, slow, stertorous. 36 +

Respiration rattling. 36 +

Respiration rattling, now and then intermitting. 36 +

Expired air of a peculiar smell. 36 +

The expired air felt, to the back of the observer's hand, cooler than usual. 36 +

Respiration is for a long time quiet, but afterwards it becomes accelerated, frequently with extraordinary energy and rapidity; expiration is quick, inspiration deep, rattling; latter there occur periods of complete intermission, followed by four or five inspirations. 36 +

Respiration slower. 36 +

Respiration slow. 36 +

Respiration slow, frequently interrupted by yawning and sighing. 36 +

Respiration very soon becomes slow and stertorous; breathing now rapidly, now slowly (four children). 36 +

Respiration 24 (after one hour). 36 +

Respiration 20 to the minute. 36 +

Respiration short and rapid. 36 +

Expiration greater than inspiration. 36 +

Respiration oppressed. 36 +

Chest pain was very bad, enormous pressure!! As if a horse was sitting on it. A lot of muscle pain, just kept turning in bed. Wanted to die! Reliving forgotten pneumonia when he was 7 years old. Was very very lonely then. Transformation, part of his depression is over now. de Sonnaville *

Respiration difficult and interrupted. 36 +

Respiration very labored. 36 +

Somewhat impeded respiration. 36 +

Sense of suffocation. 36 +

Burning in her lungs with chest oppression. de Sonnaville *

Pain attachment of sternum and between the shoulder blades (cramping, sore) worse coughing. de Sonnaville °

Constriction of the chest. de Sonnaville °

The 28-yr-old female blood pressure fell below 90 mm Hg systolically, HR: 105/min, O2 saturation: 92%); apart from elevated hepatic enzymes (AST 77 U/L, ALT 85 U/L, GGT 98 U/L; The first chest x-ray showed diffuse, confluent infiltrates with a positive bronchopneumogram (left more pronounced than right), indicating severe toxic lung edema. [393] +

Respiration was very much oppressed, with very great desire to lie down. 36 +

Weak, could not get up. 36 +

Respiration 26; on deep inspiration stitches in the lower portion of the right half of the chest. 36 +

Dullness in the lower portion of the right side of the chest; respiratory murmur impaired, with fine rales; through the other portions of the lungs were coarse rales with increased respiratory murmur. 36 +

Respiration irregular and superficial. 36 +

Breathing labored, blowing, and irregular. 36 +

Respiration rapid and sonorous, resembling a groan more than snoring. 36 +

Respiration inaudible at a short distance, extremely short and suffocative, with intervals of suspension. 36 +

Asphyxia and an increase of the pulse from 73 to 137. 36 +

Found in a comatose condition; afterward pleuro-pneumonia of long duration. 36 +

Stertorous respiration, pneumonia on the right side. 36 +

Dryness and scraping in the throat, causing cough. 36 +

Difficulty breathing, constant chest pressure. Hoarse, dry airways. de Sonnaville *

Dry throat, scraping. de Sonnaville *

Hoarseness. de Sonnaville *

Stertorous breathing. 36 +

Sense of a burden on the chest. 36 +

On breathing, feeling as if a heavy load on chest. 36 +

Short, dry cough as if chocked myself at night between 12 and 3 a.m. during sleep (Day 3, 5). 908.HA.3

Dry cough, worse move. de Sonnaville *

Short spasmodic cough. 1202 *

Dry cough at 3.30 p.m. (Day 17). 908.HA.3

Dry cough in the evening from tickling in the throat (Day 7). 908.HA.3

Episodes of dry cough from tickling in the throat (Day 14, 15). 908.HA.3

Dry cough from dryness in the throat around 5 p.m. (Day 2, 200C). 908.HA.3

Dry cough from dryness and tickling in the throat from time to time (Day 12 til 16, 200C). 908.HA.3

Dry, barking coughs several times a day suddenly without cause since February before the proving, so for 5 months. This cough disappeared during the proving completely. A corona test in May 2020 was negative. 908.HA.4

Short cough, oppression, dyspnea. 36 +

Roentgenologic abnormalities were observed in 18 cases of CO poisoning. The ground-glass appearance was the most common finding, usually representing the initial manifestation of acute carbon monoxide poisoning. This was observed in 11 cases: 6 cases as the only manifestation. The ground-glass appearance was the most common roentgen finding of acute CO poisoning, usually representing the initial chest manifestation. This lesion presents as a soft, veil-like, homogeneous density occurring predominantly in the peripheral portions of the lung. Accordingly, the ground-glass appearance in acute carbon monoxide poisoning may be considered parenchymal interstitial edema caused by tissue hypoxia and/or the toxic effect of carbon monoxide on alveolar membranes. Pulmonary changes in acute CO poisoning might be compared to a mirror image of tissue damage reflected on the lung fields. These changes suggest tissue edema or hypoxia on one hand and interfere with arterialization of the blood in the lungs on the other, resulting in a further marked degree of tissue hypoxia. [394] +

Pulse is rapid, about 120 per minute, respirations, intermittent with occasional periods of apnea; and temperature, 97.3. Decreased breath sounds and scattered rhonchi were heard bilaterally. [395] +

Generalized scattered rales are found in both lungs. [396] +

Intra-alveolar edema was demonstrated in 3 cases of CO poisoning. The gross pathologic changes of the lungs in 351 fatal cases were congestion and/or edema in 66 per cent and hemorrhage in 7 per cent of the cases. The pulmonary changes in acute carbon monoxide poisoning are attributed primarily to prolonged hypoxia and the toxic action of carbon monoxide itself on the alveolar membranes. These factors affect capillary permeability and cause pulmonary edema. [397] +

On gross examination of CO poisoned persons, the lungs are edematous and vivid-red (described as carmine red). [398] +

The right hemidiaphragm was found in 7 patients. [399] +

An infant was exposed to CO in her home. At 3 to 4 weeks of age, when she developed frequent loose stools that were followed 2 weeks later by a dry cough and rapid labored breathing. Over the next 5 to 7 days, her symptoms worsened, and she was taken to her primary physician. The following day she was admitted to the hospital for respiratory distress and wheezing; her oxygen saturation was 50%. Supplemental O2 was started, and she was given nebulized albuterol. A chest radiograph revealed an interstitial infiltrative pattern, hyperinflation, and decreased volume at the left apex. Low sodium, 129 mEq/L At 2 months of age, her mother noted breathing difficulty and wheezing for which she administered nebulized albuterol. However, the infant developed apnea with circumoral cyanosis and stiffness of her extremities. Her father administered several mouth-to-mouth breaths after which she began to breathe again, and she was transported to the local hospital. Her O2 saturation was 80% breathing room air, and she was given supplemental O2. A chest radiograph showed prominent interstitial markings at the base of both lungs with marked changes in the upper lobes that were relatively unchanged from her previous chest radiograph. Initial laboratory work revealed a metabolic acidosis as follows: pH, 7.30; Pco2, 37 mm Hg; Po2, 81 mm Hg; Hco2, 19 mEq/L; base excess, 3.0 mEq/L; and O2 saturation, 96.2%. Her potassium was 6.3 mEq/L, and lactic acid level was 8.3 mmol/L. She was also hypertensive without apparent etiology. The etiology of her hypertension was believed to be related to a renal hypoxic/ischemic insult that was consistent with echogenicity seen on a renal ultrasound. She did well until 3 months of age, when again she was hospitalized for respiratory distress and suspected bronchiolitis; apnea; hyperactive and irritable infant with a 40°C temperature, a 60 breaths/minute respiration rate, and a 200 beats/minute heart rate with an S4 gallop and grade 2/6 midsystolic murmur. Her chest radiograph demonstrated **pulmonary edema** and an enlarged cardiac silhouette. The electrocardiogram showed evidence of biventricular enlargement, and a two-dimensional echocardiogram demonstrated an ejection fraction of 40% without dilation or hypertrophic cardiomyopathy. [400] +

The patient was found comatosed with dilated pupils. Oliguric and hypotensive; **pulmonary edema**; Shortly after admission to the hospital, the patient developed severe respiratory distress with diffuse rales throughout both lungs. A roentgenogram of the chest at this time showed **extensive (diffuse) pulmonary edema**; kidney failure. The patient has been left with severe neurologic defects compatible with bifrontal and temporal lobe damage. She is barely able to walk, has a short memory span and an inappropriate affect. [401] +

29-year old married pregnant woman, **pulmonary edema**, cyanotic, markedly tachypneic, coughing, fairly alert, and markedly hypotensive with rales throughout

both lungs. Fetal movements and heart sounds were not detected. The patient developed high fever, became comatosed and spontaneously delivered a dead fetus. [402] +

On the pulmonary level, an edema can be found and is then either due to left ventricular insufficiency by myocardial sideration (cardiogenic edema), or due to pulmonary damage by direct toxicity (lesional edema). Inhalation, the risk of which is correlated with the depth of the coma, may be complicated by pulmonary lesional damage and/or infectious pneumopathy. [403] +

Lungs highly edematous and congested. 404 +

Exertional dyspnea, increases in pulse and respiratory rates, and syncope. 405 +

In a series of 24 with CO poisoning, 10 developed **pneumonia**. [406] +

Dynamic lung compliance felt airways resistance rose, and arterial blood pressure decreased. Ultrastructural study of the lungs of CO-exposed animals revealed epithelial and endothelial cell swelling, interstitial edema, and alveolar type II cells depleted of lamellar bodies. These findings support the possibility that carbon monoxide intoxication is associated with increased alveolar-epithelial permeability. 407 +

Interstitial and alveolar edema in 11 of 18 patients with acute CO intoxication. In more than one half of 567 patients who died of CO intoxication, pulmonary edema and hemorrhage at post mortem pathologic examination was found. 408 **+**

Comatose with a respiratory rate of 28 breaths per minute, heart rate of 140 beats per minute, temperature of 38.8°C, and blood pressure of 100/80 mm Hg, a white blood cell count of 23,500/cu mm, and a platelet count of 300,000/cu mm. Tachypnea and blood-stained airway foam. Bilateral alveolar and interstitial infiltrates marked intra-alveolar hemorrhage and pulmonary edema with hyaline membrane formation. Severe lung epithelial cell damage, alveolar type II cell multiplication 409 +

The ground-glass appearance on X-ray being the most common finding with alveolar epithelial and capillary endothelial cell edema, interstitial thickening, and focal infiltration with polymorphonuclear leukocytes. 410 +

Coma and acute pulmonary edema. Deeply comatose, with decerebrate posturing on noxious stimuli. Deep tendon reflexes were hyperactive. Blood pressure was 60 mmHg systolic, pulse 84 beats, rectal temperature 36, respirations 24 breaths.

Skin was cherry-red. Later left plantar reflex was extensor. Blood pressure was 160/110 mmHg, pulse 108 beats, rectal temperature 37, respirations deep, 44 breaths. An intense cyanosis was observed, and the *extremities were cold and grey*. Scattered rales were heard at the base of both lungs. White blood cell count 21,000 cells/mm3 and creatine phosphokinase 10,900 U.I. (upper limit of normal 50 U.I.). Bilateral parenchymal infiltrate and a slightly increased cardiac size. 411 +

A boy aged 8 years, marked inspiratory stridor, dysarthria, athetosis predomi- nantly of the face and upper limbs, and a spastic diplegia predominantly affecting the lower limbs was present mild spastic diplegia and minimal dyskinesia of his tongue and upper limbs. [412] +

Marked relief, especially to heaviness on chest in fresh air. 36 +

An 83-year-old man was found unconscious in a smoke-filled room, with labored agonal respirations. Vital signs were as follows: blood pressure, 104/62 m m Hg; pulse, 140 and irregular; and agonal respirations, 8. 413 +

Pneumonia, pulmonary edema, adult respiratory distress syndrome. 414 +

Increased abdominal respiratory effort. 415 +

Tachypnea and/or clinical signs of breathing difficulties (breathing through the nose, retracted thyroid cartilage towards the jugular and intercostal muscles, alternating chest and abdominal breathing, orthopnea). 416 +

Labored and rapid breathing. Pulse 140 and RR 40/m. Facial gramacing and teryifed facies. Absence of deep tendon reglexes. WBC 21,000/cu mm. SGOT, 2,400 mU/mi; lactic dehydrogenase, 1,800 mU/mi; SGPT, 1,345 mU/mi; creatinine phosphokinase (CPK) >3,600 mU/mi. Anuria, pulmonary edema, hyperkalemia, hypocalcemia, serum amylase 1,300 mU/ml, opisthotonos, gramacing, trismus. 417 +

Recorded sequence of the breathing of someone who committed suicide: moaning, whimpering and occasional coughing. Vigorous coughing and retching. Suggestion of inspiratory obstruction with laryngeal stridor. Severe coughing. Expiratory wheeze develops. Marked inspiratory stridor. Coughing gradually lessens in intensity. Respiration becomes shallow and respiratory rate increases. Intermittent periods of apnea. Otherwise shallow regular respiration. Rate slowing. Inspiratory stridor ceases. Respiratory rate now 6 per minute. Prolonged expiratory phase. Definite change in pattern to comatose/stuporose type. Very prolonged expiratory phase. Respiratory frequency 3 per minute. Slight expiratory wheeze**.** 418 **+**

Respirations were labored with a rate of 50 per minute. He was cyanotic, and there was visible engorgement of the veins in the neck. He was unable to open his mouth. The heart rate was from 150 to 160 per minute, with cardiac dilatation both to the right and to the left. There was a systolic thrill over the apex. Large visible fibrillations were seen over the chest. Both lungs were full of rales, and there were symptoms and signs of pulmonary edema. His abdomen was hard. The extremities showed tetanic contractions from three to five minutes apart, and the deep reflexes were exaggerated. 419 +

Headache, shortness of breath and a dry, nonproductive cough, as well as nausea and vomiting. Fainted and then lethargic and hyperventilating. 420 +

Dizziness, headache, inability to see and shortness of breath. 101.6 F (38.6 C); blood pressure, 260/130; pulse rate, 56 beats/minute and respirations, 20/minute. The fact that carbon monoxide intoxication can produce a fever and leukocytosis is not widely known. This relationship was recently emphasized in a report of a family of seven who had fever and leukocytosis in association with carbon monoxide intoxication 421 +

Weakness, dizziness, numbness in his legs and nausea. On admission, his blood pressure was 200/114; pulse, 88 beats/minute and respirations, 24/ minute. mild **congestive heart failure**, **angina pectoris** followed by cerebral vascular accident that left him with a spastic left hemiplegia. 422 +

Respiratory failure. 423 +

Respiratory failure. 1202 *

Weak respiration—respiratory failure. 1202 *

Respiratory failure with coma, pneumonia with coma. 1202 *

Increased respiratory rate. 1202 *

Reduced arterial carbon dioxide levels. 1202 *

Rattling of mucus in air-passages. 1202 *

Bloody mucus is raised from the suction. 1202 *

Respiration audible, almost rattling, slow, stertorous. 1202 *

Respiration rattling. 1202 *

Stertorous breathing. 1202 *

Carboneum oxygenisatum

144

Respiration slow, frequently interrupted by yawning and sighing. 1202 *

Respiration difficult and interrupted. 1202 *

Respiration very labored. 1202 *

Somewhat impeded respiration. 1202 *

Sense of suffocation. 1202 *

The anoxemia resulting from the formation of carboxyhemoglobin induces excessive breathing; and respiratory failure follows the excessive loss of CO2. 424 +

Respiratory rate of 30/min or higher. 425 +

OSI 72%, his spontaneous breathing was shallow and weak; chest x-ray showed **bilateral pulmonary edema**, glucose 198 mg/dL, WBC 17,490, platelets 336,000. red blood and pink foamy sputum were noted from the endotracheal tube, hypotension: <90/50 mmHg, serum cardiac troponin I was 4.19 ng/mL (normal < 0.4 ng/mL). The follow-up chest x-ray showed worsening pulmonary edema with increasing bilateral alveolar consolidation. Arterial blood gases showed severe hypoxemia. Acute respiratory distress syndrome was suspected. 426 +

Acute respiratory congestion was found in eight suicidal patients (10-0%) and two accidental (3-4%). 427 +

There was a short dry cough. 908 +

Short cough when moving. 908 +

The chest had ceased to expand and contract. Emphysema of the lungs, with some bloody sputa. 36 +

Remarkably weak vesicular murmur on auscultation. 36 +

Felt as if a stream of warmth passed from the abdomen into the chest, and thence into the head; it roared in his ears, it affected his respiration; he rose, and after tottering a few steps, fell down, attacked by sudden vertigo. 36 +

Complained now and then of anguish and anxiety in the chest. 36 +

In chest, severe tearing pain. 36 +

Marked congestion of the lungs and brain. 428 +

Carboneum oxygenisatum

145

Fever and shortness of breath with opacities on the right lung. Pneumonia. 429 +

Confused, doubly incontinent state, hypotensive, tachypneic and tachycardic.. Initial signs included respiratory failure, cardiac ischemia, hypotension, encephalopathy and a rash, whilst subsequent features included rhabdomyolysis, renal failure, amnesia, dysarthria, parkinsonism, peripheral neuropathy, supranuclear gaze palsy and cerebral hemorrhage. O2 saturations of 91%, *large sacral pressure sore* and a rash on his left leg. Mild renal failure and a markedly elevated creatinine kinase (CK) level of 12,752 iu/L, deteriorating dysarthria, mild bilateral facial weakness, impaired voluntary upgaze, bradykinesia and a mild flaccid tetraparesis with hyporeflexia evolving to areflexia. His CK peaked at 51,825 iu/L four days after admission and remained elevated for a further two weeks. The rash on his leg showed little improvement with antibiotics. Further examination of this lesion revealed a raised firm purple partially bullous plaque that was not typical of cellulitis. Muscle fibre necrosis. Biopsy of the plaque on the left leg revealed marked edema with a mild perivascular infiltrate suggestive of a purpuric rash. 430 +

In the subgroup of 76 patients, chest x-ray was abnormal on one or several occasions in 43 (57%) patients, showing various degrees of vascular congestion, exudates and consolidations. 431 +

Cherry-red coloring of the organs, tissues and blood and a massive hemorrhagic pulmonary edema. 432 +

Speech and Voice

Confused and uncooperative and had slurred speech. 433 +

Chest

Circulatory system

Heart

Intense cramping pain in heart for a few minutes while lying on right side and my thoughts were to me deceased aunt, disappeared after rising from bed (Day 5). 908.HA.3

Heart cramp while sitting in a chair at 5 p.m. for two minutes, no particular thoughts (Day 6). 908.HA.3

Light pain in heart for 1 minute (Day 17). 908.HA.3

Constriction left side of the chest for a few minutes after 5 minutes walking (Day 18). 908.HA.3

Constricting pain in chest. 1202 *

Chest pain. de Sonnaville *

Pain in lungs, not being able to breathe properly. de Sonnaville *

Muscle pain heart region, radiating left shoulder. de Sonnaville °

The action of the heart and of the lungs gradually decreased. 1202 *

Feeble action of the heart. 1202 *

Action of the heart slow and weak. 1202 *

Beating of the heart alternately increases and diminishes. 1202 *

The beating of the heart is at first increased, but afterwards it becomes slower. 1202 *

Intolerable pain in the region of the heart. 36 +

Intense, burning-sore pain in right superior-extern part of mamma since waking. Very much aggravated by touch and jar (running, descending stairs rapidly). Lasted whole day and next night, could not sleep on the abdomen due to the pain, lying on the painful side pain was tolerable, but when turning on the left side during sleep the pain woke me, being very intense. No lumps, no discoloration. (14 years ago mastitis in right mamma, but that time much lesser pain) (Day 12, 13). 908.HA.3

Pain in the right mamma comes back and it stays for 3 hours (Day 19). 908.HA.3

During the night again pain in the right mamma (Day 23). 908.HA.3

Pain in the left mamma aggravated by accidental touch (Day 10, 200C). 908.HA.3

Pain in the left mamma 3/10, aggravated descending stairs 4/10 and touch 5/10 (Day 11, 200C). 908.HA.3

Weakness in the chest, around the heart area (Day 3, 30C). 908.HA.8

Weakness in the heart area (Day 5, 30C). 908.HA.8

Precordial anxiety. 36 +

Precordial anxiety. 1202 *

Violent palpitation. 36 +

Violent palpitation on exertion. 36 +

Pressure in precordial region produced violent palpitation, a rapid, weak trembling contraction and expansion. 36 +

Chest pain with palpitation when he lies in bed in the evening in a post-COVID-19. 908 *

Desire to loosen clothing on account of palpitation. 36 +

The action of the heart and of the lungs gradually decreased. 36 +

Feeble action of the heart. 36 +

Action of the heart slow and weak. 36 +

Beating of the heart is at first increased, but afterwards it becomes slower. 36 +

Beating of the heart at first strong, frequent, even amounting to palpitation, although associated with slow respiration; at last it becomes irregular and intermitting. 36 +

The action of the heart is at first strong and rapid; it afterward becomes very irregular, so that there is an intermission after four or five pulsations; the intermissions become prolonged on the approach of death, and afterwards become more frequent, so that they intermit every three or four beats. 36 +

At night, in bed, pulse high, rapid, 120 beats occupying a short time. 36 +

Pulse 100. 36 +

Pulse regular, 80, weak. 36 +

Pulse rising and falling (varying between 144 and 88). 36 +

Pulse small, rapid. 36 +

Pulse rapid, very small. 36 +

Pulse quick and soft 97/min. 1202 *

Carboneum oxygenisatum

148

Pulse slow and full. 36 +

Small, slow pulse. 36 +

Pulse small and slow, scarcely to be felt. 36 +

Pulse full, 68. 36 +

Pulse 64 (after thirty minutes). 36 +

Pulse 56. 36 +

Pulse 56, soft, compressible, undulating. 36 +

The pulse is at first accelerated, afterwards retarded. 36 +

Pulse very variable (four children). 36 +

Pulse weak, wavy. 36 +

Pulse small. 36 +

Pulse small, rapid. 1202 *

Pulse rapid, very small. 1202 *

Pulse slow and full. 1202 *

Small, slow pulse. 1202 *

The pulse had stopped beating, or beat so feebly that in the agitation of the moment it was imperceptible. 36 +

CO poisoning can induce serious cardiotoxic effects which are directly proportional to the blood level of COHb. Most patients were normotensive or hypotensive, while hypertension was infrequent. Out of 34 with mild cases of intoxication, 16 persons presented with tachycardia, and 6 with bradycardia. Out of 36 with moderate cases of intoxication, 22 persons presented with tachycardia, 5 with bradycardia. Out of 14 with severe cases of intoxication, 11 persons presented with tachycardia, 3 with bradycardia. Significant increase in the level of troponin I. 434 +

Hypertension: 176/95 mm. 1202 *

Rhythm disturbances include sinus tachycardia, atrial flutter and fibrillation, premature ventricular contractions, ventricular tachycardia, and fibrillation. 435 +

A 71-year old man was admitted to the coronary care unit with retrosternal compressive chest pain at rest lasting 2 hours together with nausea and vomiting. [436] +

A 55-year old non-smoker was admitted for coronary care with retrosternal compressive chest pain of 2 hours duration occurring at rest, and accompanied by nausea and vomiting. [437] +

A 50-year old previously healthy non-smoker presented with fresh onset of angina pectoris. He described several episodes of typical retrosternal compressive chest pain on ascending one flight of steps. He also complained of headache, nausea and vertigo. [438] +

The most common cardiac manifestations are ischemic chest pain, arrhythmias and hypotension and also *ventricular fibrillation*. [439] +

A 47-year-old female patient complained of sudden chest pain for 30 minutes. Before admission, the patient had non-radiating burning chest pain. Findings from this case suggest that CO can cause coronary artery spasm. The burning chest pain occurred in the upper middle sternum and was not radiating. This symptom was accompanied by dizziness, chest tightness, sweating, nausea, and vomiting. The vomit was stomach content and the pain persisted without remission. [440] +

A 15-year-old male was found comatose at home. The arterial blood pressure was 140/80 mmHg and the heart rate 100 beats/min. At physical examination normal hearts sounds and expiratory crepitations on the right side were observed. The patient showed signs of decerebration and a positive Babinski's sign on the left side. Shortly after admission the patient developed an important respiratory distress. The chest X-ray showed an aspiration pneumonia and diffuse bilateral patchy infiltrates. Cardiac dysfunction was immediately evidenced indicating severe left ventricular dysfunction. The patient had a left ventricular ejection fraction of 20% (normal values are 63% with a standard deviation of 8.6%) with apical and anterolateral akinesia and anteroseptal dyskinesia. The patient developed traumatic rhabdomyolysis after laying on the floor for several hours. This rhabdomyolysis was complicated by an acute tubular necrosis which recovered spontaneously. The acute renal failure was initially aggravated by the low cardiac output state. [441] +

We report on a 37-year-old patient who suffered from myocardial stunning after exposure to carbon monoxide, despite having normal coronary arteries. As myocardial ischemia may be asymptomatic in these patients, close monitoring with serial electrocardiography and of serum cardiac enzymes and troponins is recommended. [442] +

