was due then to cardiac weakness. For tympanitis injections of soap and water or oil, emulsion of assafetida and turpentine were used, with occasional doses of calomel and high insertion of the rectal tube left in for half an hour, the latter being extremely beneficial and facilitating the expulsion of gas. Cracked, bleeding lips and other conditions were treated as they arose. The infant made a perfect convalescence; the first case of recovery in infancy with the meningeal complications I find reported.

The case shows how one might have been led to the diagnosis of meningitis from the increasing severity of the nervous symptoms, had not the mother suffered from typhoid, or had not the Widal reaction been persistently sought for, and a few cases, occurring under such conditions, apparently sporadically, might call for a little more care in differentiating epidermic cerebro-spinal meningitis than is perhaps sometimes utilized. After carefully considering the possible exposures of the child to the infection, when his food subsequent to the breast nursing can be excluded as a source, the apparently efficient isolation of the mother, the gradual increase in the pallor and irritability of the child during the latter weeks, which may have been due to change of food, although there was no apparent loss of weight or gastro-intestinal disturbances, I am forced to believe that the period of incubation may have been at least thirty-nine days, that the insusceptibility of the infant was overcome only after a long struggle. As, however, I saw the mother only in consultation, I am unable to vouch for the constancy of the quarantine, but if the child contracted the disease during the thirty-nine days previous to the appearance of the symptoms, more care should be used to protect the susceptible than is deemed necessary or exercised by our contemporary therapeutists.

Clinical Department.

AN ATTEMPTED SUICIDE BY ILLUMINATING GAS.¹

BY FRANK HOLYOKE, M.D., HOLYOKE, MASS.

THE toxic results of inhalation of illuminating gas are twofold, causing in some cases simple asphyxiation; in others "an intoxication by prolonged contact with the gas, leading to a toxemia and the formation of co-hemoglobin" (E. H. Bartley).

The following case is of the second type, in the acute form, and occurred on July the first, when the roomers in an apartment house were disturbed by moaning heard in a neighboring room which had continued through the afternoon and evening until eight o'clock. They knocked at the door but were unanswered. The police were called and on breaking in the door, which was locked and corked with rags and the windows closed tightly, quite effectually preventing the escape of the illuminating gas which was pouring into the room from two open jets of the chandelier, they discovered the air unbreathable and Mr. A. C., a man

of forty-four years of age, lying back in an easy chair unconscious, in a high-studded room which was 12 by 15 feet square. He was removed from the room and the windows and doors thrown open. It may be well to note that this illuminating gas is a mixture which averages, through the year, two parts coal and one part water gas, according to the state inspector's test at Holyoke.

Dr. J. C. Hubbard of Holyoke answered the call within ten minutes and found his patient perspiring profusely, his clothes being drenched with sweat; the pupils were dilated, face cyanotic, the limbs relaxed, pulse imperceptible at the wrist, but a feeble heart's action was felt, beating about 140 per minute. Respiration was very slow and gasping and the jaw had dropped — in short what appeared to be a state of collapse.

Dr. Hubbard first tried artificial respiration, which was followed by increased frequency in respiration, while $\frac{1}{16}$ gr. of sulphate of strychnia in divided doses and $\frac{1}{16}$ gr. of nitro-glycerine, during the half-hour that he was in attendance, restored the pulse to the wrist, but the doctor had little hopes of his recovery. The patient being removed to the House of Providence Hospital, I was called as medical examiner at 9.30 P.M. I found him lying in bed stripped of clothing but covered with blankets. The body was pale and cold. The arms and legs had now become cold, rigid and in a state of tetanic convulsion. The pupils were markedly dilated and did not respond to light. The breath was strong with the naphtha-like odor of gas, which did not entirely disappear until the fourth day. Temperature by rectum was subnormal $96\frac{1}{2}$, which according to statistics appears exceptional. The pulse 142, respiration 32 and shallow, with spasmodic action as though snapping for breath, and a thin frothy mucus had to be wiped occasionally from between the lips.

Physical examination showed feeble heart's action and large mucus rales over both lungs. These rales cleared completely on the second day. A strong draught of fresh air passing through the room from open windows, warmth to the body and heart stimulants hypodermically were the only measures taken for immediate relief.

The normal temperature was not reached until the third day, when it rose steadily to $100\frac{3}{4}$, the highest point reached, on the evening of the sixth day, and gradually diminished from the sixth, with slight feverish action throughout the second week. The pulse fell to 80 on the seventh day and respiration to 20. He voided urine in considerable quantity and involuntarily during the first three days. Bowels first moved by enema on the second day. Through the night of the second day he gradually regained consciousness, became quite rational and began to take liquid nourishment, though he swallowed with difficulty and cold water caused severe pain in the mouth and throat, producing a spasmodic action of the throat. He vomited his milk several times on the third day. For the first two days of consciousness he complained of great pain in his head, which was general, not localized, and also of a numbness of the limbs which continued for nearly a week.