A thirty-five year old man with chest pain. Ten months prior to admission the patient had been exposed accidentally to carbon monoxide. After the exposure he experienced a sense of fullness of the head and chest, with dyspnea and a burning sensation over the back of the neck. The following day he had chest pain and shortness of breath, and vomited several times. The electrocardiographic tracing at this time revealed evidence of myocardial damage. Shortness of breath, palpitations, easy fatigability and chest pains had persisted in patients the interval between the accident and the admission to this hospital. These pains occurred chiefly in the precordial region, radiating to the axilla and left shoulder. They were persistent at rest and after exercise. A diastolic gallop was heard. The electrocardiogram was interpreted as showing evidence of myocardial damage. [443] +

A 26-year old Caucasian male presented with syncope. In the hours before presentation he had fainted several times, with complete loss of consciousness. The duration of these episodes was unknown. The patient complained of nausea, had vomited, and apparently been incontinent of urine during one of the episodes. The pulse rate was high, exceeding 100 beats/min. Elevated troponin I serum level of 0.97 (reference: <0.48. [444] +

A female, 73-years old, dizziness, fatigue, shortness of breath, vomiting stomach contents once, acid reflux, and heartburn. respiratory frequency at 30 beats per minute, rough breath sounds in bilateral lungs, audible wet and dry rales, wheezing, heart rate 110 beats/minute, arrhythmia, electrocardiogram with the wild range ST-segment depression, emergent cardiac troponin I 4.75 ng/mL, creatine kinase MB 1494ng/mL, monitored oxygen saturation 90%. Chest radiography suggested pulmonary edema; was diagnosed with acute CO poisoning, acute coronary syndrome, acute left heart failure, lactic acidosis. [445] +

He suddenly collapsed and lost consciousness while on duty with squeezing pain in his left chest. 446 +

Premature ventricular contractions during exercise. Myocardial infarction and dysrhythmias are described in victims of CO poisoning, and acute mortality from CO is usually a result of ventricular dysrhythmias. [447] +

Increased incidence of dysrhythmias, mostly paroxysmal tachycardia and ventricular fibrillation, in decade after CO poisoning. [448] +

A 12-year-old child showed cardiac damage associated with relatively low COHb concentrations following prolonged CO exposure; acute myocardial infarction; mitral regurgitation. Increased heart rate and the presence of gallop sound. [449] +

CO poisoning has a variety of deleterious cardiac effects including arrhythmias, coronary spasm and myocardial infarction. The pro-thrombotic effect of carbon monoxide poisoning is one of the important postulated mechanisms of cardiac injury. There are multiple case reports of myocardial infarction secondary to CO poisoning. However, there is no published case of carbon-monoxide-poisoning-induced stent thrombosis. We present a 50-year-old female with late stent thrombosis of a sirolimus-eluting stent secondary to acute CO poisoning. [450] +

Acute weakness, dyspnea, nausea and vomiting. An electrocardiogram revealed sinus tachycardia, non-specific ST-T wave abnormalities, and a prolonged QTc interval. The chest X-ray revealed pulmonary edema and the 2-D echocardiography revealed decreased left ventricular systolic function with an ejection fraction of 25%. 451 +

Severe heart failure. 452 +

Myocardial ischemia, atrial fibrillation, pneumonia, pulmonary edema, erythrocytosis, leucocytosis, hyperglycemia, muscle necrosis, acute renal failure, skin lesion, and changes in perception of the visual and auditory systems. 453 +

Myocardial fibrosis from severe CO in a 63-year-old woman. [454] +

A 34-year-old woman with a non–ST-segment elevation myocardial infarction. Her heart rate was 132 beats per minute, blood pressure 126/70 mm Hg, and respiratory rate 38 breaths per minute. Serum glucose was 125 mg/dL. Shortly after blood pressure 178/102 mm Hg, respiratory rate of 40 breaths per minute. She had spontaneous eye movements with stimuli, pinpoint and uneven pupils, positive gag reflex, rigidity in all 4 extremities with extensor-type posturing, and hyperreflexia. creatine phosphokinase 252 IU/L, and creatine kinase- MB 14.3 ng/mL with a ratio of 17.62. The leukocyte count was 23,600/mL. Repeat cardiac enzymes 2 hours later showed an increase with troponin I 2.72 ng/mL and creatine kinase-MB 29 ng/mL. She was diagnosed with myocardial ischemia or infarction in the absence of significant coronary artery disease. [455] +

Two young people with no history of chronic diseases (a 24-year-old man and a 39-year-old woman) who were diagnosed with non-ST-elevation myocardial infarction in the course of severe carbon monoxide poisoning. Both poisoning cases were severe, both patients were unconscious when they were admitted to our hospital. We observed elevation of serum troponin level and changes in ECG (e.g. atrial fibrillation) significant for myocardial infarction. [456] +

A 42-year old female presented normal coronary arteriogram with serial EKG and biochemical changes in a victim of CO intoxication with evidence of myocardial infarction. [457] +

Myocardial infarction in a 46-year-old white man with normal coronary arteries after acute exposure to CO. His pulse rate 146, his respiratory rate 24, irregular, shallow, gasping respiration. Lung exam revealed bilateral rhonchi. [458] +

Prospective cohort study of 230 consecutive adult patients treated for moderate to severe CO poisoning with hyperbaric oxygen. Myocardial injury (cardiac troponin I level 0.7 ng/mL or creatine kinase-MB level 5.0 ng/mL and/or diagnostic electrocardiogram changes) occurred in 85 (37%) of 230 patients. Myocardial injury occurs frequently in patients hospitalized for moderate to severe CO poisoning and is a significant predictor of mortality. [459] +

We present the first case of severe cardiotoxicity of carbon monoxide leading to myocardial rupture and fatal outcome. A 83-year-old woman was hospitalized 4 hours after being found in her house with no respiratory or cardiac symptoms. After two days, she has suffered sudden collapse leading to cardiac arrest. Postmortem examination revealed intramural hemorrhage with myocardial rupture at the apex of the left ventricle. [460] +

The father, age 33, was admitted unconscious. His blood pressure was 180/80 mm Hg and pulse, 140/min. The deep tendon reflexes were hyperactive, and sustained ankle clonus and Babinski's signs were present bilaterally. He regained full consciousness 48 hr after admission. On the third day the serum SGOT and SGPT transaminases were 216 and 125, respectively. On the fifth day, he complained of sudden, severe, substernal pain. An electrocardiogram showed anterior subepicardial injury. Vigorous attempts at resuscitation were unsuccessful. A mural thrombus measuring 2.5 by 2 cm was found near the apex of the left ventricle. A terminal branch of the left anterior descending coronary artery was occluded by a thromboembolus for a distance of 4 cm in its most distal part. At microscopy there was no evidence of acute myocardial infarction in the area of the heart supplied by the occluded vessel. Focal degeneration of the myocardium was present throughout the heart but mainly in the left ventricle. [461] +

The wife, age 27, was disoriented. Six days after admission an electrocardiogram showed sub-endocardial injury and ischemia. Over the next several days the T waves became more sharply inverted. On the tenth day the patient complained of pain in the left upper chest with radiation to the left arm and hand. [462] +

The daughter, age 7, responded only to painful stimuli on admission. The blood pressure was 110/80 mm Hg and the pulse, 140/min. Retinal hemorrhages were seen bilaterally. One and half hours after admission she was awake and talking. A paresis of the right side was present, which disappeared over a 3-day period. [463] +

Chest pain resulting from myocardial ischemia or necrosis. Shortness of breath and low blood pressure. Dyspnea and chest pain. 464 +

Chest pain as a symptom of myocardial ischemia can occur without underlying coronary artery disease. For example, 2 weeks after accidental exposure to CO, 34% of a group of Swiss soldiers had chest pain . 465 +

Sixteen suicidal patients (20%) had pulse rates in excess of 100/min, one patient showing multiple extrasystoles. Three patients (3-8%) were in shock. Deep vein thrombosis occurred in two patients (2-5%) within 48 hr of admission and one patient died from subsequent pulmonary infarction. Of the 'accidental' group, five (8-6%) had a tachycardia, one (1-7%) was in shock, and one patient presented with congestive cardiac failure. 466 +

The study of the mortality risk in patients with moderate to severe CO poisoning at low risk for cardiovascular diseases, 37% suffered acute myocardial injury and 38% were dead within 7.6 years. The mortality rate was three times higher than the US expected mortality by age and sex. 467 +

After CO exposure, angina attacks, arrhythmias, and increased level of cardiac enzymes frequently occur. This has incomplete right bundle branch block, left anterior fascicular block (LAFB), and nonspecific ST-T segment changes, glucometer reading was 114 mg/d . 468 +

Cardiac manifestations after exposure to CO including myocardial ischemia, heart failure and arrhythmias. 469 +

Mild cardiomegaly with pulmonary edema in both lung fields, sinus tachycardia with a heart rate of approximately 130/min, body temperature 36.0°C, heart rate 97/min, and respiratory rate 28/min. Marked elevation in the serum level of pro-brain natriuretic peptide of 18,699 pg/mL. Cardiac enzymes were elevated to a troponin I of 0.88 ng/mL and CK-MB of 9.7 ng/mL round thrombus (12 × 10 mm) at the apex of left ventricle. 470 +

Several cardiac manifestations have been reported, including arrhythmias, acute myocardial infarction, and pulmonary edema, prolonged chest pain. Pulse

170/bpm. Chest radiography showed mild cardiomegaly with pulmonary edema in both lung fields. White blood cells 21,200/μL, blood urea nitrogen 17 mg/dL; creatinine 0.75 mg/dL, and blood glucose 101 mg/dL, D-dimer 0.28 ug/mL. Cardiac enzymes were elevated (CK: 3306 U/L, CK-MB: 90.6 ng/mL, troponin I: 1.899 ng/mL, lactate dehydrogenase: 334 U/L. Vital signs were blood pressure of 132/101 mmHg, pulse rate of 87/min, respiration rate of 20/min and body temperature of 35.4°C. Oxygen saturation measured using pulse oxymetry was 100% when 15 L/min oxygen was applied through a reservoir bag mask. 471 +

Systolic blood pressure 190 mmHg, his emotions were exciting, irritably, his judgment was reduced. 472 +

Creatinin kinase (CPK) was 1119IU/L (60–400) with first degree AV block. 473 +

Heart rate was 92 beats/min and irregularly irregular. 16000 K/uL, Hb: 10.6 g/dL, hematocrit: 32.1%, creatine kinase: 1305 U/L, patient could independently ambulate, but the steps were small, unsteady, and shuffling, exhibiting the performance of lower limb ataxia. Cerebellar atrophy while no abnormality in the rest parts. 474 +

Thirty (38%) patients exhibited arrhythmias other than sinus tachycardia. 475 +

Blood pressure of 115/80 mm Hg, pulse 118/min, respiration rate 19/min, body temperature 36˚C, SO2 96%, PO297 mm Hg, and blood glucose 173 mg/dl were observed. Deep tendon reflexes were absent in lower extremities. Generalized rhonchi in the lungs and tachycardia. Laboratory analyses revealed the following: white blood cells (WBC) 21.000 μl, blood urea nitrogen (BUN) 33 mg/dl, creatinine 1.35 mg/dl, CK 629 U/L, CK-MB 147 U/L, troponin T 0.136 ng/ml, lactate dehydrogenase (LDH) 678 U/L fibrinogen and D-dimer values were high. Right bundle branch block. Edema was found in cranial computed tomography. Dyspnea and cyanosis; 5 × 2 cm thrombus in the right atrium. Dilatation of the left pulmonary artery was observed. A sequences in both globi pallidi were detected, which is pathognomonic for CO poisoning. 476 +

Bilateral thrombosis of the popliteal vein with diffuse muscular necrosis following CO intoxication. 477 +

Palpitation, headache and nausea. Electrocardiogram revealed supraventricular tachycardia. 478 +

Myocardial necrosis. **479** +

Heart rate usually rises during early exposure, but then declines as COHb saturation increases. **480** +

Circulation

Thrombosis of the vein of Labbé with hemorrhagic cerebral infarction. 481 +

Fatal obstruction of the basilar trunk following benign carbon monoxide poisoning. 482 +

Atrial thrombus and partial thrombosis of the superior sagittal sinus following CO poisoning. 483 +

A case of thrombosis of the mesenteric artery after carbon monoxide poisoning. 484 +

Stroke has been rarely associated with CO intoxication. In fact, we found only 3 case reports of stroke, among 13 cases of carbon monoxide poisoning with thromboembolic events. We report a case of ICA thrombus and stroke in a patient with acute CO intoxication. A 46 year-old woman malignant infarction in the left ICA territory. Cervical vessel ultrasound showed an adherent thrombus causing a 50% stenosis at the origin of the left ICA. The temporal coincidence between CO intoxication and arterial thrombosis suggests a causal relationship. [485] +

CO is one of the leading causes of poisoning; it inhibits oxygen delivery, subsequently causing ischemic changes and ultimately death by multi-organ failure. Furthermore, thromboembolic episodes due to CO poisoning have been reported. However, intracardiac thrombus formation following exposure to CO has been very rarely described. Here, a case of right atrial large thrombus formation after CO poisoning is presented. A previously healthy 24-year-old woman; creatinine kinase-myocardial bound of 90.6 ng/mL (upper limit 5 ng/mL) and troponin I of 1.899 ng/mL (upper limit 1.5 ng/mL). A transthoracic echocardiography was performed 24 hours after the accident, revealing a 30x15 mm nodular echogenic mass in the right atrium. [486] +

Leucocytosis, mainly neutrophilic, observed during the first few days seemed to be a physiological phenomena due to a stressful situation, such as hypoxia. Levels of platelet increased steadily after an initial decrease. We found no specific findings in bone marrow. [487] +

Thrombotic thrombocytopenic purpura, on the other hand, is a rare condition, diagnosed clinically by a pentad of findings: ie, fever, CNS dysfunction, renal impairment, microangiopathic hemolytic anemia, and thrombocytopenic purpura. Patho-

logically, wide-spread fibrin microthrombi are found. Recently, respiratory dysfunction has been reported to be associated with TTP. A 27-year-old man was brought to the emergency room comatose after being jailed approximately 12 hours earlier for "unruly conduct." The temperature was 40.5 °C rectally, and blood pressure was 130/70 mm Hg; respirations, 40/min; and pulse rate, 84 beats per minute and regular. Blood was present in the oropharynx, but no active bleeding could be perceived. Examination of the chest disclosed bilateral crackles. Deep tendon reflexes were hyperactive but symmetrical, and there was sustained ankle clonus bilaterally. Ecchymotic areas were noted in the axillary and gluteal areas with erythematous bullae on the back, hands, and thighs. The hematocrit reading was 27.8%; hemoglobin level, 10.3 mg/dL; WBC count was 18,900/cu mm; platelet count was 12,000/cu mm. a left lung infiltrate developed. Deteriorating oxygenation, and was believed to have acute respiratory distress syndrome. Twenty-four hours after admission, he became hypotensive, asystole developed, and he could not be resuscitated. Autopsy showed widespread fibrin microthrombi in the myocardium, CNS, adrenal glands, lungs, gastrointestinal tract, and kidneys consistent with TTP. There were ischemic changes of the hypothalamus and right cerebellar hemispheres, and a diffuse five-lobe pulmonary edema was present, consistent with CO poisoning and non-cardiogenic pulmonary edema. The vascular damage induced by CO is strikingly similar to that seen with TTP. In fact, CO induced damage may result in hemorrhage into the vessel wall followed by aggregation of thrombocytes, which is the first step in the proposed pathogenesis of with CO poisoning, it is clear that both involve similar vascular endothelial damage. Thrombotic thrombocytopenic purpura, once thought to be a specific disease entity, may actually represent a peculiar biological reaction to a number of exogenous factors, and should instead be thought of as a syndrome. [488] +

We included 193 (117 women) patients who presented with a diagnosis of CO poisoning between June 2011 and March 2013. Control group was composed of 39 (15 women) patients. Troponin and creatine kinase MB levels were significantly higher in the CO poisoning group. Platelet counts were significantly higher in patients with CO poisoning (281 +76 vs 248 +65 109, respectively; P 1⁄4 .01). Similarly, MPV was significantly higher in the CO poisoning group (8.9 +0.8 vs 7.9 +0.9 fL, respectively; P < .001). Elevated MPV values may indicate that patients with CO poisoning have a higher risk of thromboembolic and cardiovascular complications due to platelet activation. [489] +

Almost every known central neurological syndrome has been reported as a complication of CO poisoning. Hemorrhagic infarct has rarely been considered as an early manifestation of CO poisoning. A case of cerebral hemorrhagic infarction is pre-

sented. Typical findings, neuropathology and the role of vascular injury are described. To evaluate the possible causes, a brain computed tomography was performed that showed thick clot in the left medial temporal and parasellar area, left sylvian fissure (acute intravascular thrombus) accompanied by left diffuse fronto-temporal hypodensity and midline shift. Four-vessel digital subtraction angiography two weeks after intoxication was not indicative of any vascular lesion. A 7-year-old boy was admitted with history of persistent vomiting, progressive loss of consciousness and severe right hemiparesis. He had been well several hours before admission when he and all of his family members had developed vomiting, headache and lethargy. All of them were admitted to hospital and subsequently had good recovery with standard treatment for CO poisoning. However, this young boy showed progressive worsening of symptoms including persistent vomiting, generalized convulsions, coma, and right hemiparesis. Brain CT was performed on the day after admission which showed thick clot in left medial temporal and parasellar areas, intravascular thrombus in the left middle cerebral artery, diffuse left fronto-temporal hypodensity, and midline shift. A good recovery was found except for a global aphasia. Follow-up brain CT scan one year later revealed brain atrophy in left fronto-temporal area, which appeared to have been an evidence of resolving in a large hemorrhagic infarct. Hemorrhagic infarction in the brain of patients referred for CO poisoning is very rare. We have tried to describe the probable mechanisms. Micro-vascular impairment and brain reperfusion injury patterns have been reported in CO poisoning. This micro vascular injury can produce ischemia and hemorrhage. Besides cardiovascular and hematological complications due to CO poisoning have been proposed to be another cause for hemorrhage. [490] +

Hemorrhage complicating acute CO poisoning is usually confined to the basal ganglia. The authors report three people with acute CO poisoning manifesting selective or prominent white matter involvement complicated by hemorrhagic infarction on CT and MRI. A 37-year-old woman was unresponsive and admitted to the hospital. Deep tendon reflexes were hyperactive, and plantar response was bilaterally extensor. CT head scan showed diffuse decreased white matter attenuation and bilateral hemorrhagic infarction of the centrum semiovale. She gradually awakened over several weeks and after 7 weeks comprehended fully, spoke in full sentences, and began moving her upper extremities and was transferred to a rehabilitation center. Her 3-year-old daughter was found unresponsive at the scene. At 2 weeks she was alert with normal speech and cranial nerve function. There was a mild unsteady gait with hyperreflexia and bilateral extensor plantar responses. She continued to improve, needing no assistance in daily activity by the time of discharge at week 4. [491] +

Hemorrhagic necrosis and vascular injury in CO poisoning: MR demonstration: A 29-year-old white man, cyanosis, labored breathing with a respiratory rate of 28, and, neurologically, the patient was obtunded with response to deep pain; deep tendon hyperreflexia and cogwheel rigidity in all extremities. He also had marked myoclonus in the right upper extremity and no bowel or bladder control. He presented 3 years after an acute CO poisoning with irritability, **violent actions**, and verbal aggression. This case demonstrates characteristic findings on MR imaging of CO poisoning, as well as observations that suggest prior focal hemorrhage. Typical findings, neuropathology, and the role of vascular injury. Neurologic evaluation during the current illness showed marked improvement, but deep tendon reflexes were increased, spastic clonic movements of the upper extremities were observed, and there was increased muscle tone in all extremities. [492] +

Free radicals disrupt the vascular endothelium and result in platelets aggregation and are also responsible in the inhibition of epidermal-derived growth factor and prostacyclin production disrupting normal hemostasis and resulting in thrombus production. 493 +

Intermittent claudication occurs earlier than expected in patients with low levels of COHb, and CO poisoning has been implicated in the pathogenesis of atherosclerosis. Long-term exposure to low levels of CO causes polycythemia. The major cause of death associated with acute CO poisoning is cardiac arrhythmia. Premature ventricular complexes are common, and infarcts can occur. 494 +

CO may elevate plasma cholesterol and does appear to enhance atherosclerosis when serum cholesterol is greatly elevated by diet. 495 +

Blood flow and perivascular abnormalities. Acute vascular and perivascular changes. 496 +

Leucocytosis, erythrocytosis, anemia, pernicious anemia, thrombotic thrombocytopenic purpura. 497 +

These results indicate that the increase in the young reticulocyte population and the subsequent increase in total reticulocyte count are the earliest erythropoietic responses to intermittent CO exposure and that CO-induced polycythemia is associated with cardiac hypertrophy. [498] +

The slightest movement is liable to bring on vertigo and loss of consciousness. This occurred twice in this case, the man saying he felt well and wanted to go home, and becoming unconscious when he started to get up. Total occlusion of the posterior descending branch of the right coronary artery with a large occlusive thrombus

burden. Cardiac enzymes were elevated, with a CK-MB of 153.9 ng/mL and troponin I of 30.3 ng/mL. Hypertension. 499 +

After exposure to CO, several cardiac manifestations have been reported, including arrhythmias and electrocardiographic alterations, acute myocardial infarction, pulmonary edema, and cardiogenic shock. 500 +

The blood is brighter red than venous blood of an unexposed person. However, the time-honored sign of a “cherry-red complexion” may not be apparent and is not necessary for a diagnosis of CO over-exposure. 501 +

Increased pulse and respiratory rate and then decreased heart action. Weak pulse, depressed respiration, respiratory failure. 502 +

Pulse varying in force and frequency, at times almost imperceptible, the number ranging at different times from 80 to 120. 36 +

Back

Pain in cervical region in the morning, aggravates a lot during washing dishes, when I have the sensation my neck will break, it is not able to sustain my head, vanished after walking in the open air (lasted for 2 hours) (Day 7, 8). 908.HA.3

Stiffness of neck 15 min after waking up, stiffness extending down the thoracic spine, turning of the head, bending forward and stretching back, moving to the to both sides painful and stiff (Day 4, 200C). 908.HA.4

Whole day limited head mobility, stiffness shoulder and neck, when turning the head, the shoulders must also turn (Day 4, 200C). 908.HA.4

Mobility of the neck and thoracic spine limited due to stiffness (Day 5, 200C). 908.HA.4

Painful muscle stiffening of the trapezius area, turning the head restricted. Improved by massages and pressure on the muscle (after Day 10, 200C). 908.HA.4

Strong shoulder and neck stiffness and occipital headache, extending to the left side of the head. Lying on the back ameliorate, lateral position rather worse, not quite sure. (after 30C). 908.HA.6

Soreness of all the cervical muscles while exerting the brain (second day). 36 +

In the evening I feel a griping pain in front of the left scapula, that lasts 1 minute (Day 9, 200C). 908.HA.3

Burning pain at the right scapula which soon ceases (second day). 36 +

Cramping pain lower lumbar region (L4-5) with contracted muscles on waking in the morning, worse when walking on hard ground (jar) and after getting up from sitting. Probably after too much lifting followed by cold bathing in the river (on 5th day after the first dose, lasting for 3-4 days) (Day 5). 908.HA.5

Pressing, drawing pain in the left sacro-iliac region radiating to the left hip when waking up in the morning. Worse after sleep and the first movements in bed are very painful, even on rising from bed it is painful, but less than the first motion, after a few minutes of walking it is much better, it gets better the more I walk, also hot showers improves. Standing worsens. With long standing massive increase of pain in the thoraco-lumbar area as if the back would "break apart". Sitting, lying, resting and local warmth as well as massaging improves. Stretching and treadmill training improves the pain left sacro-iliac region. (Chronic symptoms of the prover, which appeared after the 200C). 908.HA.6

Extremities

Extremities flexed. 36 +

Feeling as if the left upper and lower extremities had gone to sleep and could not be moved. 36 +

Inclination to stretch the extremities. 36 +

Clonic cramps in the extremities. 36 +

All muscles are tight. Most muscle pain in calves, kind of continuous cramp. de Sonnaville *

Clonic spasms of the extremities. 36 +

Trembling of limbs. 36 +

Convulsions in limbs and stiffness in joints. 36 +

All limbs convulsed. 36 +

Great weariness of the limbs. 36 +

Paralysis of the left arm and left leg continues after the attack. 36 +

Complete paralysis and the left arm and left leg continues after the attack. 36 +

Complete paralysis of the right foot and right hand, and also of the muscles of the right half of the face; persisted after the attack. 36 +

Pains in the extremities, followed by paralysis. 36 +

Loss of motion and sensation in the right arm, followed by atrophy. 36 +

The right leg was cold, somewhat heavy; the arm recovered under galvanism, after many months. 36 +

Incomplete paralysis of the left lower extremity, with serous infiltration of the foot, which was painful, not red; and along the dorsal surface of the metatarsus there developed numerous blisters, with brownish-red spots as large as a half dollar. 36 +

Extremities quite cold. 36 +

Extremities cold. 1202 *

Flushed appearance, loss of power of his hands, paresthesia in the extremities, blurring of vision, weakness and dizziness. Headache, anorexia, dyspepsia and tiredness. 503 +

Redness of his legs and of arms. 908 °

Skin part of fingers and toes red. de Sonnaville *

Peripheral cyanosis. 504 +

Muscle necrosis, Volkman's contracture, osteomyelitis. 505 +

This remedy is indicated in herpes zoster and pemphigus. There are vesicles along the course of the nerves. 100 *

Upper limbs

Dropped two glasses of water (I never do that). (Day 2). 908.HA.1

Spilled another full glass of water. (Day 4). 908.HA.1

Compartment syndrome on a patient's forearm related to CO poisoning: Swelling and rash were detected on the left forearm in the patient's system examinations. Paresthesia, paralysis, and pallor were detected, along with pain in the arm during

passive movements. Widespread intramuscular hemorrhage and edema in subcutaneous tissues. [506] +

Muscle pain shoulder, worse move. de Sonnaville °

Muscle pain from lying in bed. de Sonnaville °

During daily morning swim a pulling into the right upper arm (after Day 10, 200C). 908.HA.4

Pain in the left wrist in the afternoon while using the hand, disappears in the evening (Day 22). 908.HA.3

Arms flexed, could not be extended. 36 +

Tossing about of the arms. 36 +

The chorea-like movements of the right arm continued for some days, only on waking. 36 +

Slight drawing pain in extremities, not very painful, just noticeable (Day 3, 30C). 908.HA.8

Slight drawing in hands and feet and other joints (Day 4, 30C). 908.HA.8

The arms and hands are without strength (second day). 36 +

Spasm of flexors of forearm. 36 +

A remnant of pain in the left knuckle-joint, caused by a fall on the ice six months before, returned with increased severity, and extended to the corresponding parts of the right hand (second day). 36 +

Fingers clenched. 36 +

Fingers clenched. 1202 *

Numbness of three fingers of the right hand; the fingers can only be extended with difficulty. 36 +

Numbness hands. de Sonnaville *

Radial paralysis. 507 +

Bilateral brachial plexus injury associated with edema of the face and the upper limbs. Neurological examination showed a brachial diplegia, distal vibratory, ther-

mic and algic hypoesthesia, deep tendon areflexia in upper limbs. Creatine kinase was elevated. Cerebral magnetic resonance imaging showed bilateral pallidal abnormalities. 508 +

A 40-year-old Caucasian woman was exposed to CO when she was sleeping alone in her one-bedroom apartment; fortunately, the beeps from her First Alert combination smoke and CO detector woke her and she was saved from any extensive health issues. The most indicative symptoms experienced were a severe headache, blurred vision, agitation, and confusion. Following contact with the Emergency Responses Services, she was promptly transferred to the hospital via ambulance and was treated with high-flow oxygen on the way. She was discharged from the emergency department on the same day, but CO exposure had already had adverse effects on her fingernail beds. *The fingernail tips were altered and appeared as if a bite had been taken out of their distal borders.* The changes in the tips of her fingernails were significant, but they completely disappeared eight weeks later without any additional treatment. At that moment, her blood pressure was 150/100mmHg, heart rate was 88 beats per minute, respiratory rate was 18 breaths per minute, and her temperature was 97.6°F. She had a severe headache in the frontal area, felt deep pain in the orbits, and had blurred vision, light-headedness and stabbing chest pain. She was agitated and her concentration was decreased. Some areas at the top margin of her fingernails were completely destroyed and marked with a bite out of the border without any separation between nail bed and nail plate. The nail bed border under her fingernails showed areas of erosion instead of a curved pattern as shown. These changes occurred in all fingernail beds but prominently in the fingernails of the first and second fingers. The fingernail beds' color was pink, and redder and bluish at the top border. There was no pain in the nails. At the time of the 10-month follow-up examination, the nails had returned to normal. 509 +

Lower Extremities

Left thigh and groin pain. Aching, like a toothache, dull and throbbing, comes in waves, crescendo-decrescendo type pain. Better by warm application. Worse sitting still, better lying. Better with movement (Had done the day before more squatting exercises than usual). (Day 7). 908.HA.1

Easily exhausted feeling in the legs during riding with bike, heaviness in legs, it was tremendously exhausting to keep pedaling. I would have loved to stop, get off the bike and lie down, feeling overstrained (Day 2). 908.HA.7

Tension and stiffness of the thigh and calf muscles (30 min after C 30) (Day 1). 908.HA.6

Tension and stiffness of the thigh and calf muscles today also gluteal (Day 2). 908.HA.6

Stiffness of the thigh muscles like sticks (immediately after 30C). 908.HA.6

Stiffness in the thigh muscles (after 200C). 908.HA.6

Shooting pains in right nate and down sciatic nerve to foot, not worse by pressure or movement. 85 +

Weak legs, muscle pain legs. de Sonnaville °

Muscle pain in thighs, better moving for a while, want to move, worse lying quietly. de Sonnaville °

Muscle pains better warmth. de Sonnaville °

Muscle twitching in thighs. de Sonnaville °

Lower limbs tingle, better when lying down. de Sonnaville °

Muscle pain calves, cramping, tight. de Sonnaville °

Muscle pain, better lying still. de Sonnaville °

Trembling of legs. 36 +

Attempted to rise, but was unable to do so; legs stiff and powerless. The legs can scarcely sustain the body (second day). 36 +

The weakness in both legs continued to increase, to complete paralysis of the right and incomplete a paralysis of the left. 36 +

Occasional shooting pains in the right nates, just where the sciatic nerve emerges; there was noticed at this point an elliptical red spot half as large as the band, without any trace of blisters, only the skin seemed to be somewhat puffy and elastic, without fluctuation. 36 +

The pains in the nates extended along the sciatic nerve, and the external popliteal nerve, down to the foot; no pain on pressing on the os ilium or on moving the leg. 36 +

When sitting on a low chair, the gluteal muscles are painful, as if he had just got up after a severe sickness and was much emaciated (second day). 36 +

My feet and lower legs (below the knee) were so cold on waking, I expected them to be white. They were not discolored in any way. The mattress upon which my feet and legs lay, and the blanket that lay upon them, were noticeably cold (after 6 hours). 908.HA.9

Coldness feet. de Sonnaville °

Swelling feet. de Sonnaville °

Extension of the right leg became difficult (second week). 36 +

When putting on his garters in the morning, they cause pain, and he is obliged to tie them so loosely that they slip down (second day). 36 +

Cramp in left calf morning at 5:30 a.m., woke me up (Day 5). 908.HA.3

Toes could not be moved (second week). 36 +

Bed sores esp. on heel turning towards early gangrene. 1202 *

Popliteal vein thrombosis, pulmonary emboli, and possible tissue necrosis. Pulse, 110/min; respiratory rate, 40/min; temperature, 36.2°C. She was unconscious. Vomiting. Cerebral edema. Pleural effusion in the right lower zone, fever, and elevated WBC count made us consider pulmonary emboli. 510 +

Deep vein thrombosis is the formation of blood clots in the deep vein, with thrombi predominantly occurring in the legs. The incidences of DVT and PE were higher in the patients with CO poisoning. 511 +

Crack in left heel, painful while walking (old symptom, last time many years ago) (Day 2,3,4). 908.HA.3

Chill

Full-body chills with gentle movement (walking, drinking, typing) (second hour). 908.HA.9

While eating started to tremble from the cold, yet was unwilling to do anything to warm up (second hour). 908.HA.9

Feeling cold (Usually quite warm). (Day 6). 908.HA.1

Chilliness. 36 +

Chilliness. 1202 *

Cold, mottled skin. The temperature of the body was rapidly falling. 36 +

Bodily temperature remarkably lowered; skin cold, giving to the touch an impression like that caused by contact with a corpse some hour after death, before it is quite cold. 36 +

Instantly a sense of ice-cold air radiating from my hands and feet. I have no impulse to warm them (almost an indifference or apathy to become warm) (immediately). 908.HA.8

Temperature in the axilla, 34.6; in the mouth, 35.2. 36 +

Temperature 38. 36 +

Chilly with hot red head. de Sonnaville °

Fever up and down, everything changes. de Sonnaville *

High fever (40) chattering teeth, shaking, nausea. de Sonnaville *

Fever and nausea, headache, no appetite. de Sonnaville *

Fever with sweating. de Sonnaville *

Dry and warm with a high fever. de Sonnaville *

Shivery in the afternoon. de Sonnaville *

Diarrhea first, then fever. de Sonnaville °

Cold inside. de Sonnaville *

Fever and poor sleep. de Sonnaville °

Very chilly. de Sonnaville °

Very painful and tight muscles, worse moving, sometimes restless. de Sonnaville °

Alternating cold and warm. de Sonnaville °

Coldness. 36 +

Sensation of coldness in the whole body. 36 +

The attack is followed by long-continued sensation of coldness and general trembling, which may last for weeks. 36 +

Severe chill, with chattering. 36 +

Violent febrile chill, for several days. 36 +

Violent and continued shaking chill. 36 +

Daily repeated chills, with a sensation of a heavy dragging up of the abdomen when walking and standing. 36 +

Chilly all day. 36 +

Cool extremities. 36 +

Extremities cold. 36 +

Extremities cold and numb. 36 +

Hands and feel cold. 36 +

Hands icy cold. 36 +

At night, in bed, burning heat all over, without thirst; despite this heat and fever, slept lightly until one AM, after which increase of heat, with thirst and dry mouth; the thirst was satisfied by drinking only a little; the heat, as well as the thirst an fever, now gradually diminished, and the bed, which had hitherto been too warm, was now too cold, so that he had to have more covering; sleep returned. 36 +

When I put on a sweater, I quickly got too hot, when I took it off, I felt too cold again although it was a warm day outside (Day 3, 30C). 908.HA.8

Mix of hot and cold feeling as if unable to regulate temperature (Day 4, 30C). 908.HA.8

Feverish feeling, but no temperature (Day 5, 30C). 908.HA.8

Strong desire for fresh air, and cool air seems the best. A stuffy room seems unbearable. As soon as I can be outside or near an open window I feel definitely much better. I want to be outside, but not move, sitting or even lying makes me feel better (Day 5, 30C). 908.HA.8

Sensation of warmth in chest and abdomen, similar to that caused by spirituous liquors, but the hands and feet remained cold. 36 +

Sweat all over the body during falling asleep in the evening (26 degrees Celsius) (Day 2, 30C).908.HA.4

Skin covered with sweat. 36 +

Beads of sweat over whole body. 36 +

A little sweat on the upper half of the body, in the morning, in bed. 36 +

Slight frontal sweat. 36 +

A 52-year-old man presented to the emergency department with severe CO poisoning. His core body temperature, measured in the urinary bladder, was 32.4°C. metabolic acidosis with elevated lactate (pH 7.081; base excess, −19.2 mmol/L; HCO3, −9.8 mmol/L; lactate, 168.8 mg/ dL). The patient was diagnosed with severe CO poisoning complicated with hypothermia. [512] +

Temperatures below 97°F were observed in six (7-5%) of the suicidal group and one (1-7%) of the accidental group. The lowest temperature was recorded in a 38-year-old woman who presented with delirium, signs of bilateral pyramidal tract involvement and a rectal temperature of 89°F. A temperature of 100°F or more was found in four suicidal patients (5-0%) and two accidental (3-4%). The highest recorded temperature of 105°F accompanied the delayed development of severe, persisting parkinsonism in a 27-year-old man. 513 +

Temperature: subnormal 34.6 to 38. 908 +

Temperature of 102°F or higher. **514** +

Excessive sweating. 515 +

Skin

Skin bloodless; the veins show through it blackish. 36 +

Surface of body reddish-livid. 36 +

Bluish, cyanotic color of the entire skin, especially of the face, neck, antero-superior portion of the chest and back of the hands; on all which parts the skin was actually slate-colored; this color was also noticed on the mucous membrane of the lip. 36 +

The skin assumes a violet color; the veins are swollen; the lips and conjunctiva are cyanotic. 36 +

Purple maculation of the skin. 36 +

Insect stings three times during the proving. Stings very itchy, if I start scratching cannot stop scratching. Swelling is red, hot and large, one is 10 cm in diameter (Prover is usually more sensitive than others for insect bites, but during the proving it was extreme). 908.HA.3

The skin had lost its normal tone and elasticity; when pinched, the folds remained for some seconds and disappeared slowly. 36 +

Along the course of the radial nerve in each forearm a linear redness, without swelling of the subcutaneous cellular tissue, more on the right than on the left side. 36 +

Herpes zoster, excites vesication along the course of nerves and causes large and small vesicles of pemphigus. 11 *

Circumscribed spots on the anterior surface of the left forearm and on the inner surface of the left lower leg, which were totally insensible to pricking and pinching (sixth day). 36 +

A brownish ecchymosis, as large as the palm of the hand, on the lower portion of the sacrum. 36 +

The whole skin was covered with large and small vesicles of pemphigus (sixth day). 36 +

Herpetic vesicles on the temples in the place where the redness hand been noticed. 36 +

Herpes zoster on the left side of the face along the course of the trigeminal; vesicles on the forehead, above the orbit, along the course of the ramus frontalis, on the cheek below the orbit, along the terminal filament of the infraorbital nerve of the chin, along the mental nerve (eleventh day after the poisoning). 36 +

A dozen herpetic vesicles, as large as a pin's head, on the inner portion of the right forearm, somewhat externally to the place where the redness had been noticed; the subcutaneous tissue seemed to be somewhat swollen. 36 +

About twenty herpetic vesicles, as large as a pin's head, along the course of the right sciatic nerve, situated upon a slightly red base; from them some red streaks extend up to the right nates, and from the point of exit of the sciatic nerve to the crest of the ilium. 36 +

Abscesses ecchymosis. 36 +

Burning sensation in the skin, especially of the cheeks, without intense redness, and without elevation of temperature (very soon). 36 +

Formication on both legs. 36 +

Cherry pink hypostasis. 516 +

“Cherry red” appearance of lips and skin (a late finding). 517 +

Skin vesicles commonly develop at pressure points in comatose patients. Bullous lesions may also develop in areas not subject to pressure, and sweat-gland necrosis has been reported. 518 +

Severe carbon monoxide (CO) poisoning can produce several types of lesions of the skin. The lesions vary in degree from areas of erythema and edema to marked blister and bulla formation. These lesions can easily be mistaken for burns or trauma. The bullous lesions heal by eschar formation. The scalp lesion of edema and erythema evolve into areas of alopecia. Mentally confused and to have weakness of the right leg. There was a round erythematous and edematous lesion approximately 6 cm in diameter on the left side of the temporoparietal portion of the scalp. There was a 3-cm, hemorrhagic bulla on the right heel. Interestingly, the previously described left parietal scalp lesion now presented as an area of alopecia. There was a similar area approximately 2.5 cm in diameter on the posterior portion of the scalp. The bullous lesion on the heel had become a dark heavy eschar. Defective memory, parkinsonian tremors, hypothesia of the extremities, and muscle weakness. There was atrophy of the right quadriceps and of the small muscles of the hands. [519] +

Comatose, and bullous lesions were noted on the right forearm and on the right knee. In addition there was an extensive lesion of the left leg with blisters and bullae over the lower portion of the leg. The toes appeared cyanotic. After the patient was admitted to the hospital, renal shutdown developed. On the fourth hospital day, a below-the-knee amputation of the left leg was done because it was felt that the patient had gangrene of this extremity secondary. The patient seemed mentally dull. There was an eschar on the right forearm, geographical in shape, measuring 6

cm in length and 2.5 cm in width. Another eschar on the right knee measured approximately 8 cm in diameter. A smaller eschar of 2 cm was noted on the right elbow. There was an area of alopecia 4 cm in diameter on the right side of the parietal portion of the scalp. The small muscles of the hands were atrophied and neurological examination showed depressed deep tendon reflexes and sustained right-ankle clonus. The skin was diffusely cyanotic, with superficial necrosis of the skin on the anterior portion of foot and ankle. The tips of the toes were bluish-black. The entire foot showed edema of subcutaneous tissue. [520] +

A 25-year-old woman had had burning of the skin for 9 days. The sensation was described as similar to a sunburn. It began on the thighs but within several days had spread to the entire body. Her skin was sensitive and uncomfortable where clothing touched it. In addition, she stated that she felt feverish but that her temperature was normal. The discomfort persisted unchanged during the entire day. On examination her skin was normal. On further questioning she said that she had a roommate who had similar symptoms for a day or two but no longer had them. [521] +

A 39 year old housewife was found unconscious On admission she was unconscious, pulse rate was 92/rnin; hypoactive deep tendon reflexes and bilateral positive Babinsky sign. Extensive dry rales were noticed in the lungs. Dermatological examination revealed dry, pale and pinkish skin, in general. The lips and beds of the fingernails were cherry red in color. There were extensive blisters 2-15 cm in diameter, easily broken and filled with clear fluid, scattered, on a clearly demarcated erythematous base on the arms, lumbar region and the lateral surface of the right glutea and thigh. Edema which did not pit easily with pressure was noticed on the arms. [522] +

A 25-year-old man who survived CO intoxication presented erythematous cutaneous lesions with blister formation in pressure areas. Histologic examination revealed sub-epidermal vesicles with extensive sweat gland necrosis. On admission he was disoriented and tachycardic. Blood pressure was 115/70 mm Hg. The following laboratory tests were abnormal: SGOT 360U/ml, SGPT 141 U/ml. LDH 1.37b U/ml, CPK-NAC and CPK-MB 929 and 890 U/ml. The patient presented several sharply circumscribed, dusky erythematous patches and plaques and large dusky erythematous lesions with tense bullae. The main sites of involvement were the left lower leg, left thigh, inner part of the right foot and the hypothenar region of the left hand. All these areas were along bony prominences against which the comatous patient had lain. [523] +

The postmortem examinations showed bright, pinkish, livid stains and similar coloration of the body musculature and internal organs. 524 +

Cutaneous blisters are common in severe CO poisoning, likely secondary to pressure necrosis. The “classic” finding of “cherry-red skin” is a rare and usually postmortem observation likely due to the combination of CO induced vasodilatation and tissue ischemia. 525 +

With a 5 by 3 cm erythematous area above the left breast with blisters of serous material. Similar lesions were found on the hand, hip, small of the back, right leg, and left ankle. The largest lesion covered the entire dorsal surface of the left hand and extended down to the right small finger. 526 +

Bulla, erythema, swelling, ulcer, gangrene, alopecia. 527 +

A patient who survived CO poisoning developed necrosis of sweat glands, leukonychia, intra-epidermal and sub-epidermal vesicles, a bulla, and intracellular edema and occlusion of the epidermal portion of the sweat ducts. Additionally, there was evidence of acute cerebral cortical damage, acute renal tubular necrosis, hemolytic anemia, and muscle necrosis. Pulse rate 90/min, respiration was 30/min; and temperature was 102.4 F. Cheyne-Stokes respirations. The face was hot, red, and covered with perspiration. An erythematous patch with an irregular border was found on the left thigh and a similar lesion was found on the medial aspect of the left arm; each measured about 3.0 cm in diameter. The left conjunctiva was red and covered with yellow exudate. The right pupil was larger than the left. White blood cell count 27,000, SGOT 1,360 units/ml, and lactic dehydrogenase 2,880 units/ml, a hemolytic anemia with hemoglobin of 6.4 mg/100 cc, Three days after exposure to CO, two vesicles developed in the erythematous area on the lateral aspect of the left arm. The lesions measured 0.2 cm each in diameter. At the same time, the lesion on the lateral aspect of the left thigh developed a bulla which measured 2.0 cm in diameter. The area on the medial aspect of the left forearm showed increased erythema in the region of the sweat pores. Thirty-six hours after admission, the BUN had risen to 156 mg/100cc and creatinine to 17.7 mg/100cc. The urinary output had dropped to 50 cc/day and red blood cell casts were found in the urine. It was thought that he had acute tubular necrosis secondary to hypoxia and muscle necrosis. He again became unresponsive on the tenth hospital day and had several clonic seizures. The BUN was 164 mg/100cc. The patient returned for observation 52 days after exposure to carbon monoxide. At that time, his fingernails and toenails were pearly white for a distance of 4 mm from the nail folds. Bright red flushes and scattered and irregular in shape bullae have been reported. In some patients, decubital ulcers occurred at points of pressure. Our patient's face

and neck was a cherry red color. Vesicles developed in erythematous areas three days after exposure to CO.528 +

The classic cherry-red discoloration of the skin and cyanosis are rarely seen. 529 +

A rare physical finding is cherry red coloration of the skin. It is much more common to observe superficial blisters in dependent areas when patients have been lying comatose for a period of time. 530 +

Cherry red postmortem lividity was present over back. 531 +

The literature on dermatologic lesions associated with CO poisoning is scant. Three types of skin lesions have been described: 1) tense vesicular lesions with an erythematous base; 2) dusky, erythematous plaques; and 3) large, tense bullae bordered by a rim of localized erythema. There is overwhelming agreement that bullous lesions associated with CO poisoning typically appear within 24 hours and are found primarily on the palmar surface of the hands and the plantar surface of the feet. Any areas of pressure or friction are vulnerable to the development of such lesions. These areas include the posterior scalp, wrists, knees, legs, ankles, and dorsa of the feet. The dermatologic examination revealed an 8 × 9 cm bulla on the plantar aspect of the heel as well as a 2 × 3-cm bulla beneath the cuboid, both on the left foot. A localized rim of erythema surrounded each bulla. The bullae were deroofed, revealing contents of an “apple jelly” color and consistency. 532 +

Abnormal skin coloration. Of the suicidal group, fifty-one (63-7%) were considered to be of normal appearance, fourteen (17-5%) flushed, seven (8-8%) cyanosed, four (50%) 'cherry pink' and four (5-0%) pale. Forty-four (75-9%) of the accidental group were described as having normal coloration, three (5-2%) were flushed, five (8-6%) cyanosed, four (6-9%) 'cherry pink' and two (3-4%) were pale. 533 +

Bright red lividity characteristic of CO poisoning. Hypostasis, sub-scalpel tissue, muscles of chest and abdominal wall were cherry red in color suggestive of CO toxicity. The lungs were wet, shining with multiple discrete anthracitic patches and moderate to severe edematous. Petechial hemorrhages as hallmark of asphyxia were also seen over the epicardial tissue and brain. 534 +

Sleep

Suddenly so tired that must go to sleep immediately (after 4 hours). 908.HA.9

Very very tired, little fever. de Sonnaville *

Already very tired when waking up, needs to get going. de Sonnaville *

Very tired when waking up in the morning. de Sonnaville *

Sleeps a lot. de Sonnaville *

Suddenly tired, suddenly exhausted. de Sonnaville *

Tired of least effort, worse talking. de Sonnaville *

Has to rest after, for example: one flight of stairs, has to lie down for more than an hour and can't do anything. de Sonnaville *

"Lifeless, exhausted". de Sonnaville *

Awoke from the nap that felt “unnatural”: it was the unconsciousness of a drugged person, rather than a refreshing, natural sleep (after 5 hours). 908.HA.9

Took a 2-hour nap at 4pm—again, an “unnatural”, almost narcotic sleep (seventh day). 908.HA.9

Sleepiness at 1 p.m. when sitting (Day 2). 908.HA.3

Extreme sleepiness at 1 p.m., could barely keep my eyes open, lasted until 4-5 p.m. (Day 14). 908.HA.3

Sleepiness at noon (Day 20, 30C). 908.HA.3

Sleepiness at noon (Day 3, 200C). 908.HA.3

Sleepiness from 1-3 p.m. (Day 5, 200C). 908.HA.3

Sleepiness in the afternoon, take a nap from 4-6 p.m. (Day 9, 200C). 908.HA.3

Sleepiness from 10 a.m., at 12 p.m. I try to sleep, but I cannot fall asleep (Day 10, 200C). 908.HA.3

Sleepiness during day (Day 16). 908.HA.3

Sleepiness evening, after supper around 9:30 p.m. immediately back to bed and slept (Day 5 30C). 908.HA.4

Dog-tired after 9 p.m. Legs become restless, must go to bed early (Day 4, 200C). 908.HA.4

Tired and sleepy late afternoon, slept 1.30h deeply and firmly (very untypical for me) (Day 5, 30C). 908.HA.4

Sleepiness, fell asleep while riding as a passenger in the car for a short trip (unusual for me) (Day 2, C6). 908.HA.8

Yawning with lachrymation. 908.HA.2

Prolonged sleep (9,5 hours) (Day 15). 908.HA.3

Unusual long sleep until 8.30 am (Day 8, 30C). 908.HA.4

Sleep very well and deep, difficult to wake up in the morning (Day 3). 908.HA.7

Morning in bed fall asleep again and again (Day 4). 908.HA.7

Deep **prolonged sleep**; sleepiness for several days. 72 +

Sleepiness. 36 +

Great sleepiness for several days. 36 +

Drowsy, but unable to sleep on account of the headache and pains in the stomach. 36 +

Somnolence. 36 +

Sound sleep (third night). 36 +

Sleep deep and prolonged, interrupted by cramps in cheeks and toes. 36 +

Never slept so long before. 36 +

Sleep deep, prolonged, interrupted by cramps in cheeks and toes. 85 +

Drowsiness. 535 +

Great sleepiness for several days. 36 +

Never slept so long before. 36 +

Drowsy and kept drifting back to sleep whenever left alone. 536 +

Awake several times first around 4 a.m. till 6.30 a.m., then got up (Day 5 30C). 908.HA.4

Awake around 4 a.m. in the night (Day 8, 30C). 908.HA.4

Awakens at 3.40 a.m. and around 6 a.m. (Day 2, 200C). 908.HA.4

Awakening at 4 a.m., fell asleep after short time (Day 4, 200C). 908.HA.4

Waking up a few times at night (I usually sleep well), but went back to sleep right away (Day 3, 30C). 908.HA.8

Restless sleep (Day 1,2). 908.HA.3

Relationships

Antidoted by Coffea.