On the morning of the second day my attention was called to a peculiar condition on the back of the shoulder blades and over the sacrum and buttocks, which parts came in direct contact with the easy chair where he was originally found sitting. These parts were thickened and brawny, bright red, hot and very sensitive as though scalded but the brawny thickening was too deep for an external burn or for a recent bed sore. The skin was not blistered, but loosened in a

¹ Read before The Massachusetts Medico-Legal Society, Oct. 7, 1903.

few days, leaving a large denuded surface which healed over very slowly and was sensitive for several weeks. Numbness of the legs disappeared in about one week's time and as this abated he suffered with sharp shooting pains in the left leg and foot, accompanied with motor paralysis of the muscles controlling foot and toes, from which he has not at expiration of twelve weeks fully recovered, though the muscles respond to the faradic current.

It was also interesting to observe the result of paralysis of the vasomotors of this leg, which, when allowed to hang from the bed, or, as in the second week, when he tried to stand up, rapidly filled with blood, the skin becoming turgid and deep purple with distended vessels until he felt faint. The right leg was unaffected, retaining its normal color. This vasomotor paralysis disappeared entirely by the sixth week and also the shooting pains in the limb.

The latest article that I have seen on "poisoning by illuminating gas" is a very clever one by Dr. Paul M. Pitcher published in the *Brooklyn Medical Journal* of May, 1903, in which he concludes that: "The toxic effects of inhaling illuminating gas are due primarily to chemical changes produced in the constituent elements of the blood, this being a substitution of carbon monoxide hemoglobin for the oxy-hemoglobin, so diminishing the supply of oxygen to the tissues. Further that the *direct cause* of most of the symptoms is the diminished percentage of oxygen contained in the arterial blood supplying the central nervous system" (compare Hal-dane, p. 443).

We may concede this effect of carbonic monoxide (the chief poisonous agent of illuminating gas) to be the primary cause of the paralysis of the sensory, the motor and vasomotor nerves, producing the variety of symptoms here observed, possibly also the exquisite sensitiveness of the mouth and throat to cold drinks. I am at a loss, however, to account for the peculiar *external* appearances over the buttocks and shoulders. The question naturally arises if it can be due to any local irritation from the poisoned sweat in prolonged and close contact with a surface preventing the free escape of the perspiration from those parts, or is it because the pressure, long continued, has caused a temporary emptying of the blood vessels which, being suddenly relieved from pressure at that part, react *more* strongly and fill to *excess* by reason of the *general* tendency to vasomotor paralysis, causing here such a congestion as to result in inflammation of the skin tissues? The latter supposition seems to me more plausible.

A CASE OF TRAUMATIC CEREBRAL HEMORRHAGE.¹

BY C. A. ATWOOD, M.D., TAUNTON, MASS.

I PRESENT to your notice to-day a case of traumatic cerebral hemorrhage.

It seems to me that the case may be of in-

¹ Read before the Medico-Legal Society, Oct. 7, 1903.

terest, not only in its medico-legal relation, but surgically.

E. M., a French-Canadian, age forty-four, was returning from the city to his home on an electric car at about 9 p.m., June 7, 1902. While the car was in motion and fearing probably that it would not stop in front of his house, he attempted to save time and distance by getting off. The result was that he was thrown into the road, striking upon the back of his head. The road had recently been repaired by filling with sand and it was soft. He got up and went to his home. A short time after he complained of not feeling very well and sent for his physician. The physician at that time found no serious symptoms. E. M.'s condition, however, became worse and the physician was again summoned, who found him unconscious and pronounced the case one of apoplexy, for which he bled him.

E. M. never regained consciousness and died at 6.40 a.m., June 8, 1902. I viewed the body and performed an autopsy at 3 p.m. There were no visible marks of violence on the body. The symptoms followed by coma before death all pointed to some acute cerebral injury. On reflecting back the scalp, beneath it and near the occipital protuberance of the occipital bone was found a blood clot. The cranium was opened and there was no fracture of the skull. The dura was tense. When opened quite an amount of cerebral fluid escaped. The sinuses and vessels of the pia and brain were full and engorged. In the frontal lobe of the left hemisphere near the longitudinal sinus was a blood clot the size of a golf ball. This was the only lesion found and caused death by cerebral pressure. Macroscopically there were no other lesions.

The theory is that the man fell forcibly on to the back of the head with sufficient momental violence to cause by contre-coup a rupture of a blood vessel in the anterior portion of the brain opposite the site of the original injury.

The Court asked this question: "If there were no external marks of violence and no fracture of the skull how was it possible to state that the cause of death was due to violence or the fall?" The answer was: "The man never having had any cerebral symptoms of disease before, and in this case having come on immediately or soon after an injury, with the finding of an ante-mortem blood clot beneath the scalp, in the region where he was struck when he fell with force, was evidence enough to consider the hemorrhage of the brain, which caused death, to be due to injury and not to previous disease, together with the fact that there was no other pathological lesion present." *The Court so ruled.*

It may not be too much out of place to mention surgically something about this case. At the present time I believe that some of our more progressive surgeons advise even in the absence of localizing symptoms, if coma and other conditions are progressive, to interfere; that is, to open the skull in the sense of an exploratory trephining. The usual rule would probably be in a case like this, if undertaken, to trephine at the seat of injury. I have thought since if this had been attempted that it would certainly have been a long hunt for the seat of injury.