Follows well after

Pulsatilla. 908

Cases

1- **A comparison of carbon monoxide and lues.** In January 1919, a male patient, bachelor 55 years of age, silk weaver, came for consultation. He complained of some rectal trouble, simulating hemorrhoids, meanwhile some brown, lentil-like pigmentation here and there. History: He lives in an attic, in a small room, where he does light housekeeping. His only room is lighted by gas. His illness began with a cough, followed gradually by increased weakness and dyspnea. The cough was worse during cold day, when the windows were closed. Five or six days later anal bleeding, as in hemorrhoids, occurred. He did not complain of piles, but in childhood he had suffered from prolapsus recti. He, on his own initiative, used Daly's Pain Extractor, a patent salve.

In December 1918, the cough became much worse. He finally changed his position in bed, head on opposite end, but then smelled gas and his cough grew worse. He realized then that gas was escaping from the pipes located near this end of the bed, and he located the leak, there being a sufficient amount of gas to make its escape through the defective faucet. This was attended to and the cough as well as dyspnea gradually stopped.

Depending upon this clear - cut history and looking in Allen's *Encyclopedia*, Carboneum oxygenisatum in Vol. X, pages 442-443, we find: “Face pale, covered with copper-red spots of various size and forms, which are also found on parts of the trunk and extremities; short cough, oppression, dyspnea, constriction of sphincters.”

In the absence of any other history but that given, I attributed his condition to a slow poisoning by illuminating gas.

In a week or so he reported that his rectal condition was now normal under the use of the above-mentioned ointment.

In January 1919, a week after my first examination, I noticed a binocular iritis, and administered Mercurius corrisivus 3X; meanwhile a blood examination was made on the 11th of February 1919 and a strong four plus Wassermann reaction was found. We were now facing a case of Lues, camouflaged by carbon monoxide objective symptoms.

How came this contamination and the initial sore? The man had an initial lesion at the anus. He was put under strict specific treatment and is now a well man.[537]

2- During the Covid-19 pandemic, Carboneum oxygenisatum in the 200 and 1M potencies was tried in elderly people who were severely physically and mentally handicapped and were in the severe/critical stage of Covid-19.

Three days after its introduction in a nursing home in Lyon, France, Dr. Frédéric Rérolle wrote on Friday April 10 evening before retiring for the Ester weekend, “Here is our first case in which Carboneum oxygenisatum was prescribed. I can confirm that the severe forms of Covid-19 that I have unfortunately had in older people have presented a picture very similar to the MM of Carboneum oxygenisatum.

“It is still too early, but I am happy to be able to give you the beginnings of the first results on a patient who has been seriously affected since at least March 23 and who was managed to stay alive but without stable results, forcing us to switch from one remedy to another with a saturation which remained low between 83-(87% under Carb-v.) under O2, an oscillating fever and severe breathing difficulties.

“Under Carboneum oxygenisatum 200 and then 1M since the evening of April 8: the saturation rose quickly to 90, 93% and remains stable, no fever and very good clinical improvement on auscultation. The whole team finds him transformed, rejuvenated! To be followed but after many failures and deaths I regain hope.”

By the end of the epidemic on April 19, at least three of these patients were saved by the use of Carboneum oxygenisatum.

My experience in a nursing home. I had heard that a colleague, Dr. Frédéric Rérolle, had an epidemic of Covid among his residents in a nursing home in Lyon,

France. He was the only physician on staff, around the clock seven days a week for 120 residents mostly between 85 and 105 years old.

The nursing home was on three levels:

- Ground floor: 27 Alzheimer residents

- First floor: 53 non-autonomous and severely mentally (i.e., dementia) or physically (i.e., quadriplegic) handicapped residents.

- Second floor: 40 less dependent residents.

On March 17, when Dr. Frédéric Rérolle heard of the beneficial use of Camphora in Iran, he gave one dose of Camphora 1M on two successive days to 118 out of 120 residents and the majority of staff.

On March 19 and 20, about 15 residents of the second floor only developed diarrhea, which was not serious and self-limiting.

From March 19 onward, the first 6 cases with flu-like symptoms with fever made their appearance, but only on the first floor. And then about 9 others became febrile in the following days. Fred thinks that eventually all 53 and certainly 40 of the first floor residents became infected. Within two days 2 had died. In total 11 have died (10 in the nursing home and the only one who was hospitalized).

Incidentally, the two residents who refused to take Camphora were among the first ones to die of the 11 who have died of Covid. When Fred saw that the epidemic was taking hold of the first floor, he treated everyone affected with Bryonia. All got better quickly. However, two days after he stopped giving Bryonia a number of these relapsed. He resumed Bryonia and they all improved again. However, some of these became worse with time. And for these we had to use remedies adapted to each case which saved 6 severe/critical cases.

He has had no Covid cases on the ground floor or second floor. However, no staff was aloud to cross from the other floors to the first floor without changing their entire PPE.

Dr. Rérolle in the begining had access to only 3 SARS-Cov-2 test kits, and was able to get more. So finaly only 12 patients were tested for Covid and 9 tested positive. However, the 3 who tested negative must have been false negative as they presented a typical clinical picture of Covid-19, and two of these died.

Fred wrote, “For the follow-up of very serious cases, I was helped by André Saine (whom I thank again) who, thanks to the time difference, ensured the follow-up at

night by telephone from Canada, because for some the vital prognosis was very pessimistic and the remedies had to be adapted at every moment. Many cures for severe cases of two or third stage disease have been tried. I can mention Carbo vegetabilis, Kali carbonicum, Beryllium metallicum, Ammonium carbonicum, [Antimonium tartaricum], Opium, Arnica, Belladonna, Gelsesmium, Arsenicum album, etc. which had temporary effects but no cure. The most useful remedies that have cured serious cases (desaturation requiring oxygen by mask up to 9 l/min) are Carboneum oxygenatisum (Carbon Monoxide) for asphyxic forms and Hyoscyamus for major confused forms and a case of Ammonium carbonicum given at the very beginning of lung congestion before desaturation.

For the convalescence phase, I have the impression that Kali carbonicum or sometimes Opium have a positive effect. I don't have any experience with Silica yet.

And for the early phase, Bryonia still in the lead ...”

To support Dr. Rérolle in his heroic task, I offered my help on April 1. I did 19 consecutive night shifts of 9-12 hours each, from April 1 to April 19. Dr. Rérolle would come to the nursing home at 9 A.M. and would leave at around 8 P.M. and sometime later. I would take over from about 8 P.M. until 5-8 A.M. the next morning Central European time (CET) or 2 P.M. to 11 P.M.-2 A.M. Eastern time.

I did this despite having an already extremely busy practice of my own. It is interesting to note that after the introduction of Carboneum oxygenisatum on April 7, there were no deaths for 10 consecutive nights and days.

Personally, I was in shock when two patients (Madame Jacquy and Madame Perrot) died under my care, as in forty years of practice and close to 300 cases with pneumonia of all degrees of severity I had never lost a single case. I was in shock partly because 2 nights before I had stayed up until 2 A.M. my time to keep Madame Perrot alive. She began the night with an oxygen saturation index at 84% and a pulse at 65. However, at 2 A.M. (CET) she began to desaturate down to 70% with a pulse at 48. I was in contact with the nurse every 20-30 minutes until 8 A.M. (CET) and we were able to stop the downhill spiral she had entered and kept her alive until Dr. Rérolle was able to arrive at the nursing home at 9 AM.

Madame Perrot died on the night of April 18. She had been relatively stable all evening with an oxygen saturation index (OSI) of 73%, 75%, 77% and 77%. Alexandra, the nurse, knew she needed to keep a close eye on her. So, when I phoned around 2:15 A.M. (CET) to obtain the reports of the 6 patients under observation, Alexandra told me that she had just finished examining Madame Perrot who had a temperature of 36.0, an OSI of 68% and pulse rate of 58. She told me that her

breathing was less labored and shallower. I knew that she was sinking again as she had done at the same time two nights earlier. I then asked Alexandra to go back to her room to examine her further while I remained on the phone. Alexandra told me that, as she was approaching her, she didn't seem to be breathing anymore and that she had become completely relaxed. I asked her to get her OSI and pulse rate, which were 66% and 49 respectively, and at that exact moment Alexandra told me that her pulse had just stopped. Afterwards she said that she had gone quietly like an angel.

Alexandra and I took a rare moment of the night to talk about our experience in the treatment of these severe and critical cases of Covid.

And at this moment, I learned a bit more about Madame Perrot. She was 91 years old with dementia from Parkinson disease. She was completely non-autonomous. She was in diapers and had to be fed by the spoon. She had not talked or moved her limbs in over two years and no one had seen her eyes during this period of time.

From long personal experience and the one of other homeopathic physicians, it is difficult to imagine a person dying from pneumonia as long as a competent homeopathic physician is at the bedside of the patient and has an adequate access to homeopathic remedies, even in patients in the direst condition, such as the ones who are on their deathbed, whether the implicated microorganism is viral, bacterial or fungal, and regardless of the severity of the illness, the underlying complications such as immune deficiency, heart failure, kidney failure, lung cancer or meningitis, or the age of the patient, as in centenarians left for dying without anymore treatment, or in patients infected with resistant microorganisms.

I strongly believe that in a better therapeutic environment, a patient even as frail as Madame Perrot should not die of pneumonia.

In order to achieve this, a greater number of personal would be required to monitor patients more closely with direct access to a homeopathic physician and to an adequate supply of homeopathic remedies.

It would even be better if the homeopathic physician was able examine each patient individually and have access to the prior history of each patient.

Four positive outcomes

Despite the very difficult and unfavorable therapeutic conditions at the nursing home (greatly understaffed, poor access to remedies and poor range of potencies,

and in my case no direct access to the patients and no access to the file of the patients) there are positive aspects of our experience in this epidemic at the nursing home, which are:

Certainly 22 of the 40 of the highly mentally or physically handicapped and non-autonomous patients who were clearly infected and perhaps up to 35 of the 53 of these who are suspected of having been infected were saved from going into the severe or critical states.

Not a single patient on the ground and second floors and no one of the health care personal was infected.

Six of the residents were saved out of the severe or critical states.

For 10 consecutive days and nights we had as a rule 6 patients in and out of the severe or critical states and we had no deaths. However, eventually everyone became exhausted and the quality of care suffered.

These four positive outcomes would not have been possible without the use of homeopathy for both the prevention of the patients and health care personal from becoming infected and for infected patients from progressing into the later stages of the disease, and for saving the lives of 6 patients out of the critical stages of the disease despite the fact they were all highly handicapped patients and working with extremely deficient symptomalology, as I was mostly limited to base my prescriptions on the changes in a few objective symptoms, such as the temperature, the oxygen saturation index, the pulse rate, auscultatory exam and the demeanor of the patient. Saine 2020

3- **A suspicous COVID-19 case.** March 23, 2020: M. vd S, a woman of 60 years old came to me with chief complaint of migraine. This went rather well with Natrum muriaticum and since on occasion Aconite for its acute manifestations. She now calls as she has been ill for two weeks. She received antibiotics from her MD because for a chlamydia infection a week ago.

She is indeed quite ill with a temperature of 39.5C in the evening; she is tired, she is very chilly, but her complaints are not very impressive and vague: a bit of pain in the abdomen, she wants rest. She has a cough, but no clear modalities for her. She has slight hay fever symptoms (bit sneezing, some irritation of the eyes).

She thinks she has Covid-19.

It started two weeks ago with nausea and vomiting: when she did drink a little, she had to lie down very quietly otherwise she would vomit. She had a headache on the right side, better cold applications and rubbing.

Now she can eat, desires salt, is thirsty for warm drinks and has aversion to cold drinks (this last one is normal for her).

Bryonia 200, 3 times: morning, afternoon and evening.

March 26: At first she became a bit better but now she is clearly worse: headache, nausea, sore muscles (now she wants to move) and she started to cough. The cough is dry, worse warm to cold, talking, excitement, standing and walking and better lying, rest is worse cold drinks. She prefers warm drinks. Sneezing worse warm to cold, undressing.

I ordered Rumex 200 to be sent to her, and let her continue taking Bryonia 200 four times a day. If this would work well, she should then continue taking it.

March 28: She became better: 38˚C, on the 26th she was nauseous and had a diarrhea, that's gone by now. She feels better, less muscle pain. Cough slightly better. But today she is nauseous because of her migraine. When she is ill she often gets migraine. She has a rather stressful relationship.

Go on with Bryonia 200 and for the migraine Aconite 200 3 times every hour.

March 30: The cough is better, no muscle pain, temperature 37.7C this morning. Less ill but still her migraine is there.

Bryonia 200, 2 times a day and Acon, 10M 1 time (she did have this remedy at home).

April 3: She is still ill, she feels it in her lungs and her whole body. But it is a bit better.

Tired, headache, a bit nausea. Her temperature can change: about 37, a bit later about 38, and a bit later again about 37.

Bryonia 200 4 times a day.

April 9: She reports by email that she is a bit better, but it is going up and down. More fever: 37.7 to 38.2

She is very tired, heaviness of her head, sore muscles, constriction or pressure of her chest, some cough, a bit nauseous, change and loss of smell and taste. I asked her to phone me.

April 10: She is a bit better, but remains ill: 37.7C in the morning, very tired, slight headache on waking, a bit nauseous, and she feels her lungs: a slight burning when breathing.

Her migraine got only slowly better after the Aconite 10M (but that was better than expected).

Bryonia 1M, three times a day and Aconite 10M 3 times every hour if needed.

April 16: She got the remedy after 4 days. In between she took the Bryonia 200 more frequently.

After taking the Bryonia M she is a bit better: the fever is gone, her lungs are still hurting, and she has the feeling she needs more air, she is a bit oppressed. Still a bit nausea.

She started doing more: a bit of walking and biking, but she is very tired.

She does not feel herself or her old self.

Bryonia 1M at 8 and 9 am and 8 and 9 pm. When no effect: at 8, 9 and 10 am and pm.

May 5: No fever anymore for 10 days. The progress is very slow. She still is very tired. At times she suddenly is prostrated worse with trembling and perspiration, especially about 6 or 7 pm.

She still feels some burning in her lungs and a bit oppressed. She is a bit anxious and fears she never will be totally well.

At times she has a migraine in the early morning (about 4 am). 4 times Acon. 10M makes it disappear. Carboneum oxygenisatum 200K once a day

May 15: She got the remedy on May 11.

She is better, no cough, no pain in lungs anymore, no fever, no nausea.

She did not have a headache anymore, which is much better than expected, in particular because she had much stress by ending her relationship.

But she is very tired and her physical fitness is very bad.

She has to go to work now.

It was striking for me that she just waited, not asked me about the remedy not arriving. She was a bit anxious but at the same time quietly waiting, a greater passivity then I know from her.

Carboneum oxygenisatum 200 once a day.

June 6: I did not hear anything from her. So I wrote her an email and she wrote back that she was improving day by day. She is building up her exercises and her condition is getting better but slowly. After the remedy things started being better.

She only feels a very slight restriction in her lungs. She is a bit worried about her body: are her kidneys, heart et cetera damaged?

She stopped the remedy 6 days ago, it seemed to do nothing anymore.

Carboneum oxygenisatum MK once a day. de Sonnaville 2020

4- **A COVID-19 case with positive testing.** March 13: A woman of 30 years. I know her as a very nice, lively and rather reserved person who takes care of her own business on her own. She coughs, but feels not ill except for sometimes some hours: tired, a bit headache, sometimes nausea. One time she vomited after eating many rather old nuts. Cough worse after going to bed, evening.

Sensation as if there was mucus in her lungs. She tries to cough it out but no expectoration. Sometimes she has occipital headache. No clear modalities. All the symptoms are vague and unclear. And changing a bit every now and then. Sleep, appetite, digestion, et cetera: no symptoms. Her energy is well. Bryonia 200 3q8h

March 30: She took Bryonia 200 and later on Bryonia 1M, in several frequencies, but they did not bring much change. She is not feeling well. She sounds a bit subdued and resigned. Because of her work she could be tested for Covid-19 today.

April 16: She tested positive for Covid-19. The first two weeks after testing positive for Covid-19 she was in quarantine in her own house. She liked it: nothing to bother! She started to finish things in her home, painting things, repairing doors et cetera. Tuberculinum 200, 3q8h for three days.

May 6: The remedy did nothing. She has to work again, but she feels as if everything is too much and desires rest. She is a bit dull, has *less initiative* and still coughing every now and then. She is not feeling well, but she can not clearly explain it. She needs more rest and is tired. Carbn-o. 200 3q1h morning and evening.

June 9: She took at first Carbn-o. 200 as prescribed and later on Carbn-o. 1M 3q8h. She can only say by hindsight that she feels better. Not totally well. She still sometimes has a cough but no irritation in her lungs anymore. She is more aware that she is busy with making essential choices in her life, choices about her work, her attitude and the future. She feels that that is costing her lots of energy.

She started to eat vegan 3 weeks ago. She worries a bit: could this be a part of her lack of energy? She sounds better: more lively, more jokes, quicker. Her energy is about 50% better. de Sonnaville 2020

5- **Another suspicous COVID-19 case** (she is the partner of case #4). March 20: A woman also about 30 years old. She got muscle pain, mostly in her back and shoulder blades. Then the headache came. Now she has already for 10 days a temperature between 37.7 (in the evening) to 38 and above in the afternoon.

She coughs, worse exertion, rather dry, only a bit expectoration. She is tired, worse morning in bed, exertion, she “comes to nothing”.

Chilliness, especially het torso. No thirst. Aversion to cold drinks. She is dull. Gels 200 three times (every hour) a day

March 23: She is a bit better, but not much. No clear other indications. Gels 200 twice a day three times

March 25: Her energy became better, she did several things, only in the mornings. The cough worsens, dry, worse on rising, on lying down, when moving, while eating, while talking. She coughs from irritation in her throat. She has pain in the cervical area, worse with higher temperature, better leaning backwards in her sofa. She is more thirsty. Bryonia 200 3x/8 hours (three times a day).

March 28: Generally better, the fever is gone. But she coughs more. It Is more difficult to fall asleep. She has an irritation in her lungs. The feeling that there is mucus in her lungs under her sternum, but there is no expectoration, dry cough (dryness particular in the morning). Her throat is dry. Cough worse eating, evening in bed, when beginning to walk. Bry 200 3q1h, 2 times a day.

March 30, morning: The cough is worse. More and more dry, as is her throat. At first it was better. Yesterday it became worse, and more difficult respiration. Dryness of throat. Her menses began yesterday, and because of the pain she took 2 Ibuprofen (!). No new symptom. Bryonia 200 4xq30 min.

March 30 afternoon: She thought the Bryonia did nothing, so she took the Gelsemium again, 3q1h. She feels better, throat less dry, less irritation, less cough. She

was more thirsty. Continue with Gelsemium 200 3xq1h now and I sent her Bryonia 10M.

April 3: She took the remedy 3q3h, her cough is better, generally better, no new symptom. Continues the same.

April 8: No fever anymore, no headache, sore muscles, no pain in lungs. Energy a bit better, less cough.

April 15: More burning in lungs, respiration more difficult, very tired. More perspiration.

April 26: She still is coughing, worse eating. After Tuberculinum 200 her throat got worse: pain. So yesterday she took Bryonia 1M again, 3q1h.

This morning for the first time her nose was clear. Smell and taste are normal

New: worse when hungry (tired, weak) better after eating. New: throat pain worse morning, better ice-cream. Phos. 200 once a day for three days.

May 6: Now she has headache again. Her symptoms are changing a bit every time. Now she feels a pressure on her chest, a constriction, with a feeling of difficult respiration, worse exertion, walking. She feels a bit light headed. She is very tired and sleepy, sleeps in the afternoon now. Better warm bathing (she always loves bathing!). Throat pain is a bit better, and worse from warm tea. I hear a bit of despair of recovery. The Phosphorus helped her, but then her menses came: April 29 to May 3. She took ibuprofen again and became worse (!). Carbn-o 200 3q1h morning and evening.

May 8: This morning much coughing, now with some mucus (she swallows it). Pain attachment of sternum and between the shoulder blades (cramping, sore) worse coughing. The constriction of the chest is gone. The burning in her lungs is much better. Generally better, more hope of getting better, more initiative. Carbn-o. 200 3xq1h morning and evening.

May 18: Worse again, no energy, feels not well, burning in lungs again. Carbn-o. 1M 3xq8h.

May 28: The Carbn-o. 1M gave her *a boost!* Things went better and better. No symptoms anymore, she is back on track!

June 8: Everything is okay. de Sonnaville 2020

6- The first case of Carboneum oxygenisatum was of a 18 month infant with celiac disease who *kept going round in circles* all through the 90 minute session, whenever he was away from his mom's lap or busy climbing up and down. Lot of restless energy, unlike the one seen in Carbo vegetabilis.

Only remedy under generalities in the rubric, Circles, inclination to go around.

He was on a gluten free diet.

But his mom during pregnancy would get severe dyspnea during which she would crave carbonated spring water and needed to actually keep sipping on that while sitting out in a temporary tent just outside their home, even in December in Vancouver.

The combination of both would provide some relief.

This aspect was given importance while selecting the remedy for the infant who responded very well and started to tolerate restricted foods over a period of time and started to gain weight rapidly. This case was followed for over two years. Anand 2020

7- The other 2 cases were of a 12-year old girl with fibromyalgia and an elderly woman on 75 who had bad bronchitis.

Both would find great relief from **carbonated drinks** and stopped craving it as they improved.

The need for carbonated drinks would reduce in each of these cases along with their progress.

Since then I have also confirmed the same in Carbo animalis, Graphites, Petroleum and Petroleum raffinatum (unleaded petrol).

So in that sense they were cured symptoms. Anand 2020)

8- **Proving of Carboneum oxygenisatum 6C, 30C and 200C**

Proving was done in the spring and summer 2020 with 9 provers.

908.HA.1: female 59y

Proving done in June 2020. The prover took 3 doses Carboneum oxygenisatum 30C three globules dry on the the first 3 days of the proving. The symptoms lasted for 11 days

908.HA.2: female 72y

Holding the Carboneum oxygenisatum 200C two times in her hand for 15-20 minutes, symptoms occur only during that time

908.HA.3: female 44y

Took only once one granule of Carboneum oxygenisatum 30C. The symptoms lasted a long time for 25 days in July/August 2020

Took only once one granule of Carboneum oxygenisatum 200C in September 2020, the syptoms lasted 17 days.

908.HA.4: female 61y

The proving was started at the holiday resort in Brissago/Switzerland, i.e. after a car journey of about 1200 km over 2 days.

Took Carboneum oxygenisatum 30C 3 days in a row, one granule more every day and observed for 4 days after that. Took Carboneum oxygenisatum 200C 3 days in a row, one granule more every day and observed 21 days after that. Very hot weather during proving

908.HA.5: female 61y

Proving done in July 2020. The prover took 3 doses Carboneum oxygenisatum 30C on the first (1 globule), second (2 globules) and forth day (3 globules) of the proving. The symptoms lasted for 8 days

Beginning of May 2020 she had Covid 19 infection, the symptoms lasted about 2 weeks, at the end Arsenicum album helped a lot. The remaining symptom after the disease was that she could not breath deeply, deep inspiration caused an irritation of cough. This symptoms became much better during the proving with 30C and disappeared later after a dose of Carboneum oxygenisatum 200C

908.HA.6: female 58y

The prover took Carboneum oxygenisatum 30C several times over a period of 17 days. Then she observed for another 5 days. Then she took a total of 4 doses of 200C ocer e period of 6 days and observed a week after. Often sultry hot weather during proving.

908.HA.7: female 52y

Took Carboneum oxygenisatum 30C on Day 1 and day 3, very hot wether during proving. The reaction started quickly, but after a few days it was over.

908.HA.8: female 57y

Proving done in August/September 2020

Took one globule of Carboneum oxygenisatum 6C on August 15. Only mild symptoms after 6C for 3,4 days

Took two globules of Carboneum oxygenisatum 30C on September 14. Much stronger symptoms, after a week the proving was antidoted with Bryonia and Arsenicum album, symptoms lasted in less degree for some days after that.

908.HA.9: female 47y

Took one dose of Carboneum oxygenisatum 30C once on June 18, 2020 and experienced symptoms that began immediately and lasted for one week.

Generalities

Tiredness

Very tired in the morning. Had difficulty just getting out of bed. The whole day exhausted. Nothing made it better. After 6 p.m. a bit better, but still tired (Day 4). 908.HA.1

Unusual exhausted on waking. Can hardly keep eyes open. Slept well during night but still felt exhausted on waking. Lasted most of day. Nothing really made it better or worse, just time. Better by dinner time. Felt hungover most of day (Day 6). 908.HA.1

Sluggishness, weak and sleepy whole day, better during driving in car, but came back after returning home (Day 6). 908.HA.3

Low energy at 4 p.m., need to lie down (Day 7 and 9). 908.HA.3

Energy very low with hot weather (Day 11). 908.HA.3

Very sluggish and listless at noon (30 degrees Celsius) (Day 5, 30C). 908.HA.4

Very tired and limp in the afternoon (Day 5 and7, 30C). 908.HA.4

Weak and tired all day long (Day 4 and 5, 200C). 908.HA.4

During the whole day I feel extraordinarily tired and exhausted (Day 1). 908.HA.5

Tired and exhausted (Day 1). 908.HA.5

All day strong lassitude. (after 30C) 908.HA.6

During the proving more sensitive to sultry hot weather than usual. After cooling down to 28 degrees Celsius significant improvement of all symptom. (after 30C). 908.HA.6

Exhausted, lazy, not imaginable to move my body (Day 3). 908.HA.7

Easily exhausted from exercise, desire to stop and to go to sleep, too weak to move (Day 4). 908.HA.7

Heaviness in the whole body, very tired (Day 2, 6C. 908.HA.8

Mild weakness and feeling somehow sick after rising in the morning (Day 3, 30C. 908.HA.8

Later during the day more weak and strong desire to lie down, while resting in bed, I felt almost normal (Day 3, 30C. 908.HA.8

Weak, want to rest, not work, like having a flu, desire to stay in bed and not move. (Day 4, 30C. 908.HA.8

I rest in the afternoon, want to sit or lie without moving (Day 5, 30C. 908.HA.8

All the symptoms seem stronger when I get tired or work too much, they get better when I rest. Walking outside (which usually helps to improve my symptoms when sick) does not make me feel better, I would rather prefer to rest (Day 7, 30C. 908.HA.8

Feeling cold (Usually quite warm) (Day 6). 908.HA.1

When I put on a sweater, I quickly got too hot, when I took it off, I felt too cold again although it was a warm day outside (Day 3, 30C. 908.HA.8

Mix of hot and cold feeling as if unable to regulate temperature (Day 4, 30C. 908.HA.8

Feverish feeling, but no temperature (Day 5, 30C. 908.HA.8

Strong desire for fresh air, and cool air seems the best. A stuffy room seems unbearable. as soon as I can be outside or near an open window I feel definitely much better. I want to be outside, but not move, sitting or even lying makes me feel better (Day 5, 30C. 908.HA.8

Mind

Chilly but almost an indifference or apathy to become warm (immediately). 908.HA.9

Too apathetic to even breathe. 908.HA.9

Utterly calm; barely breathing and it doesn't bother me at all. Pulse 88. (first hour). 908.HA.9

Feels slightly stupid (first hour). 908.HA.9

Happy to sit still in my rocking chair and disappear into a perfect reverie (daydreaming), from which I can pull myself out of this state easily (but I don't want to). (first hour). 908.HA.9

Aware that her face was utterly still and relaxed, a pleasant numbness; blinking occasionally, but dreamily (first hour). 908.HA.9

Daughter is speaking to her and she registers that she is talking, but she doesn't pay attention to a single thing she's saying. She can, but she doesn't want to (first hour). 908.HA.9

While eating started to tremble from the cold, yet was unwilling to do anything to warm up (second hour). 908.HA.9

After eating, my mind started to clear and was able to think more linearly and logically (second hour). 908.HA.9

Since yesterday morning, I have been experiencing a kaleidoscope of symptoms that are unremarkable, insipid, and utterly unworthy of remembrance and changing every 5-10 minutes. They have no discernible relationship to each other. I have absolutely no desire to write them down, and have no desire to make any effort whatsoever to dignify them with the tiniest whiff of posterity (second day). 908.HA.9

Indolence not for daily activity, but only to write the symptoms of the proving (fourth day). 908.HA.9

I am sensitive to the remedy (and further exploration will be necessary), then Carbn-o. is a slow acting, insidious remedy with no discernible characteristic other than its insidious, unremarkable, and forgettable nature that can manifest in any conceivable way with a degree of apathy that would be *very difficult (if not impossible) to quantify and qualify in practice.* 908.HA.9

Poetic feeling (first hour). 908.HA.9

Brain fag significant. Thoughts felt slow and imprecise. Difficult recalling words. Not clear thinking (Day 6). 908.HA.1

Very difficult to think, I am unable to follow my own thoughts in the evening (Day 7). 908.HA.3

Dazed in the head, clear and concentrated thinking not possible. (after 30C). 908.HA.6

Hard to concentrate, like in fog, dizzy, not focused (Day 3 and 4). 908.HA.7

Hard to collect my thoughts (Day 4, 30C. 908.HA.8

Was only able to read a mindless easy novel, but not able to read a more difficult text or something work-related (Day 3,5, 30C. 908.HA.8

Lazy, postponing sport, unmotivated (Day 2). 908.HA.7

Forgot to brush my teeth in the evening before going to bed, had to get up to do so. Forgot to take my eye-drops, had to get up again, unusual for me (Day 1). 908.HA.7

Went in the cellar several times to get things and forgot what it was (Day 4). 908.HA.7

Time passes more slowly during a Qui Gong exercise. (after 30C). 908.HA.6

Vertigo

Dizziness and tired all day (Day2). 908.HA.7

Strong dizziness, like getting black before eyes, like almost fainting, need to lie down and close my eyes, physical and mental weakness (Day 2). 908.HA.7

Head

Outer Head

Head hot, cool damp forehead (4 p.m.) (Day 5, 200C).908.HA.4

Head Pain

Pressing headache

Pressing headaches in the morning on waking (Day 6, 30C). 908.HA.4

Head pressure after getting up (Day 2, 200C). 908.HA.4

Head pressure in the morning (Day 3, 200C). 908.HA.4

Head pressure on the forehead on awakening, forehead (Day 4, 200C). 908.HA.4

Head pressure all day long (Day 4, 200C). 908.HA.4

Pressing in head (Day 5, 200C). 908.HA.4

Head pressure at 2:30 p.m. after physical exertion (32 degrees Celsius) (Day 8, 30C). 908.HA.4

Broad pressure on the vertex at 8 a.m., which disappears in the open air (Day 2). 908.HA.5

Pressing pain on the vertex again around 10 p.m., which goes away with sleep (Day 4). 908.HA.5

Constant, non-descript frontal headache (2/10) with an ever-so slight clenching of the teeth (second day). 908.HA.9

Slight head pressure rising from the occiput directly at the transition of the cervical spine and skull calotte and rising on both sides in a hood-like manner. On the left side a painful spot (this is where I used to hit the concrete wall when I fell). Head pressure on the forehead and neck better after a cold shower, cold foot baths and a cold wet cloth on the forehead. Sensitivity to light, darkness improves, after two hours of sleep in the afternoon between 2 p.m.and 4 p.m. the head pressure is significantly better.Had strong shoulder and neck myogleoses. (after 30C) (Day 17). 908.HA.6

Headache in the morning on waking

Headaches in the morning when waking up (Day 9, 200C). 908.HA.4

Headaches morning when waking up, throbbing all over the head for several days. (after Day 10, 200C). 908.HA.4

Headaches morning when waking up on the top of the head with sensitivity of the brain, like a mild soreness for several days (after Day 10, 200C). 908.HA.4

Headache and facial pain better after getting up, better by movement (swimming) and better after the first cup of coffee at around 9 a.m. (after Day 10, 200C). 908.HA.4

Headache left temple at 8 p.m., extending to left eye and left jaw, pressing, went to bed at 10 p.m. still with headache, now focussed in left orbita. (2 hours after 30C) (Day 1). 908.HA.7

Headache left temple extending to left eye and yaw (again 2 hours after 30C) (Day 3). 908.HA.7

Headache on the left side from forehead to occiput in the afternoon, pretty intense 7/10, but it lasts only a few seconds (Day 4, 200C. 908.HA.3

Migraine

Headache with nausea (Day 2). 908.HA.6

Increasing nausea during the morning (Day 2). 908.HA.6

Around noon for three hours more severe nausea with dryness in the mouth, must lie down, vomited three times. Every movement such as turning around in bed and getting up from bed worsens the nausea; talking, being spoken to, eating, drinking, stronger pressure on the stomach worsens the nausea; laying hand on the stomach improves slightly (Day 2). 908.HA.6

Lie very still, in the dark, do not want any noise, so nausea and headaches are more bearable. Cold and damp cloth on forehead and eyes relieves headaches, a bit also the nausea. With the nausea and headache a feeling of fullness, bloated abdomen, ice-cold dry hands and a slight feeling of inner cold. Short sweats only after lying down (Day 2). 908.HA.6

Slight relief of nausea after vomiting. After the third vomiting with violent choking and only bilious vomiting, the nausea disappears and shortly after that the headache disappears as well. Afterwards very strong weakness and tiredness, face paleness and freezing, wants to continue lying in the quiet, dark room and sleep a little. Sleep about 1 hour. - After that I can get up again, I am still a little weak but very clear in my head and can meet friends, long train ride, a lot of talking. Strange,

pleasant clarity in the head, feeling of calmness and balance, slow thinking, more sensitive to outside stimuli and altogether more sensitive . Watery, painless diarrhoea in the afternoon. (after 30C) (Prover had migraine in past, but usually during stress, at the time of the proving in vacation, no stress) (Day 2). 908.HA.6

Eyes

Heavy feeling in eyes (Few minutes after 30C for some time) (Day 1). 908.HA.1

Heavy feeling in eyes,can hardly keep eyes open (Day 6). 908.HA.1

Pin-pick pupils of both eyes (after 3 hours). 908.HA.9

Right eye discomfort, soreness and senstive to light. Have to keep squinting that eye. Better with closing the eye and putting pressure on it and keeping lights low of off (Day 6). 908.HA.1

There is something wrong with the eyes, but its nature is eluding. It is almost as if their inner light is dimmed—a dampening of intelligence, charm, and wit (3/10) (second day). 908.HA.9

Floaters in the eyes in the morning on awakening (Day 3, 30C). 908.HA.4

Light pain behind the eyes while driving, for 30 minutes (Day 11; 200C). 908.HA.3

Ears

Earache left morning on waking, better by moving the jaw and pulling on the auricle (Day 2, 30C).908.HA.4

Ears very blocked and squeaked and popping Day 5). 908.HA.1

Hearing

Suddenly ear ringing (minutes after 30C for 30 minutes, two times after the 30C) (Day 2). 908.HA.1

Ears very blocked and squeaked and popping (Day 5). 908.HA.1

Crackling in ears (Day 7). 908.HA.1

Nose

Burning dryness in the cavum, behind the nose and in the throat, dissapears in 1-2 hours (Day 7). 908.HA.3

Burning in the cavum, behind the nose in the afternoon for aprox. 15 minutes (Day 11). 908.HA.3

Dry felling inside nose, like velvet in the evening in bed, urge to clean, but no discharge (Day 4 and 5). 908.HA.7

Blocked nose

Nose like blocked, but can breathe through the nose (Day 4, 200C). 908.HA.4

Blocked left nose morning on waking (Day 5, 200C). 908.HA.4

Blocked nose, dry nasal mucous membranes in the morning for some days. (after 200C). 908.HA.6

Crackles occasionally in the nose (Day 4, 200C). 908.HA.4

Occasional sneezing in the forenoon (Day 2, 200C). 908.HA.4

Flow rhinitis 2 times in the forenoon (Day 4, 200C). 908.HA.4

Nasal root every breath felt. (Day 6, 30C). 908.HA.4

Face

Face feels numb, as if had drunk too much wine. It is un-wielding and unresponsive to facial expression (second hour). 908.HA.9

Left-sided headache, pressing pain on the face up to the upper jaw on waking in the morning, feel the left maxillary sinus in the depth.(Day 3, 30C). 908.HA.4

Sensitive sinus maxilaris more left side, I feel the sinus maxilaris. (Day 6, 30C). 908.HA.4

Sensitive sinus maxilaris, feel the sinus maxilaris (Day 2, 200C). 908.HA.4

Pressure on the face in the region of sinus maxilaris (Day 4, 200C). 908.HA.4

Pulling pain in the face, forehead, around the eyes and in the sinus maxilaris. Daily together with the headache. Pulling from the top of the head over the face. (after Day 10, 200C). 908.HA.4

Headache and facial pain better after getting up, better by movement (swimming) and better after the first cup of coffee at around 9 a.m. (after Day 10, 200C). 908.HA.4

Clenched my jaws very firmly during nap (Day 4). 908.HA.7

Clenched jaws several times (Day 5). 908.HA.7

Cracks in corners of the mouth (Day 12 til 16, 200C). 908.HA.3

Mouth

Small canker sore inside left cheek, lasted only one day (Day 1). 908.HA.1

Tongue feels thick and un-wielding (after 3 hours). 908.HA.9

Mouth is dry with no desire to drink (after 3 hours). 908.HA.9.

My mouth is dry, my throat is dry with an aversion to drink (after 6 hours). 908.HA.9

Taste

Strange taste in my mouth, not clear what kind of taste, just different (Day 1). 908.HA.1

Throat

Post-nasal drip and mild cough with slightly throat tickle. Kept clearing my throat frequently (short time after 30C for one hour) (Day 1). 908.HA.1

Between 4-5 p.m. a burning sensation in the trachea (Day 8). 908.HA.3

Between 4-5 p.m. a burning sensation in the throat (Day 9). 908.HA.3

Dryness of throat on waking (Day 10, 200C). 908.HA.3

Soreness of throat a few hours in the morning and in the evening, agg. empty swallowing, ameliorated while eating (Day 17, 200C). 908.HA.3

Sensation of a dry, swollen throat with pain extending to the ears on empty swallowing; no pain or discomfort on swallowing food (after 3 hours). 908.HA.9

Slight sore throat both sides equally. Swallowing doesn’t seem to influence it. Warm tea makes it better for a while (Day 5, 30C). 908.HA.8

Stomach

Unquenchable thirst for small sips often (icy cold water) with dry mouth not better by drinking (seventh day). 908.HA.9

Stomach gurgling. 908.HA.2

Repeated belching. 908.HA.2

Discreet nausea with constant feeling of hunger, but eating does not improve the nausea. (after 30C). 908.HA.6

Slight nausea morning on waking which persisted for a few hours then went away. Eating did not affect it. (Day 2,3, 6C). 908.HA.8

Slightly nauseous, ate only a small piece of bread for lunch, afterwards I felt a bit stronger and less nauseous (Day 3, 30C). 908.HA.8

I didn't feel like eating, but still had dinner and again felt better afterwards, stronger and not nauseous (Day 3, 30C). 908.HA.8

Nausea in the morning after rising, eating breakfast made it a little better (Day 4, 30C). 908.HA.8

Nausea got worse in the evening, but I ate against my inclination and was surprised to feel much better afterwards (Day 4, 30C). 908.HA.8

Nauseous in the morning a few minutes after rising, better by eating, then returns about 3 hours after the last meal (Day 5, 30C). 908.HA.8

Food desires

Desire for beer (Day 5). 908.HA.3

Av. and agg.

Nausea from smell of a cooked sausages (Day 7). 908.HA.3

Abdomen

The abdominal cramping with diarrhea vanished instantly. (curative). 908.HA.9

Stool and Rectum

Large soft easy bowel movement (usually very constipated) (Day 2,5,6,7,8). 908.HA.1

Watery, painless diarrhoea in the morning. (after 30C). 908.HA.6

Fullness in abdomen from constipation, worse at night (Day 10). 908.HA.3

Stool incomplete (Day 10). 908.HA.3

Constipation with no urging for stool (Day 2, 200C). 908.HA.3

Urinary organs

Urinary frequency in early afternoon. I had to urinate every few minutes for a few hours. No dysuria or abdominal pain. Symptoms eased in a couple of hours (Day 9). 908.HA.1

Woke from sleep around 2:30 a.m. with urinary frequency. Had to urinate every 5 to 15 minutes for a couple of hours. Fell back to sleep at 4 a.m. and urinary frequency was normal. No dysuria or abdominal pain (Day 11). 908.HA.1

Burning sensation at the urethral opening, which persists throughout the day. (One hour after taking the third dose 30C) (Day 4). 908.HA.5

Urine

Urine strong odor and darker than usual (Day 4, Day 5). 908.HA.1

Female

Burning entire perineum including anus and labia when showering and soaping (Day 2). 908.HA.5

Respiratory organs

Respiration

Inclination to breath deep

Compelled to take deep breaths for no reason. Was not short of breath, but found I kept taking very full and deep breaths throughout the day (Day 6). 908.HA.1

Compelled to breath deeply spontaneously in morning when first woke up and throughout the day. I am able to take deeper breaths now than before the remedy. I can expand my chest in all directions, pleasant feeling (Day 8). 908.HA.1

Need to take a deep breath, a couple of deep breaths.908.HA.2

Inclination to breath deeply (immediately after first and second dose) (Day 1 and 2). 908.HA.5

Respiration is noticeably slower; don't seem to need to take a breath; when breathing, it's slow and shallow; utterly calm; pulse 108 (first hour). 908.HA.9

Breathing is a somewhat shallow 10 bpm and feels like not getting enough air (2/10). Want to sit outside in the shade in the wind, which does not help this air hunger. Taking a deep breath does not help (second day). 908.HA.9

Difficult to breathe deeply, irritation of cough after deep inhalation. (Healing symptom). 908.HA.5

Difficult respiration on ascending. (Healing symptom, permanent better). 908.HA.5

Difficult respiration on exertion. (Healing symptom, permannt better). 908.HA.5

Cough

Short,dry cough as if chocked myself at night between 12 and 3 a.m. during sleep (Day 3, 5). 908.HA.3

Dry cough at 3.30 p.m. (Day 17). 908.HA.3

Dry cough in the evening from tickling in the throat (Day 7). 908.HA.3

Episodes of dry cough from tickling in the throat (Day 14, 15). 908.HA.3

Dry cough from dryness in the throat around 5 p.m. (Day 2, 200C). 908.HA.3

Dry cough from dryness and tickling in the throat from time to time (Day 12 til 16, 200C). 908.HA.3

Dry, barking coughs several times a day suddenly without cause since February before the proving, so for 5 months. This cough disappeared during the proving completly. A corona test in May 2020 was negative. 908.HA.4

Chest

Intense cramping pain in heart for a few minutes while lying on right side and my thoughts were to me deceased aunt, disappeared after rising from bed (Day 5). 908.HA.3

Heart cramp while sitting in a chair at 5 p.m. for two minutes, no particular thoughts (Day 6). 908.HA.3

Light pain in heart for 1 minute (Day 17). 908.HA.3

Constriction left side of the chest for a few minutes after 5 minutes walking (Day 18). 908.HA.3

Intense, burning-sore pain in right superior-extern part of mamma since waking. Very much aggravated by touch and jar (running, descending stairs rapidly). Lasted whole day and next night, could not sleep on the abdomen due to the pain, lying on the painful side pain was tolerable, but when turning on the left side during sleep the pain woke me, being very intense. No lumps, no discoloration. (14 years ago mastitis in right mamma, but that time much lesser pain) (Day 12, 13). 908.HA.3

Pain in the right mamma comes back and it stays for 3 hours (Day 19). 908.HA.3

During the night again pain in the right mamma (Day 23). 908.HA.3

Pain in the left mamma aggravated by accidental touch (Day 10, 200C). 908.HA.3

Pain in the left mamma 3/10, aggravated descending stairs 4/10 and touch 5/10 (Day 11, 200C). 908.HA.3

Weakness in the chest, around the heart area (Day 3, 30C). 908.HA.8

Weakness in the heart area (Day 5, 30C). 908.HA.8

Back

Cervical spine

Pain in cervical region in the morning, aggravates a lot during washing dishes, when I have the sensation my neck will break, it is not able to sustain my head, vanished after walking in the open air (lasted for 2 hours) (Day 7, 8). 908.HA.3

Stiffness of neck 15 min after waking up, stiffness extending down the thoracic spine, turning of the head, bending forward and stretching back, moving to the to both sides painful and stiff (Day 4, 200C). 908.HA.4

Whole day limited head mobility, stiffness shoulder and neck, when turning the head, the shoulders must also turn (Day 4, 200C). 908.HA.4

Mobility of the neck and thoracic spine limited due to stiffness (Day 5, 200C). 908.HA.4

Painful muscle gellings in shoulder area, turning the head restricted. Improved by massages and pressure on the muscle gellings (after Day 10, 200C). 908.HA.4

Strong shoulder and neck stiffness and occipital headache, extending to the left side of the head. Lying on the back ameliorate, lateral position rather worse, not quite sure. (after 30C). 908.HA.6

Dorsal spine

In the evening I feel a gripping pain in front of the left scapula, that lasts 1 minute (Day 9, 200C). 908.HA.3

Lumbar spine

Cramping pain lower lumbar region (L 4-5) with contracted muscles on waking in the morning, worse when walking on hard ground (jar) and after getting up from sitting. Probably after lifting too hard and then cold bathing in the river (on 5th day after the first dose, lasting for 3-4 days) (Day 5). 908.HA.5

Pressing, drawing pain in the left sacro-iliac region radiating to the left hip when waking up in the morning. Worse after sleep and the first movements in bed are very painful, even on rising from bed it is painful, but less than the first motion, after a few minutes of walking it is much better, it gets better the more I walk, also hot showers improves. Standing worsens. With long standing massive increase of pain in the thoraco-lumbar area as if the back would "break apart". Sitting, lying, resting and local warmth as well as massaging improves. Stretching and treadmill training improves the pain left sacro-iliac region. (Chronic symptoms of the prover, which appeared after the 200C). 908.HA.6

Extremities

Easily exausted feeling in the legs during riding with bike, heaviness in legs, it was tremendously exhausting to keep pedalling, I would have loved to stop, get off the bike and lie down, feeling overstrained (Day 2). 908.HA.7

Tension and stiffness of the thigh and calf muscles (30 min after C 30) (Day 1). 908.HA.6

Tension and stiffness of the thigh and calf muscles today also glutaeal (Day 2). 908.HA.6

Stiffness of the thigh muscles like sticks (immediately after 30C). 908.HA.6

Sltiffness in the thigh muscles (after 200C). 908.HA.6

Left thigh and groin pain. Aching, like a tooth ache, dull and throbbing, comes in waves, crescendo-descresendo type pain. Better by warm application. Worse sitting still, better lying. Better with movement (Had done the day before more sqating exercises than usual) (Day 7). 908.HA.1

Slight drawing pain in extremities, not very painful, just noticeable (Day 3, 30C). 908.HA.8

Slight drawing in hands and feet and other joints (Day 4, 30C). 908.HA.8

Dropped two glasses of water (I never do that) (Day 2). 908.HA.1

Spilled another full glass of water (Day 4). 908.HA.1

My feet and lower legs (below the knee) were so cold on waking, I expected them to be white. They were not discolored in any way. The mattress upon which my feet and legs lay, and the blanket that lay upon them, were noticeably cold (after 6 hours). 908.HA.9

Crack in left heel, painful while walking (old symptom, last time many years ago) (Day 2,3,4). 908.HA.3

Cramp in left calf morning at 5:30 a.m., woke me up (Day 5). 908.HA.3

During daily morning swim a pulling into the right upper arm (after Day 10, 200C). 908.HA.4

Pain in the left wrist In the afternoon while using the hand, dissapears in the evening (Day 22). 908.HA.3

Perspiration

Sweat all over the body during falling asleep in the evening (26 degrees Celsius) (Day 2, 30C). 908.HA.4

Skin

Insect stings three times during the proving. Stings very itchy, if I start scratching can not stop scratching. Swelling is red, hot and large, one is 10 cm in diameter (Prover is usually more sensitive than others for insect bites, but during the proving it was extreme). 908.HA.3

Sleep

Sleepiness

Sleepiness at 1 p.m. when sitting (Day 2). 908.HA.3

Extreme sleepiness at 1 p.m., could bearly keep my eyes open, lasted until 4-5 p.m (Day 14). 908.HA.3

Sleepiness at noon (Day 20, 30C). 908.HA.3

Sleepiness at noon (Day 3, 200C). 908.HA.3

Sleepiness from 1-3 p.m. (Day 5, 200C). 908.HA.3

Sleepiness in the afternoon, take a nap from 4-6 p.m. (Day 9, 200C). 908.HA.3

Sleepiness from 10 a.m., at 12 p.m. I try to sleep, but I cannot fall asleep (Day 10, 200C). 908.HA.3

Sleepiness during day (Day 16). 908.HA.3

Sleepiness evening, after dinner around 9:30 a.m. immediately back to bed and slept (Day 5 30C). 908.HA.4

From 9 p.m. on dog-tired. Legs become restless, must go to bed early (Day 4, 200C). 908.HA.4

Tired and sleepy late afternoon, slept 1.30h deeply and firmly (very untypical for me) (Day 5, 30C). 908.HA.4

Sleepiness, fell asleep while riding as a passenger in the car for a short trip (unusal for me) (Day 2, 6C). 908.HA.8

Yawning with lachrymation. 908.HA.2

Carboneum oxygenisatum

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Prolonged sleep (9,5 hours) (Day 15). 908.HA.3

Unusual long sleep until 8.30 am (Day 8, 30C). 908.HA.4

Sleep very well and deep, difficult to wake up in the morning (Day 3). 908.HA.7

Morning in bed fall asleep again and again (Day 4). 908.HA.7

Awake several times first around 4 a.m. til 6.30 a.m., then got up (Day 5 30C). 908.HA.4

Awake around 4 a.m. in the night (Day 8, 30C). 908.HA.4

Awakens at 3.40 a.m. and around 6 a.m. (Day 2, 200C). 908.HA.4

Awakening at 4 a.m., fell asleep after short time (Day 4, 200C). 908.HA.4

Waking up a few times at night (I usually sleep well), but went back to sleep right away (Day 3, 30C). 908.HA.8

Restless sleep (Day 1,2). 908.HA.3 Habich 2020

9- **A case of COVID-19. Presenting complaints:** A 65 years old patient presented to me in the first week of August 2020 with complains of heat in eyes, sore throat, mild coryza, lethargy and feverish feeling and weakness from exertion, he was accompanied by his wife who suffers from Covid-19 phobia and hysteria. Amongst all his symptom weakness and sore throat was more intense. He always believes in homeopathy and would like to have some pills for the above complains.

What I was thinking?
Since his wife was hysterical and there was strong fear and panic of Covid-19 around him there was big pressure underneath his mind about consulting me in my clinic and taking homeopathy (his brother in law is already admitted in the hospital and is very serious because of respiratory failure due to Covid-19). The patient kept on denying that he does not get influenced by anyone and want to consult me, However, I thought I will wait and watch and see what happens next.

My suggestion.
Don’t do Covid-19 test, I was very sure he has Covid-19 since he was exposed to his brother in law who is now admitted in hospital, ss this will generate more fear in the family.

Rubrics

Carboneum oxygenisatum

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MIND - INACTIVITY

EYE - HEAT in

NOSE - CORYZA

THROAT - PAIN - sore

FEVER - INTERNAL heat

GENERALS - WEAKNESS - exertion - agg.

Remedy: Kali phosphoricum 200C, 3 doses for the next 24 hours.

Next day: Much better, less weakness. Better energy**.** But no taste**.** Smell was normal**.** Sore throat was there but better**.** Eyes heat better but there**.** Coryza absent. Mild coughing.

New rubrics

MIND - INACTIVITY

EYE - HEAT in

MOUTH - TASTE - wanting, loss of taste

THROAT - PAIN - sore

COUGH - DRY

FEVER - INTERNAL heat

GENERALS - WEAKNESS - exertion - agg.

Magnesia muriaticum 200C, tid for two days.

After 48 hours: Cough better but there no modalities**.** Nausea-constant**.** Taste getting better but there**.** Eyes watering**.** Sneezing. Feverish feeling. Energy getting better.

New rubrics

EYE - HEAT in

EYE - LACHRYMATION

MOUTH - TASTE - wanting, loss of taste

THROAT - PAIN - sore

STOMACH - NAUSEA - constant

COUGH - DRY

FEVER - INTERNAL heat

GENERALS - WEAKNESS - exertion - agg.

Phosphorus 200C, tid for three days.

Secret

Behind my back the patient is forced to go for blood test. D-dimmer 8.8 (N<0.5). Lymphopenia. Leucopenia. X-ray chest shows 40% shadows of Covid-19 infiltration. Also, patient never took Phosphorous but lied to me that he is on my medication. Actually, is called another medical doctor and started allopathy. Started favipiravir 1000 mg twice a day on 1 day, and 400 mg favipiravir 2 times a day for 4 days, hydroxychloroquine 400 mg, dexamethasone 0.75 mg daily. He was too shy to tell me so I lost contact with the patient.

After 5 days of starting allopathy, his health detoriated and was shifted to hospital and once again I was consulted to treat him all over again.

What happened? His chest was fully congested. X-ray showed total (around 80%) fluffy shadows. Ct scan confirmed the same pathology: Covid-19 pneumonia.

Excessive anxiety; expression anxious.

Dull with impaired respiration; on oxygen 5 l/min to maintain 94% saturation if quantitiy of oxygen is reduced then saturation falls. Senses dull. Staring look. Face pale. Tongue red. Respiration slow. *Saturation falls by slightest exertion*. Short spasmodic cough. Precordial anxiety. Faint feeling. Chilly patient. Pulse quick and soft 97/min. Bp 176/95 mm. Trembling of the body. Temporal headache. Tightness of jaw. Respiratory failure. Constricting pain in chest. Generalized soreness. Extremities cold. Cuprum metallicum 30 every 2 hours for 12 hours.

Reason for Cuprum met

1. MIND - DULLNESS
2. HEAD - PAIN - Temples
3. EYE - STARING
4. FACE - CLENCHED jaw

5. FACE - DISCOLORATION - pale
6. FACE - EXPRESSION - anxious
7. MOUTH - DISCOLORATION - Tongue - red
8. RESPIRATION - FAILURE
9. COUGH - SHORT
10. COUGH - SPASMODIC
11. CHEST -ANXIETY in
12. CHEST - CONSTRICTION
13. CHEST - INFLAMMATION - Lungs
14. EXTREMITIES - COLDNESS
15. GENERALS - COVID19
16. GENERALS - FAINTNESS
17. GENERALS - HEAT - lack of vital heat
18. GENERALS - HYPERTENSION
19. GENERALS - PAIN - sore
20. GENERALS - PULSE - frequent
21. GENERALS - PULSE - slow
22. GENERALS - PULSE -soft
23. GENERALS - TREMBLING - Externally

Reaction: No improvement in any symptom. Saturation still poor. General condition same. Advice – Mechanical ventilation which patient relative refused.

Remedy: Hydrocyanicum acidum 200C every two hours for 48 hours.

Reason for Hydrocyanicum acidum

1. MIND - DULLNESS
2. HEAD - PAIN - Temples
3. EYE - STARING
4. FACE - CLENCHED jaw
5. FACE – DISCOLORATION - Pale
6. RESPIRATION - FAILURE
7. COUGH - SHORT
8. COUGH - SPASMODIC
9. CHEST - ANXIETY in
10. CHEST - CONSTRICTION
11. EXTREMITIES - COLDNESS
12. GENERALS - FAINTNESS
13. GENERALS
14. HEAT - lack of vital heat

15. GENERALS
16. PULSE - frequent
17. GENERALS - PULSE - slow
18. GENERALS - PULSE - soft
19. GENERALS - TREMBLING - Externally

Reaction: More breathless within 3 hours of Hydrocyanicum acidum, aggravation? Saturation dropped to 85%. Relatives and patient worried. Carboneum oxygenisatum 200C every half an hour for 24 hours.

Reason for Carboneum oxygenisatum

1. MIND - DULLNESS
2. HEAD
3. PAIN - Temples
4. FACE - CLENCHED jaw
5. FACE - DISCOLORATION - pale
6. FACE - EXPRESSION - anxious
7. MOUTH - DISCOLORATION - Tongue
8. RESPIRATION - FAILURE
9. COUGH - SHORT
10. COUGH - SPASMODIC
11. CHEST - ANXIETY in
12. CHEST - CONSTRICTION
13. CHEST - INFLAMMATION - Lungs
14. EXTREMITIES - COLDNESS
15. GENERALS - COVID19
16. GENERALS - FAINTNESS
17. GENERALS - HEAT - lack of vital heat
18. GENERALS - PAIN - sore
19. GENERALS – PULSE - frequent
20. GENERALS - PULSE - soft
21. GENERALS - PULSE -slow
22. GENERALS - TREMBLING - Externally

Reaction: Within 4 hours saturation improved. Doctor delayed decision of ventilator. Coughing reduced. Breathlessness reduced. Continued Carboneum oxygenisatum 200C for another 24 hours.

Reaction: Much better. Saturation improved. Breathlessness improved. Started tapering steroids and HCQS. Carboneum oxygenisatum 200C, 8 hourly.

Reaction: CT scan reduction by 40% of fluffy shadows. Improvement persists. Pneumonia getting resolved. Carboneum oxygenisatum 200 to be continued for more 24 hours.

Reaction: Oxygen now 2 l/min. Saturation maintained at 97%. Breathlessness reduced. Cough reduced. Body ache better. Energy better. Carboneum oxygenisatum 1M, 3 times a day for 7 days.

Reaction: Repeat CT scan shows only 20% fluffy shadows. Overall better

General observation of Carboneum oxygenisatum in Covid-19 as well as in other patients:

Coma.

Convulsions with coma.

Excessive secretion from with coma.

Paralysis with coma.

Decorticate rigidity with damage to the corticospinal tract with coma with convulsion.

Cardiac irregularities with coma.

Coma with moaning.

Pupils insensitive with coma.

Eyes wild staring and protruding with coma.

Left hemiplegia with coma.

Fingers clenched.

Respiratory symptoms.

Weak respiration—respiratory failure.

Respiratory failure with coma, pneumonia with coma.

Increased respiratory rate.

Reduced arterial carbon dioxide levels.

Rattling of mucus in air-passages.

Bloody mucus is raised from the suction.

Respiration audible, almost rattling, slow, stertorous.

Respiration rattling.

Stertorous breathing.

Respiration slow, frequently interrupted by yawning and sighing.

Respiration difficult and interrupted.

Respiration very laboured.

Somewhat impeded respiration.

Sense of suffocation.

The action of the heart and of the lungs gradually decreased.

Feeble action of the heart.

Action of the heart slow and weak.

Beating of the heart alternately increases and diminishes.

The beating of the heart is at first increased, but afterwards it becomes slower.

Pulse small, rapid.

Pulse rapid, very small.

Pulse slow and full.

Small, slow pulse

Brain

Brain herniation.

Hydrocephalus.

Coldness of breath with face cold to touch, coldness of hand and nose.

Cerebral oedema.

Intoxicated confused stupid.

Answers with difficulty.

Cerebral haemorrhage or cerebrovascular accident or apoplexy with violent headache with tightness and constriction of frontal and temporal area.

Violent pulsation of temporal artery.

Eyes in coma.

Eyes half open with coma.

Pupils dilated with coma.

Pupils dilated and not reacting with coma.

Involuntary urine ad stool with coma.

Scanty urine and sugar in urine.

Retinal haemorrhages with apoplexy or coma.

Face

Face pale, red, cyanotic, and puffy.

Distorted face.

Trismus with coma.

Lips bluish.

Mouth

Tongue rosy red.

Froth from the mouth.

Paralysis of all sphincters.

Aphasia with apoplexy.

Bed sores esp. on heel turning towards early gangrene.

Sensibility of sight, hearing, smell, and taste also greatly lessened, Blunted sensibility of the whole skin.

Anaesthesia of the skin. Master 2020

10- **A suspected Covid-19 from Malta.** October 18, 2020, J.B. 52 years old developed symptoms of influenza, headache, runny nose and sneezing. I gave her Bryonia to take four times daily. The next day she felt much better. She thought it was all gone, but although the symptoms of influenza were gone she then developed pains in her rib cage and had no energy at all.

She could not cook or clean and even drying herself after a shower was too strenuous for her. She thought she might have the COVID-19 virus and was given an appointment for a swab test for the following week. She had the test on Friday, October 30, but the result was negative.

Meanwhile she was still feeling quite weak so we persisted with Bryonia but on Sunday November 1, she suddenly developed extreme shortness of breath and could not talk or take 3 steps without being out of breath. I immediately prescribed Carboneum oxygenisatum 200C to take 3 times in succession 10 minutes apart and then let me know. She felt better after the three doses so I told her to take the remedy again before bed and again in the morning on waking. She phoned me the next day to say that she was very much better and within a couple of days she had completely recovered. We both suspect that she may have had a false negative result. Borg 2020

11- **Another suspected Covid-19 from Malta.** C.S., my mother who is 100 year old, developed an unexplained fever on October 18. There were no symptoms of cough, cold or sore throat. She started treatment with Gelsemium 200C, as she was very sleepy and flushed. Gelsemium helped slightly but her fever would increase around 2 or 3 p.m., so I switched to Belladonna 200C which dealt with the fever. She then developed aches all over her body and any motion hurt so I gave her Bryonia 200C. She seemed to have recovered completely for a few days after that but then on the afternoon of October 23 she suddenly developed extreme shortness of breath. Because of her age we thought this was the end. We felt helpless and it was very difficult to watch her struggling to breathe. However, I decided to try Carboneum oxygenisatum and gave her the 200C, three doses ten minutes apart. Within about 15 to 20 minutes I noticed a slight change. She was no longer struggling so heavily to breathe and she also accepted to try eating something.

I left her with instructions to her carer to I give her another dose before sleep and again the following morning. The next day I was delighted to hear that she had slept all night. She described it *as a miracle* and could hardly believe it herself! She continues well to date (November 11, 2020). It is interesting to note that this patient had taken Bryonia 200D on a daily basis when COVID broke out in the spring in Malta until the beginning of August when she developed a goitre. She had a thy-

roidectomy on September 16. She only resumed taking Bryonia 200D only some days after she had fallen sick on October 18. Borg 2020

12- **Covid-19 patients and Carboneum oxygenisatum.** Since my last communication, more than 25 patients with Covid-19 with mild to moderately severe complaints have been treated with homeopathic remedies, first mainly with Bryonia, later on more and more with Carboneum oxygenisatum.

Treatment as dissimilar diseases: first the acute complaint, later on the chronic appropriate remedy for chronic complaints (“constitutional remedy”), possibly alternating.

My approach has been at first to prescribe the indicated acute remedy, which was mainly Bryonia, and sometimes Arsenicum album or Gelsemium

Despite having clear pictures of Arsenicum album, Bryonia or Gelsemium and these remedies were prescribed, but most often Bryonia worked.

Later on when the picture wasn't very clear, I prescribed Carboneum oxygenisatum, especially with the following symptoms being present: **extreme exhaustion** (lying down all day), **resignation** (while Arsenicum album has fear, Bryonia is irritable and Gelsemium worries) and **great difficulty breathing**; this approach worked out well.

Then, Carboneum oxygenisatum became the remedy of choice, unless there were additional features from practice: **Very dull, hard to think, confused**, sometimes so tired that a good anamneses was impossible, **difficulty breathing deeply** or through, **being very cold**, **very painful and tight muscles**, **worse motion** and sometimes **with restlessness**.

The picture became clearer with more specific symptoms, that is the **resignation with serious complaints** and with very low oxygen saturation index (sub-acute low 80s with exertion), **dullness during the fever**, **enormous weakness, and sudden weakness from the sightless exertion**, **loss of smell and taste**, **shortness of breath**, **cough worse breathing** and **talking**, **pain in lungs**, **not being able to breathe properly**, **severe headaches**, **especially occipital**, **worse bending forward** and **bending the head backward**.

Remarkable:

In acute covid-19, the remedy needs to be given relatively often, in severe patients two times a day three times an hour. A clear improvement is often visible within

(half) a day. When improvement sets the remedy is given twice a day for two doses one hour apart, then reduce further.

Saw regularly a relapse for instance after 1 or 2 weeks. This appears to fit with corona. Now I give the remedy for a longer period of time, that is if the patient feels well, a dose each day for three days each week for three weeks in a row.

With long covid (now five people being treated) I immediately start with Carbn-o 200, 3x/24 hours, and then wait and repeat as needed.

All five patients seem to be improving; many said within a few hours my head is more clear and start to trecover. Later on here too, if necessary, I will switch to their constitutional remedy

Here are some typical cases: October 21, 2020. Man in his sixties, tired, muscle aches, headache, poor sleep. He wants to be alone, moving is too tiring and hurts. Lies down, too tired to walk up the stairs. He's a little short of breath, chest pressing pains.cough isn't a big deal. He wanted to call moe early but couldn't. Very heavy thighs. Hot and clammy, fever? He has already taken Bryonia 200C himself, spread three times over the day. Bryonia 4x q30min, possibly tonight, call tomorrow

22-10-20: A little better, slept better, less headache, tired. Bryonia 200C tid.

25-10-20: Significantly worse: lung problems, breath is stuck, chest pressure worse. More muscle pain, some sore throat. Taste and smell are now less. Carboneum oxygenisatum 200C tid.

26-10-20: Feels very sick, too lame to call. Chest pain was very bad, enormous pressure!! As if a horse was sitting on it. Saturation 97%! Pressing pain better after remedy. A lot of muscle pain. Carboneum oxygenisatum 200C 3xq30min hour, if better tid.

27-10-20: Yesterday slowly better, now a lot of muscle pain, also when lying down. Very tired. Not short of breath or cold, chest pain clearly better. Throat fungal infection “by paroxetine”, white tongue, known symptom. Very very tired, like after running a marathon. Carboneum oxygenisatum 200C qid.

12-11-20: Contact with my colleague twice, gradually much better. Feeling better, more enrgy, can handle more and more things.

It was very bad, huge chest pressure. A lot of muscle pain, just kept turning in bed. Wanted to die! Reliving forgotten pneumonia when he was 7 years old. Was very very lonely then. Transformation, part of his depression is over now.

Afterwards he told, when I asked about it:

“Little Karel, aged 7, felt lonely and alone and abandoned. He called out for mommy inside, he couldn't actually call his mommy, he couldn't or could barely make contact with the world and with others, he remained deeply hidden within himself, which was even a bit nurturing with a deep longing not to go any further in this world. His lungs, inflamed with pneumonia, ached in a burning, screaming manner.

“As a 65-year-old man with COVID-19 (October 2020) I thought that with the same feelings as that 7-year-old boy I would rather be a refugee in a dangerous war zone with parents or people who are there for me anyway, than with parents who were nowhere to be seen.”

To me as a doctor this experience mirrors exactly the experience of people in the ICU! Could this experience of loneliness be an essential part of this epidemic? De Sonnaville

13- **Another COVID-19 case.** 23-12-2020: Woman, late fifties. So tired! Wants to lie down all day. Feels very very cold. Short of breath worse exertion. Back pain, just below scapulae, pressure on chest. Dry cough, tickle in throat, worse temperature changes. Pain throat worse talking, lying and on lying down, getting warm in bed. Dry mouth, thirst, des and better cold water. Muscle pain, a bit restless. Very nauseous, aversion to food. Taste and smell okay. Dull, tired, don't want to talk any more. Bryonia 200 3doses one hour apart.

24-12-20: Less stuffy, less back pain. Now nose obstructed and running. Temp was 36.1, now 37.2. Generally a bit better. After 2nd dose: bubbling belly, suddenly dark diarrhea, afterwards belly calmer. So: a few days Bryonia 200C three times per hour, then reduce.

28-12-20: Has generally improved, energy is also a bit better, no chest pain. But: smell and taste are gone. Coughing and shortness of breath with little movement or effort. Dizzy when walking, e.g., to the toilet. Carboneum oxygenisatum 200C three times per hour.

15-1-21: First better then varying. Appears better after the remedy: waited and better gradually but not okay despite taking Carboneum oxygenisatum 200C.

At physio for recovery: oxygen saturation with little effort from 93% to 84%. Does not breathe freely. Slow, as if she forgets what she thinks. Tired when rising in the morning, worse talking, when walking dizzy, unsteady. Memory is bad, even forgets

what she did. No smell or taste. Skin part of fingers and toes red. Carboneum oxygenisatum 200C, 2nd glass, as needed.

18-1-20: The next day her oxygen saturation dips down to 83% with little effort, the day after 96% with the same effort. During exertion pressure on her chest. Can breathe more deeply. More vital. No more feeling worse after eating. Further treated, later also with Carboneum oxygenisatum 1M. Steadily better, oxygen saturation stayed above 95%. But she is still tired. Turns out to be an history of fibromyalgia, later on changed remedy to Pulsatilla and she gradually improve. De Sonnaville 2021

14- **Long COVID-19 case**. July 27, 2021: Woman in her mid-40s got COVID-19. March 2020. She is overweight. She was very sick! Her acute symptoms were great shortness of breath (respiratory rate 26/min), coughing enormously, hoarse; couldn't do anything, dead tired; very chilly. Had a long time fever, very variable fever.

She has severe muscle pain, very nauseous, intestinal distress. Smell and taste diminished. Emotionally very unstable. Tired since then, muscle aches, shortness of breath, difficulty breathing. Can hardly do anything, e.g., after going up and down stairs she has to rest for hours. Not able to do any housekeeping.

All muscles are tight. Most muscle pain in calves, kind of continuous cramp. (Allen: tonic cramps). Gained 15 kg this year. Swelling of feet. Difficulty breathing, constant chest pressure. Hoarse, dry airways. Carboneum oxygenisatum 200 tid for one day.

August 13, 2021: After first dose: head clearer *within a few hours*. After 2 days: can breathe deeply again (has to get used to it again!), muscles much smoother and less painful, lots of energy. Now: everything is lighter and happier, feels much better. Less stiff, little pain, more energy, less cold, blood flows better, hoarseness has improved, abdomen is calm, stools twice instead of six time a day. After one week: “Energy went from 3.5 to 6.5!” Last week was busy, now slight relapse. She got her menses. Carboneum oxygenisatum 200C tid for one day.

August 30, 2021: She forgot to call: “Again one dose 9 days ago.” This is incorrect, as it turns out she took three doses of the remedy on August 13. She is not thinking optimally yet.

After half a day a lot better, energy and breathing deeper. Can climb stairs without having to rest (otherwise rest of day tired). Did a lot more last week (busy compa-

ny), and cleaned the house (that was not possible for so long). Then tired and hoarse again, a lot of muscle pain. After some rest she recovered (!).

Belly changes, but is gluten intolerant, better than before COVID-19? Calves much better, a bit stiff, no more pain, climbing stairs okay. Better than after the first dose, and mental brighter too. Carboneum oxygenisatum 200 q2wk.

Conclusions:

1: In my experience over the past year, Carboneum oxygenisatum seems to be the drug most suited to the genius epidemicus. It is now the number one remedy for prevention for me.

2: Carboneum oxygenisatum works well, both in the acute form of COVID-19 and in long COVID-19.

3: In the acute phase it has to be *given quite often*. In severe patients (very short of breath, low oxygen saturation index) I start with twice a day three times an hour.

4: I continue to give Carboneum oxygenisatum (in decreasing frequency) even if the patient has largely recovered. And give it the three weeks after that (e.g., every week three days once a day).

5: I give less frequently with long COVID-19, for example 3x/24 hours.

6: Treating this disease as any “dissimilar disease”: first the acute illness, then the chronic predisposition. Sometimes the chronic remedy follows after the Carboneum oxygenisatum, and the appropriate remedies sometimes have to be alternated with Carboneum oxygenisatum.

7: After going through COVID-19 and after the vaccination, a number of people have a clear deterioration of their chronic disease ("constitution"). Long COVID-19 is often the result of this.

More specific symptoms in several COVID-19 patients:

Complaints improved after Carboneum oxygenisatum: in regular print: in 1 patient, in bold: in 2 or more patients.

Very dull, hard to think, confused.

Sometimes so tired that a good history was impossible.

Resignation with serious complaints and with very low saturation.

The enormous weakness, and sudden weakness after little effort.

Sometimes so tired it's hard to get symptoms!

Want to lie down and nothing else.

Day seems very long.

Anxious, afraid of dying, worried, tearful.

Dull, can't think clearly.

Poor concentration.

The resignation with serious complaints and with very low saturation.

The enormous weakness, and sudden weakness after little effort, worse talking.

Slow and slow, in moving and thinking.

Desires to rest, being alone, leave me alone, aversion sound, music.

Gloomy, quiet.

Restless.

Despair recovery.

Lying down, don't have the energy to do something, apathetic.

Confused, lost.

It's hard to concentrate.

Very forgetful.

Vertigo worse after slight exertion, better eyes closed.

Staggering when walking.

Eyes painful better close.

Worse touch eyes, light.

Headache, band, hairache touch.

Headache worse noise, **better pressure, coolness, eyes closed, moving.**

Headache worse mental effort, light, cough, noise.

Better pressure, heat, massage, **lying.**

Very heavy head worse after exertion.

Pain head and neck, stiff worse awakening, better hot shower.

Severe headache, especially occipital, worse bending, worse bending head back.

Pulsating.

Stitching.

Vertex, forehead, occiput.

Worse slightest movement, lifting head impossible.

Worse motion.

Ear, stopped.

Nose obstructed.

Things smell bad.

Loss of smell and taste.

Ashen appearance.

Dry mouth.

Salivation, then nausea and vomiting.

Hoarseness.

Dry cough, worse move.

Dry throat, scraping.

Pain throat: worse swallowing, at night, eat, **talking, better drink,** cool drink.

Pain extending to ears.

Desires sour things.

Heartburn, worse after eating or drinking.

Rumbling abdomen, and then dark diarrhea.

Pain abdomen from coughing.

Abdominal pain, nausea.

Abdominal pain before stool, with rumbling in abdomen, then diarrhea, better after stool.

Nauseous upon waking.

Abdominal pain, followed by diarrhea.

Cramping pain, better pull up legs, better pressure on abdomen.

Chest pain.

Pain in lungs, not being able to breathe properly.

Muscle pain heart region, radiating left shoulder.

Shortness of breath, cough, worse breathing, talking, lying down.

Need to take a deep breath.

Band around chest.

Cough is not too bad, little mucus.

Worse talking.

Out of breath very quickly.

Difficulty breathing deeply.

Shortness of breath from little exertion, with high heart rate.

Difficult breathing better fresh air, fanned.

Worse bending forward, lying on side.

Worse moisture and warm.

Worse after dinner.

Worse pressure of clothing (bra).

Respiration difficult worse cold air, talking, dress up.

Respiration difficult with pain back, dorsal.

Cough worse exertion, talking.

Cough worse geting out of bed, turning in bed, moving, taking a deep breath, inhaling.

Expectoration sweet.

Tickling cough better after expectoration.

Worse tension, excitement.

Worse lying flat.

Numbness hands.

Muscle pain shoulder, worse move.

Muscle pain from lying in bed.

Weak legs, muscle pain legs.

Muscle pain in thighs, better moving for a while, want to move, worse lying quietly.

Muscle pains better warmth.

Muscle twitching in thighs.

Lower limbs tingle, better when lying down.

Muscle pain calves, cramping, tight.

Muscle pain, better lying still.

Coldness feet.

Swelling feet.

Chilly with hot red head.

Fever up and down, everything changes.

High fever (40) chattering teeth, shaking, nausea.

Fever with sweating.

Dry and warm with a high fever.

Shivery in the afternoon.

Diarrhea first, then fever.

Cold inside.

Sick and nauseous.

Fever and poor sleep.

Very chilly.

Very painful and tight muscles, worse moving, sometimes restless.

Alternating cold and warm.

Very very tired, little fever.

Already very tired when waking up, needs to get going.

Very tired when waking up in the morning.

Sleeps a lot.

Suddenly tired, suddenly exhausted.

Tired of least effort, worse talking.

Has to rest after, for example: one flight of stairs, has to lie down for more than an hour and can't do anything.

"Lifeless, exhausted."

Body feels heavy.

Fever and nausea, headache, no appetite.

Change of symptoms, varying course, intermittent.

Good day, suddenly bad day, or suddenly exhausted.

Slow recovery.

Also after recovery: suddenly tired again, or suddenly sharp worse after exertion.

Sick with fever, backache, abdominal pain and pain when urinating, frequent urging to urinate but no cystitis in pregnant woman.

Worse first movement, better continued movement. De Sonnaville 2021

15- A Covid-19 Case with Severe Acute Respiratory Syndrome. August 31, 2021: KD is a 72-year old insurance broker who fell sick with Covid-19 eleven days ago on August 20. He is in a pretty bad shape. DJ, his business partner, found him already in a bad state four days ago and she decided to contact me to see if I could help him.

KD had a carpenter around the office for the last month, as he was rebuilding an old garage. On Wednesday August 18, the girlfriend of this carpenter who had cold like symptoms came into the office and talked to KD for just one minute. No one had mask.

On Friday evening August 20, KD felt sick with a febrile condition and DJ, his business partner, fell sick the next morning with similar symptoms. DJ never had any encounter with the carpenter or his girlfriend.

On Saturday KD phoned the carpenter to tell his girlfriend that whatever she has is very contagious and she shouldn't mix with people. He learned at that moment that both the carpenter and his girlfriend had been diagnosed earlier that day with covid-19. Eventually the carpenter was hospitalized for ten days heavily mdeciated and on C-PAP machine.

A week later both KD and DJ became profoundly ill, but KD was in a worse state. DJ's husband and two children fell also sick subsequently.

KD was bedridden and had stopped eating after he began experiencing severe respiratory distress and became mentally very confused, when DJ visited him four days ago. He says he doesn't know how to sit up in bed or when he is already sitting up how to lie down.

He has had diabetes for 6-7 years, which he tried to control with diet and exercise but his fasting blood glucose (BG) still remained between 120-130 mg/dL (N: 70-100; pre-diabetes: 100-125; diabetes: ≥126).

He is 6'1" (1.85 m) and used to weigh 195lbs (88.4kg), but he has certainly loss 15lbs (7 kg) since the fever.

He has had a persistent fever since Friday evening August 20, which is associated with a continually recurring violent cough with rare brownish expectorations. His face turns white and his fingers blue when he coughs.

He is sleeping most of the time and is frequently waken up by this very violent cough, which I heard in the background when I was taking his case and it sounded like rolling thunder, and what DJ described, “Like lion roaring.” Most of the time he is lying with eyes closed with carphologia (picking at the bed sheets).

Heat radiates from his face, ears, neck, occiput and back regardless of his position. He tends to put his hand on his forehead to cool it down.

He objectively shivers for long periods of time after any exertion, such as just lying down from sitting up. He got up this morning at 7.30 AM, walked about 20 feet and back from the washroom and shivered afterwards in bed for about 30 minutes, which was accompanied by violent coughing and severe shortness of breath (SOB). At this time he wanted extra covers.

His hands begin to shake when he sits up in bed and diminish as soon as he lies down again.

With any exertion, he becomes profoundly lethargic and weak. In the last two days he has showed signs of arrested breathing, which especially happens when he lies on his back.

He moans after falling asleep with shivering. He has been extremely confused for the last four days about what is going on. He often says, “I don’t know what is happening. My body is off. I need to go back to my schedule. My rhythms are off and my natural function is put to the grind.”

When awake he complaints that his energy is floundering. His pulse is around 87 (N: 60-64), but is now at 104, RR 39 (not 15) and O2 index was 90% at rest, but it drops in the 80s% after a period of shivering after exertion.

His breath is fast and superficial. He has paradoxical breathing (abdomen going in on inhalation) with his mouth open when he lying on his back, which is a sign of respiratory failure.

He complaints that he can’t take a deep breath. The cough is worse when he attempts to take a deep breath. Before this spasmodic cough he would thrash around in bed and his legs would go off the bed, but since he has been so depleted of energy he stopped trashing around. He shivers after a coughing fit. He quickly lost his senses of taste and smell after the onset of fever.

Both KD and DJ experienced soreness of the vertex of the head that spreads to the rest of the body. Cold applications to the forehead make him feel better to him.

KD has been very irritable with his business partner, which is very unusual for him. He has to have control of the situation and she has to follow his “schedule.” By nature he is a dominant individual. He was extremely critical of her today and was upset with her when she said she was calling a doctor.

He is extremely weak, as she has to help him even just to sit up. His energy is 0.1/10. He often says, “My hot head is problematic.”

When he sits up in bed he bends his head forward, stretching his neck one way and then the other way. He says that he has pain in his neck down to his scapula over the part is covered by the trapezius muscles. “It is very tight.” It comes and goes and is better with warm applications (2).

About the SOB, he says, “There is not enough air. I feel I have been through the ringer like never before.”

He occasionally drinks water, which he prefers to be cold, but not ice cold. He will drink room temperature water. He sometimes expresses hunger, but he says he doesn’t know what to eat. He prefers to be covered during the fever and having the room on the cooler side at 70˚F (21˚C), without a fan. He tends to be either very, very cold or very, very hot. Putting the fan on would make him cough and he would begin to shiver. He shivers more at night and on waking. The cough is worse talking (1) and when he tries to take a deeper breath. He becomes agitated when he coughs.

He complained of a headache behind his eyes that is worse when he coughs (2). He has SOB from the least exertion such as any motion, or moving or turning in bed. It could take him fifteen minutes just to change his position in bed. The SOB is worse lying on his back and better lying on either of his sides. He uses only one pillow.

His complexion turns sometime gray with a red nose. He has been complaining of having sore gums. His tongue is cracked and is furred white. The temperature of his forehead is objectively normal. His feet have been cold. When their temperature was checked now, the tick wool socks were removed and the right foot was cold while the left one was “very, very hot.” He tends to stick his feet out of the covers, but with his tick wool socks on.

Three times he experienced dizziness with anxiety and weakness, which were all improved 20 minutes after sweating.

He was feeling guilty when his business partner and all the members of her family felt sick with covid.

He has no appetite. He doesn't know what to eat. He is belching all the time which is a chronic symptom of him.

He is not restless with the exception of the shaking with the cough, picking at bed-clothes and doing long exhales.

If the room becomes too warm he will put the AC at 70˚F and soon he shakes from being too cold. It goes back and forth constantly.

I asked the business partner if he wanted to go to the hospital. She says around here it is a bit useless, as nobody with covid comes out alive. Also KD has tried to stay away from doctors, as he always said that he was never the same person after having received the polio vaccine at five years old, "I loss my brain power." He has relied on homeopathy and natural approaches for the maintenance of his health throughout his life.

His scalp is sensitive to touch.

Homeopathic history: He has relied on homeopathy for his health for decades. He had received Sulphur for many years, Psorinum more recently as well as Lachesis.

Remedies tried so far since the covid are Sulphur, Phosphorus, Rhus toxicodendron, Arsenicum album, Nux vomica and Belladonna.

You are stricken by the violence of the cough, the paleness of the face and blueness of the fingers during the cough, the extreme sensitivity to hot and cold room temperature and the difference in temperature of the feet.

Ass.: Phos.: not very thirsty and can drink room temperature drinks.

Ars.: the lack of anxiety, aversion to warm room.

Plan: Ipecac 200C, one pellet in ½ glass of water, stir vigorously 20 times, one teaspoon q1h until we talk again in 3 hours.

The prognosis is guarded because of the severity and later stage of the disease (the twelved day of the disease) with no direct access to the patient and minimal support (just one person around the clock and she has a family with two kids).

August 31, 2021 at 4.48PM: He took the remedy at 1.53PM, 3.10PM and 4.25 PM. After the first two doses cough got worse and he became immediately more shaky

with heat all over his body, which lasted about 20-30 minutes each time. There was no shakiness after the third dose.

He is better. There is more a sense of something positive happening. His energy is better. He is not as wiped out from the slightest exertion.

He has not coughed since the third dose and he doesn't seem to have SOB.

Before the remedy his head and upper limbs would be shaky from weakness when he would sit up.

He slept after for the first and second doses.

Before the remedy his pulse was 104 and it has dropped to 92 soon after the remedy.

The difference in the temperature of his feet is unchanged.

If his well-being was 1.4/10 before the remedy it is now at 1.7.

Plan: Repeat the remedy every hour or earlier if needed. You can also skip doses if he is sleeping and an improved state.

August 31, 2021 at 8.15PM: he took three doses of Ipecac 200C since we last talked, that is at 5.25PM, 6.25PM and 7.38PM.

He is better.

He is less confused, the cough is better and the SOB is much better.

At 7.09PM he went to the bathroom on his own and returned at 7.34PM. He was shaking all over in bed. He got his last dose of Ipecac at 7.38PM and the shaking lasted another four minutes, which is much shorter than usual. He has coughed since he returned from the washroom.

The difference in the temperature of the feet is unchanged.

Plan: Repeat the remedy before bed and each time both of them are up at night.

September 1, 2021 at 9 am: He slept most of the night and was given the remedy a few times, but his condition deteriorated this morning. He slept uncovered most of the night. He is now in deep sleep with muttering and his RR is 39/m and his O2 is 80-84%. He was delirious at time at night. He would put his arms in the air, while he legs were shuddering. His breathing is labored and irregular and intermits with arrested breathing. His legs have been restless, he keeps crossing and uncrossing

them, and bending and straightening them. He is passing loud flatus and passed an involuntary stool. His energy is very low. He doesn't answer questions. His hands are very cold. He coughed only three times since last night, still violent but less so. The difference in the temperature of the feet is mostly gone, the right one is now normal and the left one is slightly on the cool side. Ipecac was repeated every 10 minutes for six doses and there were no change by 10.50AM. His legs and arms have turned red to dark red. His RR is 43 when it was counted for a full minute if he doesn't stop breathing and his pulse vacillates between 94-104. He can stop breathing for up to 15 seconds at the time.

Plan: Carboneum oxygenisatum 200 q15m for three doses.

September 1, 2021 at 11.55AM: The breathing is less labored and less frequent, from 39 down to 32. The redness of his legs are 50% better and of his arms 30% better.

Plan: Carboneum oxygenisatum 200 every 20 minutes.

September 1, 2021 at 12.55 PM: He got two doses of the remedy. He is better. He is more awake. He went to the bathroom. It was easier to get up and he could walk by himself. The breathing is much better.

Summary of the case: His condition vacillated between being better and worse, but mostly toward continual improvement. The O2 went as *low as 67* at one point. He also received Lachesis 200C, Sulphur 10M, Eup-per. 200C, Gels. 200C, Lachesis 1M, 10M and 50M, Medorrhinum 10M, Carboneum oxygenisatum 1M, 10M, 50M and 75M.

On September 27, he said, "This is the best I have felt in 25 years."

On October 7, his O2 maintain itself between 96-98%, his weight is down to 173 lbs (79 kg). He is very active, painting on a scaffold the side of his new garage. His blood sugar has been below 110 and mostly in the 90s. He had had diabetes for seven years.

November 18: "He feels his brain has returned 85-87%. I think perhaps it is a little lower functioning, like 82%. He does have improvements cognitively. He wants to come back to the practice in a week or so, but I know that his brain is not completely functioning. He did speak with a client last evening with me and he did ok, but not his usual sharpness. He said that his stamina is at 75%."

"He has experienced numbness of the right upper arm for over a month now. Sometimes it is painful. This is a symptom that he had never experienced before.

“He does periodically have the red discoloration of his nose.

“He does have small veins on the sides of the head that do come out (not nearly as large as they were during COVID-19).

“On October 19, he mentioned that his cataract in his right eye had improved to a small degree because he could see the alarm clock in the morning. He has not noticed any further improvements or mentioned again that he could see the clock, so I’m not sure that the improvement remains.

“Numbness in toes had been getting better and I think it is now going back to his precovid state.

“One of the digits on his right hand pinky finger is sensitive to touch which causes a nerve pain. It is random, so not every time it is touched.

“His facial coloring can vary from a decent coloring to gray and then also a gray/yellow. He tends to look less vibrant in the past few weeks and perhaps has more wrinkles showing up. His weight is 175.

“Right lower eyelid is still swollen, but not as large. The left is more swollen now as well.

December 27: He was prescribed Sulphur, but he was put “back on Carboneum oxygenisatum 1M, twice daily (although he sometimes forgets the 2nd dose) a little over a week and a half ago and that seemed to really help restore his mental faculties to almost completely normal (what a gift!) The improvement with memory and mental functioning seemed quite dramatic to me.

Also, he was having pain in the chest and throat pit on exertion and also in cold air, which has improved at least 40%, perhaps even more than that at this point. His stamina is still lacking, he said, however it is definitely improving. His muscle tone is continuing to restore. Blood sugar fasting numbers have been under control (mostly 97-107). His sleep is not the best; however, I believe that the apnea has been better recently. He did have difficult sleep prior to COVID-19, so not a new issue. His mood has been considerably happier since the Carbo-o. and he seems much more like his old self, which is a welcome shift.” (DJ)

On December 29: “I was at KD’s today for a period of time. I gave him the Carbn-o. 1M at around noon. He had forgotten to take it in the morning. His nose was quite red and slightly blue when I arrived; however, by the time I was leaving at 3:45 p.m. his color was drastically better. I asked him to take another dose, which he did. He called me this evening and said that his stamina exercising had improved (he could

pedal his bike faster) and the pain he has been experiencing in the throat and chest was also improved. He noticed a real shift taking the remedy twice, so I will be sure to remind him because I feel it is still providing benefits for him." (DJ)

January 4, 2022: "His mental faculties have made a drastic improvement since he started it again. His disposition and spirit seem much lighter." (DJ)

January 14, 2022: "Full return of mental faculties. His mind was working as well as, if not a little bit better than prior to COVID-19. He even started working on a book project on January 1, which he hasn't touched in years. Most days he devotes time to writing. It seems that it is easier for him to gather his thoughts and put them on paper. He has also been more relaxed and had just a lightness in his being that has been delightful. He has been very lighthearted. He has mentioned that he has had some flashbacks to when he was really sick with COVID-19. These were memories that he didn't have access to previously. He even apologized to me because he remembers now that he was difficult during some times.

"Noteworthy improvements since back on Carbn-o. 1M are as follows:

- Weakness of memory for names
- Weakness of memory for what he was about to do
- Weakness of memory for recent facts
- Weakness of memory for words
- Confusion of mind, morning

"His hearing has restored completely to what it was prior to COVID-19. It had been profoundly affected wherein I would have to speak very loudly to him during the worst of his illness. He also had a very difficult time understanding the human voice, which is also improved.

"While on Carbn-o. this last round, he had experienced 35% improvement (not a cure) in the following symptoms:

- Chest pain, cold air agg.
- Chest pain, exertion agg.
- Throat pain, throat pit, cold air agg.

- Throat pain, as if raw
- Throat pain, throat pit, exertion agg.

“His oxygenation levels are still lingering around 97. Blood sugar has been averaging around 102 (always below 110). The diabetic peripheral neuropathy is slightly better than before COVID-19. One morning last week it was 87. His weight is approximately at 180; however, he looks good and has been exercising 1-2 hours daily. His muscle tone appears to be almost completely restored. I noticed that his facial color was improved on the Carbn-o., where prior his nose was a little blue or red. Face would be either yellowish or grayish alternating with a healthy color. One morning, I went over so that we could sit together with a client and he forgot to take the Carbn-o, so I gave it to him. I did observe that he had clarity of his mind and his color improved after taking the remedy.

“A couple of interesting notes, his hair started to fall a few weeks ago and his hair as also changed in texture completely. It is now extremely fine, downy-like hair. It also looks more gray than prior to COVID-19. He said, ‘This is not my hair,’ which I would agree it is not the same. Additionally, all of his fingernails have a horizontal indentation a quarter way up from the nailed at this time.” (DJ)

16- **A Patient with Covid-19 on a ventilator.** October 1, 2021: A.S. is a 47 years old man who was intubated and put on high mode ventilator with 50% oxygen due to covid-19 severe acute respiratory syndrome on September 12. His BMI before COVID-19 was 40.3, which is defined as being as grossly obese. Part of one of his kidneys was removed for cancer in January 2021.

On September 7, he felt feverish with body aches on waking. On September 9, he had sore throat with a cough and took Tylenol (acetaminophen) every four hours.

He was hospitalized on September 11 and the next day he was transferred to the ICU as he was experiencing extreme SOB and his oxygen saturation index had dropped in the low 80%. He was put on a facemask with 5 liters of oxygen per minute, but as his condition continued to deteriorate the next day, on September 12, he was intubated and put on a high mode ventilator with 50% oxygen.

His hemoglobin level kept dropping. After blood transfusion it climbed up to 8 from 6.9 g/dL.

He was on two antibiotics, but they were stopped. He is on steroids.

His wife was asked three days in a row to give the physicians permission to disconnect her husband from the ventilator, as he was clinically dead. She refused

each time. The third time she couldn't stop crying and decided to talk to one of her friends who had talked to her about homeopathy years before.

Her friend told her to call Dr. Saine in Montreal, which she did and we went from there.

For the first time, his wife was able to visit him today in the ICU. He is lying on his abdomen unconscious under anesthesia.

I had his wife rubbed Carboneum oxygenisatum 500 for a few minutes on his leg each day. If the staff would ask what she was doing I told her to say that she was applying holy water and that he always liked to have his feet rubbed, and I also told her to watch the monitors and see if she can notice any changes soon after rubbing the remedy on her husband lower legs and feet, which it did occurred.

October 7: She was able to obtain the remedy and did the treatment on four consecutive days, that is on October 4, 5, 6 and 7. For the first time since he fell sick he is showing signs of recovery. He has been getting better every day in the last four days. Today has been his best day, as his hemoglobin began to climb on its own to 10 g/d (it was 8 after the transfusion). He is showing signs of breathing by himself. He has been opening his eyes, since they stopped the fentanyl. He is only on sedatives.

His pulse rate before the application was averaging 87 and is now averaging 106. His oxygen saturation index was 87, it is now 97-98%. His blood pressure was 130/85 and is now 116/71. His color was pale now he is pink. She reported that his legs became hot each time after rubbing the remedy over his legs.

When he was first intubated she was told that he had a 50% chance of survival but they said it was now much better odds. I told her to get Carbn-o. 1M and do the treatment twice a day, which would be soon after arriving in the ICU and just before leaving the ICU four hours later.

October 12, 2021: She was able to visit him again only yesterday, October 11 when she was able to rub the Carb-o. 1M twice on his legs, as the ICU had been closed to visitors for three days. He had been stable since her last visit on October 7, but after the Carbn-o. 1M he clearly began to improve again, which she witnessed as being more alert. He was extubated yesterday (he ended up being intubated for 29 days) and his oxygen saturation index remained at 99% with an Omni mask with 50% oxygen.

He will begin therapy tomorrow. He can't talk but he was able to communicate that he had sore throat. Plan: continue the same.

October 18, 2021: He came out of ICU on October 17. He is in pre-rehab. Three of his ICU physicians came down to see him earlier today, as "he was their miracle man, as he was clinically dead and was not supposed to be alive now," not knowing that homeopathy had been introduced at the time of his turn around. His voice is clearer. He was continued getting the Carboneum oxygenisatum on ascending potencies.

On October 26: He is still in pre-rehab. He can sit up and stand, but he can't walk or use his hands. He reported sweating on his scalp and the back of his neck when he is sleeping since he began to have a fever. He was prescribed Calcarea carbonica 200D tid.

November 3: He became more mobile after taking the Calcarea carbonica. He can now walk on his own. The sweat on his head stopped on the second night after beginning Calcarea carbonica. His blood pressure is 151/99, pulse is 113 p/m and his O2 index is 95%. Calcarea carbonica was stopped and Carboneum oxygenisatum was resumed in the 50M potency q4h, and to begin the 75M potency when it arrives in the mail.

November 8: He started the 75M earlier today. His moods have been better. He has been walking better. The burning of his left hand never changed. His vital signs improved within two days of taking the Carbn-o., the blood pressure went down to 134/86, pulse is 86 p/m and his O2 index is 97%. His speech is now perfect. His memory is also perfect except for the time he was hospitalized. The nurse and doctor in rehab remarked that they had never seen someone recover so quickly after such a prolonged period of intubation.

He returned home on November 12. He was ecstatic. Symptoms that had not change are the loosing of his hair, burning of his left hand, body aches especially in his thighs the SOB, pressure in his abdomen when lying on his side, left foot drop and chest pain with palpitation when he lies in bed in the evening.

November 26: After beginning Carboneum oxygenisatum 125M, the palpitation were 95% better and the chest pain was 80-90% better. He was now able to take a deeper breath. He was told to continue Carboneum oxygenisatum 125M q4h.

Summary of the case until January 20, 2022: On December 1, 2021, A.S. was complaining of increased chest pain that was better standing still and walking and worse taking a deep breath and while lying down, which was associated with in-

creased shortness of breath that was worse exertion, ascending stairs and lying down. He had an increased desire for ice cold drinks with dry mouth. The burning of the left hand had not yet changed. He was prescribed Phosphorus 200D tid. On December 3 he reported that the chest pain was gone and the shortness of breath was 50-60% better. The thirst was less and the dry mouth was 50% better. The burning of the hand remained high at 12/10. On December 8, he took Spigelia 200D which help the cardiac symptomatology. On December 10, he resumed taking Carboneum oxygenisatum 125M. On December 17, he was complaining of nausea that had became worse in the previous week that was aggravated lying down and better sitting and standing and he still had shortness of breath ascending stairs and chest pain but only when he took a deep breath. He was prescribed one dose of Carbn-o. 150M. On December 22, he reported that the chest pain was 90% better, the nausea 60% better, the shortness of breath ascending stairs 50-60% better and the energy 20-30% better since taking the remedy. The remedy seems to help keeping the blood pressure at a lower level, 135/88 instead of 150-184/95. Without repeating the remedy he continued to improve. However, the burning of the left hand became worse 12-13/10, “It is like on fire.” On January 17, he reported that the burning of his left hand had improved from 12-13/10 down to 9-10/10 after an acupuncture treatment but as he felt that his mental capacity was only 70% compared to before being sick he was asked to take a double dose of Carboneum oxygenisatum CM (it should have been the 150M or 175M potency) and to see if he improves, especially on the cognitive level. On January 20, he reported that the burning of his left hand had further improved from 9-10/10 down to 4-5 and that the cognitive function had improved from 7 up to 8-9/10 in the last few days. He had incidentally not received any more acupuncture treatment in the interval. I told him to take one dose of Carboneum oxygenisatum 150M and report in one week. Saine 2021-2022

17- I had one late-stage IV cancer patient who developed COVID-19 symptoms on August 12, 2021 (who was not taking Bryonia) was briefly admitted to the ER. She was discharged after having tested positive and was told that, until she started turning blue, to stay at home.

She responded immediately and quickly to Carbn-o. in ascending potencies. Within one week, she was completely healed. As of this writing (Dec. 5, 2021), she has no long-term sequalæ from the disease.

“I have treated three patients with post-COVID-19 weakness. They responded well to either Carbn-o. or what was obviously their chronic remedy. Scholtz

18- **Another severe case of COVID-19.** I have only two severe cases of COVID-19, who have known for many years. If they had been new patients in my practice, they probably would have gone to the hospital and I would not have been aware of their illness or involved in their care.

First, this 74 years old woman, she had a hemorrhagic stroke (causing expressive aphasia with mild right-sided paresis) in Sept 27, 2020, and while in Inpatient Rehab she was exposed to COVID-19. After her discharge, it was thought at that time that she was not sick with COVID-19. Several days later, I noticed episodes of tachypnea though she did not seem short of breath.

The ER physician visited her at her home to evaluate her—she was afebrile, normal vital signs, pulse ox about 92% with episodic tachypnea and rare cough but otherwise no apparent distress—she talked with my colleague for one hour. Chest X-rays were ordered, which showed bilateral ground glass opacities typical of COVID-19 pneumonia.

The ER doctor said to bring her back to the hospital if her oxygen sat dropped below 88%—it decreased to 88 multiple times (and once to 84%).

She had no characteristic symptoms, and I could not perceive a remedy for her though Stannum had a palliative effect and it would increase her sat from the 80s to up to 92

When she got up to walk, I noticed that her lower lip would turn outward and swell transiently—it was noticeable but very subtle. I recalled this symptom when reading materia medica under Carbn-o. in Allen's *Encyclopedia*: "The lower lip immensely swollen and turned outwards." I gave her a dose of Carbn-o. 30C—her oxygen sat quickly decreased, so I antidoted it with Stannum.

The next day I suddenly realized that she needed Arnica. It was not based on repertorization—the idea simply came to mind when I thought about her behavior. I realized that her pathology was expressed mostly on a mental level with few physical symptoms. The striking mental symptoms were:

> 1- Mind, well, says he is, when very sick—when I told her that a doctor, and then an X-ray technician would visit her, she literally yelled at me and told me to cancel the appointments because there was nothing wrong with her. Because she initially did not believe there was anything wrong with her, she reported no symptoms.
>
> 2- Mind, suspiciousness, mistrustfulness, and Mind, refuses, medicine, to take

the - she knows that I put remedies in water, so when I brought her a glass of water, she looked at me suspiciously and walked to her kitchen and emptied the glass of water, but then filled the same glass of water so she did in fact receive the remedy; she refused to let me check her pulse ox—she would put her hands in her axilla and close tightly so that I had no access to her finger; she refused to use the oxygen.

3- Mind, rage - she was enraged when she was refused to do as she wanted.

Because she is very sensitive, I initially gave her Arnica 30C (in water) and her oxygen saturation immediately increased; I repeated it when the saturation began to drop, and it increased again; I later gave her the 200C (in water).

Oxygen sats continued to improve. She commented that her head felt “sore” (left parietal area at the site of the craniotomy). The pain resolved within a few hours.

After taking Arnica 200c, she became cooperative because she knew she was sick.

Later that day, she suddenly began to weep and said (in a very sad voice) “I’m not well, I’m dying.” I then thought that she was out of the Arnica state.

I changed the remedy to Carbn-o. 30C and this time her oxygen saturation improved. I gave her 2 doses followed by Carbn-o. 200C, one dose. One week after the first X-ray, I asked that the chest X-rays be repeated. It was worse, showing severe bilateral ground-glass opacities but clinically she was essentially well. A week or so later, I gave her an additional dose of Carbn-o. 200c (in water) for shortness of breath with exertion. She had some dyspnea with exertion, which continued for a while but otherwise made a full recovery from COVID-19. In addition to the remedies, she took vitamins C, D, and zinc and was given oxygen 2L/min via nasal cannula (at home) mostly at night.

What I learned was that I initially focused too much on the symptoms of the COVID-19 pneumonia and missed the most profound Arnica state I have ever seen. Of course, Arnica is known to also be a remedy for patients with influenza so it is not surprising that her experience of COVID-19 pneumonia was initially expressed as Arnica. I also think that the Carbn-o. did not work initially because she needed the Arnica first [and perhaps since her craniotomy in September 2020].

Using the pulse oximeter was extremely helpful because it was objective evidence that she was improving or deteriorating. I would not go to bed unless it was 93 or higher. Sebastian 2021

19- A severe CODID-19 pneumonia case with encephalopathy and cerebellar stroke. This is a 73-year old man who had significant co-morbidities. While living alone in a rural area, he developed bronchitis and had been sick for a few weeks. He became dehydrated and fainted in a restaurant and was taken to the ER. He tested positive for COVID-19 but was told that he only had bronchitis and that his “lungs were clear.” He was discharged with prescriptions for azithromycin and prednisone 60 mg daily for 5 days.

His symptoms of cough and shortness of breath were typical of his usual complaints which typically resolved with Sulphur 30c, and this remedy did in fact bring about the usual dramatic improvement. Once feeling better, he realized that he forgot to take the medicines prescribed in the ER, so he then took them and he quickly deteriorated and repetition of Sulphur did not work, nor did any other remedies (Bry., Phos., Stann., Senega 30c potency was the only potency he had).

I was treating him long-distance and he had no pulse oximeter. A family member had been checking his pulse—initially about 80, but it began to decrease to 57.

He also had **the lower lip turned outward and swollen**—his was very noticeable and constant.

I prescribed Carbn-o. 30C at that time and the pulse increased to 80. When it dropped again, I repeated Carbn-o. 30C, the pulse increased again. He was taken to ER and was hospitalized.

An ICU doctor asked his authorization for ventilation (he agreed) but they said they would monitor him for a while first—his oxygen sat was in the 70’s upon arrival. He was started on remdesevir, azithromycin, and dexamethasone, and Vapo-thrm.

Visitors were not allowed in the ICU, but I told a family member to put Carbn-o. 200C in a bottle of juice and ask the nurse to give it to him. He drank the whole bottle of juice that day. He began to improve and never needed to be placed on the ventilator. He was discharged after the 5 days. The ICU doctor told him that he had not expected him to live.

Upon discharge, he had a good appetite and was doing well. But after about 1.5 days, his family said that he seemed weak and confused, appetite decreased, and he had neck pain which he thought was due to sleeping in an awkward position. He was also having a slight tremor of forearms. He had a history of deep vein thrombosis and pulmonary embolism.

Because of the weakness and confusion, I began to be concerned about a blood clot in the carotid/vertebral arteries, and I called for paramedics to take him to ER for evaluation.

Years ago, he had gone to the ER because of shortness of breath and profound weakness; D-dimer was positive, but because pulse ox was normal, he was sent home but was told to return for CT scan of chest the following day. His wife called me and told me that *his lips were cyanotic*. I was very stunned that they did not rule out PE in the ER.

I told his wife to give him Carb-v. 30C and to call me back in 5-10 min—he told me that he was 300% better—I told him to repeat the remedy if any shortness of breath returned. The CT scan the next day showed a small PE which was not consistent with his symptoms (from the day before.) I assumed the clot had mostly dissolved. So, because of the fear of a blood clot, I told a family member to give him Carb-v. 30C on the way to the ER.

It took about one hour to arrive at the ER—when he arrived there, he was no longer confused and had no complaints so they discharged him. I asked if they checked D-dimer or did an imaging study to rule out a stroke, but they refused to talk to me and said there was no evidence that he had a stroke and that he was just recovering from COVID-19. They never asked him to stand and did not do a thorough neuro exam.

During the next 1-2 days, he continued to remain weak, had no appetite, confusion returned, tremors were worsening, and he was unable to stand.

I told family to take him to the ER again. An MRI of the brain was done and it was found that he had had a cerebellar stroke (they had not done MRI on previous visit). They also diagnosed him with hospital-acquired pneumonia, and he was readmitted.

I told family member to give him Hyoscyamus 30C because of possible metastasis from lungs to brain, twitching/tremors, alternation between coherence and confusion.

No visitors were allowed, but I was able to talk with the nurse. Tremors/jerking/twitching continued and he was confused and seemed to want to take off his gown.

He became agitated and they decided to move him to the ICU. He became more confused and was resistant when they wanted to move him. A male nurse socked

him in the face and he lost consciousness and had respiratory arrest and had arrhythmia—“mini-code” was done and he was put on ventilator.

He was taken off the ventilator a day later. He continued to be agitated and was unable to sleep. His ex-wife told me that he was combative. I assumed that the Hyoscyamus was not correct, and I thought that he needed Stramonium. I traveled to the hospital, and when I first saw him, he appeared to be in *status epilepticus*—he was twitching and jerking from eyelashes to toes and was muttering unintelligibly. I told the ICU doctor that he appeared to be in status epilepticus and she said, “It’s just due to the stroke.”

I said that I would apply a cool compress but the purpose was to give him Stramonium 30C topically (wrists, forehead) and then I touched his mouth with the cloth.

The jerks began to subside, then resolved completely except for his feet. He was able to talk—he was very weak and he whispered to me that he was “brain-dead.” He did not seem confused at that time. I considered repeating the remedy that night, but chose not to do so.

When I arrived the following morning, he was agitated (but not having the twitching/jerking/tremors) and the ICU doctor made the decision to intubate him so that he could sleep. She said that he had not slept in four days and that that was why he was so agitated. The plan was to keep him intubated for four to five days, but each day when they decreased the sedation to see how he was doing, he was not breathing on his own at all. After one week, the doctor said that his pupils were pinpoint and sluggish and wanted to know if he had advance directives and we were told to discuss with the family what they wanted to do.

During the days he was on the vent, I had been reading about the remedies with metastasis from lungs to brain. I became certain that Hyoscyamus was the correct remedy—according to Guernsey: ‘Every muscle in the body twitches, from the eyes to the toes. This is one of his chief indications for its use in convulsions, whether epileptic or not.’

This is exactly what I witnessed. Nurses had also told me that he was grasping at things, picking at the bed sheets, trying to take off his gown. I had already given him Hyoscyamus, but there was no evidence that it helped. I thought that perhaps he never took the remedy (due to confusion or weakness), or he took the remedy but the pace of the illness was too fast for 30C potency and he needed a stronger potency or more frequent repetition. There was also the possibility that because of his sensitivity, he had a proving reaction to the one dose— I did not think this was likely.

Nash also talks about how Hyoscyamus may deteriorate into an Opium state, and this was my main question. While I was confident that he had needed Hyoscyamus earlier, did he still need it or did he now need Opium?

I felt that the doctor's questions about advance directives implied that he was dying and this was consistent with the pinpoint pupils, etc. So I gave Opium 30C on his tongue while he was on the vent.

The next morning, he opened his eyes somewhat and squeezed my hands. I then gave him Hyoscyamus 30C, and the following day he opened his eyes and was breathing on his own. He was extubated that day, and was coherent. I watched for twitching and tremors, etc., and I gave him another dose of Hysocyamus 30C, followed by 200C at next indication.

He was fully conscious, but had irregular breathing (Cheyne-Stokes) as well as ileus—I gave him Opium 200C. He slept for 4 days, then he awakened, sat up in bed, and the next day he was able to stand. He spent 2 weeks in inpatient rehab (he had difficulty eating and walking due to the lack of coordination). He subsequently made a full recovery from the stroke. He lost 30 lbs during the hospitalization, with loss of much muscle mass, and he still has some weakness and shortness of breath with exertion, but he is able to walk on a treadmill.

In addition to the above, he also had acute renal injury due to meds, intermittent atrial fibrillation, and anemia, which all resolved.

His final diagnosis was COVID-19 encephalopathy and cerebellar stroke. It was later determined that he never had the hospital-acquired pneumonia.

For a second time, a doctor told him that he did not expect him to live.

What is the reason for the metastasis to the brain? Remdesevir? steroids? After the first hospitalization, he had significant coughing, and I gave him the second dose of Carbn-o. 200C—the cough got dramatically better—was this suppressive? I suspect that remdesevir or the steroids caused the suppression, but I am not sure. Sebastian 2021

20- I have had several patients with COVID-19 pneumonia with desaturation who responded to either Carboneum oxygenisatum and more commonly Carbo vegetabilis. Actually, Carbo vegetabilis was more frequently successful with these patients in Illinois or Indiana. One patient, an experienced homeopathic vet called me with COVID-19 pneumonia and a OSI of 86% and dropping, who was ready to go to the ER. He had just taken Carbo vegetabilis 10M and within a couple minutes

the OSI was at 89-90%. By the time I spoke with him it was dropping again and I said repeat it every few minutes, which he thought he couldn't do. He repeated it often and it quickly came back to the mid 90s and he never had to go to the ER and recovered. He presented a webinar for us a few weeks later fully recovered. Fior 2021

21- **A Delta variant COVID-19 case.** New Jersey, October 2021 DC: 40 year old male. No comorbidities. Excellent general health. Good physical condition. Not overweight. DC did remarkably well on Carbn-o. Of note, his anxiety was quite intense since he first contracted COVID-19, leaving him fearful for his health, fearful to eat and these anxieties and fears were resolved within 14 hours of taking the remedy (overnight.) Other symptoms listed that were cured approximately 1 day to 5 days after commencing the remedy.

- Cough – lying on side agg.
- Cough violent
- Chest pain – after cough agg.
- Respiration – ascending stairs agg.
- Respiration – difficult – exertion agg.
- Respiration – accelerated -during sleep agg.
- Respiration – during sleep agg.
- Expectoration – blood streaked
- Anxiety about own health
- Fear of being poisoned
- Fear of being injured
- Fear of eating
- Fear of suffering
- Confusion of mind – as if intoxicated
- Confusion of mind – as of time
- Making mistakes in time
- Answering, reflecting long
- Answering, incorrectly
- Everything seems strange
- Inclination to sit and stare
- Weakness – with desire to sit down
- Weakness – slight exertion agg.
- Weakness - sitting agg.
- Delusions – on going to sleep
- Throat- sensation of coldness – esophagus

- Diarrhea – morning after rising

Patient was prescribed Carbn-o. 200C wherein the above symptoms were cured. Deuvall 2021

22- **Another Delta COVID-19 case.** October 2021. D.B. is a 70-year old male from Pennsylvania, who is not a patient of our office, and is not overweight and is in good physical condition. He has a family history of early heart disease. He was vaccinated in 2021 to prevent COVID-19 and reported developing hypertension reported it. He tested positive for CODID-19. His presenting symptoms were:

- Confusion of mind – as of time
- Answering, reflecting long
- Prostration of mind
- Anxiety about one's own health
- Confusion of mind – as if intoxicated
- Smell wanting (complete loss of smell)
- Taste wanting (complete loss of smell)
- Hypertension
- Weakness –exertion agg.

D.B. was not a prior patient of our office, but contacted me with COVID-19. His symptoms of acute COVID-19 were cured mostly with Bryonia; however, his taste and smell were completely wanting even a month and a half after recovery. He also had lingering mental dullness and confusions in time. He reported that he had suffered from hypertension ever since the COVID-19 vaccination. D.B. started Carbn-o. 200C, which restored his taste and smell within two weeks to 75%, after three weeks to 90% and he is still currently taking the remedy (January 2022), which is continuing to yield benefits. During his last check in, he mentioned that his stamina to ride his bicycle had improved dramatically since Carbn-o. and his blood pressure was decreasing steadily. I mailed Carbn-o. 1M out to him this past week and he will start that as soon as he receives it. Deuvall 2022

23- **An Omicron COVID-19 case.** December 2021. R.L. is a 55-year old female from NewYork. Prior breast cancer diagnoses with conventional treatment. Thyroid medications taken since childhood. Homeopathic treatment after multiple surgeries and unable to cope with side effects. Covid vaccination 2021. Minimally overweight, perhaps 10 pounds. Tested positive for COVID-19.

- Respiration – ascending stairs agg.

- Respiration – difficult – exertion agg.
- Chest pain –Deep breathing agg.
- Head pain – forehead
- Head pain – forehead – pulsating
- Head pain extending to the eyes
- Head pain - stooping amel.
- Head pain –noise agg.
- Head pain - from light agg.
- Head pain accompanied by nausea
- Head pain accompanied by mental dullness
- Heaviness of head, forehead
- Stomach – nausea – motion agg.
- Stomach – nausea – motion of eyes agg.
- Prostration of mind
- Anxiety about health of loved ones
- Fear about health of loved ones
- Ailments from cares and worries over a loved one
- Fear something bad will happen
- Confusion of mind – as if intoxicated
- Confusion of mind – as of time
- Inclination to sit and stare
- Answering, reflecting long
- Everything seems strange
- Weakness – with desire to sit down
- Weakness – slight exertion agg.

RL was prescribed Carbn-o. 200C wherein the above symptoms were cured. Patient responded to the remedy and reported within two hours that her mental confusion, dullness and fogginess were already significantly improved. Within 12-18 hours, respiration had improved. Additionally, RL had been prescribed remedies that diminished her head pain symptoms approximately 75%; however, those symptoms stated above regarding head pain and nausea where completely cured after commencing the Carbn-o. 200C. Deuvall 2022

24- **A series of Omicron variants COVID-19 cases.** From the end of December 2021 into the first two weeks of 2022, I was consulted by 25 patients in areas where Omicron had begun to be dominant.

The symptomatology was completely different that the original Wuhan strain and

the Alpha and Delta variants in which Bryonia was indicated in about 65-70% of my patients.

Twenty-three of the 25 patients with confirmed or suspected Covid (tested and untested) in areas where the Omicron variant is dominant and who had more or less the same symptoms responded quickly and very well to each dose of Carbnoneum oxygenisatum.

The two patients who didn't require Carbn-o. were a patient in Greece with MS and one in Illinois with lupus who both contacted me about a week into their acute sickness and who just repeated their chronic remedy more often during irthe acute illness (Sepia for the one and Causticum for the second one) and felt that they were recovering well by the time we talked.

The most characteristic symptoms are **great to extreme lethargy, apathy, loss of will, mental dullness and prostration, confusion, weakness, great sleepiness and extreme shortness of breath that are worse from the slightest exertion and better from open air.** Another interesting symptoms that can be present when Carbn-o. is indicated is **disorientation in time and space** and **awkwardness**.

The patient can have a panoply of other symptoms: cough, sore throat, sore skin, painful joints, fever, chilliness, high thirst but none of these would be decisive to find the remedy.

The state of apathy is very particular to Carboneum oxygenisatum. One patient who had been in bed for 13 days said that I know I have to go to the washroom but I get so exhausted from just thinking about it that I don't go. She said once she waited four hours to get up to go to the washroom. She took Carbn-o. 1M around 11.30AM q30m and when I called her back around 1PM she said that she fell so well after the second dose that she got out of the same pj she had been in since Dec. 24, took a shower and got dressed for the time in two weeks.

Carbn-o. was first thought of and used for patients in the later stage of the COVID-19. However a colleague from Holland had found it to be the genius epidemicus during the Delta phase in his area.

It may be difficult for most to prescribe Carbn-o. as it will not come out in repertorization unless you would have a version of Complete Repertory 4.5 in which we have made over 2000 additions that were taken from a review of its toxicology and the excellent proving that the MMPP conducted of it in the spring of 2020.

If you refer to one of the cases (case 16 in this mongraph), I presented the AIH October 16 update webinar of a patient that had been on a ventilator for about three weeks and they were ready to pull the plug because the patient was “clinically dead” I told the wife to rub some “holy water” with Carbn-o. and this patient came out of ICU in about five days and he is still responding to Carbn-o. in increasing potencies for the long term effects of severe Covid and intubation, i.e., chest pain, palpitation, SOB, hypertension, paresthesia, paresis, energy, diabetes, etc.

Beware that the remedy must be given often. Patients have often said that they feel improvement after each dose and when they wait before repeating when they stop improving they either stay at the same level or have already started to deteriorate.

Many of the cured symptoms in these patients are confirmation of known symptoms of Carbn-o. and include **inability to write**, poor concentration to read, **sitting and staring**, **answering reflecting long, irritability at trifles**, **chest pain worse taking a deep breath**, **pressing forehead headache with eye pain and nausea**, **weakness with the desire to sit down**, **prolonged sleep**, **feeling sore all over the body** worse touch, pressure, rubbing and lying on the part, sensitive scalp worse touch and brushing the hair, vertigo with nausea and shortness of breath worse motion, exertion, ascending a hill and closing the eyes, **sore eyes worse touch** and pressure**, cough worse deep breathing**, **talking, ascending stairs,** becoming cold and from cold drinks and better open air, shortness of breath worse in the evening and stooping, wheezing on lying down in the evening, **chest pain from coughing**, **back pain from coughing**, expectoration difficult, glassy eyes, **back worse jarring and walking**, **appetite wanting**, **bad taste in the mouth**, nausea riding in the car, nausea worse from odors, smells of food, drinking motion and better open air, thirstlessness during the chills, involuntary stools, worse warm room, **nasal obstruction, dry throat** worse at night and with thirst**,** rawness of the throat worse swallowing, better cold and warm drinks, swollen cervical lymph nodes, heaviness of the tongue, desire for cold drinks, perspiration of the back at night, perspiration at night and during sleep, worse evening and night, **awkward, drops things**, **continued low-grade fever**, **chilliness not better from the warmth of the stove**, worse in general at 3 or 4 PM and **better after sleep and siesta**. Saine 2022

25- **Two other Omicron cases.** Here is the unfolding symptoms that required Carbn-o. The first remedy that helped was Hepar sulphur during the deep chill and face pain stage. Then many remedies were tried with basically no effect until Carbn-o. Carbon-o made a huge difference but did not take the case to resolution. A few weeks later there is still low energy and constant post nasal drip. Here's greater detail:

Eight days ago, on January 11, 2022, I received Peter Gold’s email containing Dr. Andre Saine’s discourse on Carbn-o. in regards to the variants.

To support Andre's efforts I would like to report 2 cases, myself and my husband that struggled for over 2 weeks trying all the basic remedies, Ars., Gels. and Oscillococcinum and numerous others with no appreciable benefit until discovering a direct hit with Carbn-o. and the initial results were dramatic!

#1 - The case started with a sore throat for days, developing into a cough with a sense of a deep postnasal drip tickle, almost feeling like it tickled down to the sternum. Next I had five days of extremely painful face pains, like a deep sinus infection with no sign of congestion or mucus. I was very cold for days (and I'm in Florida where it's quite warm and I still could not get warm) and a dose of Hepar sulphur helped quite a bit in this regard but the fatigue was profound and unhelped by the Hep. I also tried Silica but no help and then other, still no help. My symptoms seemed to move about considerably and aggravate the next day at about 4 pm so I thought Pulsatilla should help but no assistance was experienced by Pulsatilla either. I also had a fever of about 100.2˚F for days. The most prominent symptom was extreme lethargy and a profound sense of discomfort in my back. For days on end I felt like my intercostal tissue were swollen and pulled down and no position was comfortable to be in. I spent many days in the same pajamas with no interest or motivation to shower or get dressed. I could barely prep food for myself, the slighted movement was daunting and felt very effortful. If I thought of doing something in my art I would have a sense of anxiety about it and almost a burning pain in my body about the idea of making the effort which I tried a few times and would inevitably turn into an exercise in futility as I would just stare at what needed to be done and go over in my head the steps that I would need to do only to go in circles and get nowhere, super frustrating. Or I would think about an artistic task that I normally could do and I’d get super anxious and start burning inside the more I thought about it. I lost a bunch of weight out of lack of energy to eat, even though I was hungry. I tried more Gels. and it seemed to give me a little more energy but not enough to be too encouraged. By the second week all of a sudden I lost my sense of smell, I could not smell rotten fish, ammonia, tea tree oil or coffee!

Odd symptom, the toe pads on bottom of my foot each morning feels swollen. More than 2 weeks into this I finally intuited the possibility of Carbn-o. out of desperation. I found that just holding a 30C sent my symptoms into instant increase! I knew I was onto something! I instantly lied down for a few hours and could tell I had a lot more energy when I got back up. I'm on dose three right now and making steady progress. This progress lasted a few days to get my energy in a better place but then ceased to improve further. A full week later and I am about the same each day

with a postnasal drip, occasional cough and low energy. Doesn't seem as if there is a remedy to help with this. [Comment by André Saine: I found that in this state the potency must be changed every 5 days about.]

#2 - My husband got what I have about a week later and has suffered considerable head pain on the vertex for days, he too was extremely lethargic also with low-grade fever. He tried Ars. and Gels. and felt a bit better but not strongly so. I gave him one oral dose of the Carbn-o. 30C and it had a dramatic uptick in energy for him. The second dose caused him to be extremely agitated and angry. He felt like bugs crawling all over him and could not get comfortable and was really angry with me for giving it to him. I bit my tongue and prayed it would settle out in the morning and it did just that! He was feeling much better in the morning of January 12, 2022, I then gave it again the next day with no further benefit. Now 8 days later he still has morning phlegm that he has a fit of trouble coughing up, and low level fatigue. [Same comment as above the potency must be raised]. Tobey 2022

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