In the case of ethylene and acetylene the matter is more serious. I have never used the latter as an anaesthetic, but I give an oxygen-ethylene mixture for dental surgery as a routine, because I find it better than nitrous oxide. I have been told that bad explosions have occurred with both these gases, and that fatal consequences have ensued. In these cases, however, an explosive mixture exists not only in the patient's lungs, but in the bag used, and there is some risk of an explosive mixture accumulating in a room since so much larger quantities are used. With even saturation in a small room of 1,000 cubic feet a cylinder of 500 gallons would have to be emptied to provide with air a mixture of 1 in 12. This is a considerable volume of ethylene, but saturation will not ordinarily be even; an accumulation of sufficient concentration is always present near the facepiece, so that a lighted cigarette (or, it is conjectured, a spark from a static charge gained by the gas in issuing from the cylinder union) could cause a violent and tragic explosion.

Dr. F. W. Higgins, working at Manchester University in connexion with mining gases, found that the ignition point of an oxygen-ethylene mixture was unusually low, and that the ignition point of a nitrous oxide-ethylene mixture was still lower. In dental practice I use quantities of 10 to 50 gallons frequently, with nitrous oxide and oxygen added, often in a room containing an open fire. I am careful about lights (dentists often have a small Bunsen burner alight) near the patient. A short time ago I kept a man under with an oxygen-ethylene anaesthetic mixture in a dental chair for over an hour and a quarter with nothing but a nose-cap; his condition afterwards was not abnormal.

In view of the likelihood of minute sparking in lamp holders, whether of the ordinary design (bayonet) or any other, I dislike electric lights too near. Where they have to be used it is advisable to make sure that the electrical connexion between the lamp and the socket is sound.

CEREBRAL HAEMORRHAGE IN A YOUNG WOMAN.

BY

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THE following case presents several features of interest, being the account of an apoplectic fit actually observed during a medical examination, so that an opportunity for first-hand information of its onset and progress was presented. It may be recalled that in *The Story of a Surgeon* Sir John Bland-Sutton mentions a case that he personally observed. The type of hyperpiesia in the present case is also discussed, and the occurrence of an unusual example of automatic speech is described.

A school teacher, aged 30, consulted her doctor because of painless haematuria, and was found to have a systolic blood pressure of about 300 mm. Hg. In consequence she was admitted to the Prince of Wales's Hospital under Dr. Browning Alexander to be investigated. The patient had had measles and chicken-pox as a child, but no scarlet, rheumatic, or other severe fever. Her appendix had been removed five years previously, and she had diphtheria severely a year later, being confined to bed for two months. Subject to headaches for many years, she had suffered more acutely for the last three months. Occasionally there was an aura of black spots, and she had often felt sick during the attacks, though vomiting was rare. Two months before admission she had an attack of severe backache, which persisted for five weeks. For two weeks there had been a blurring of vision in the right eye, and she remembered a similar blurring in the left eye some time back; latterly her nose had bled on two occasions. There had been no frequency of micturition, and the menses were always regular; latterly she had felt breathless at times, and had had palpitations. None of the symptoms troubled her sufficiently to seek medical advice until the appearance of blood in the urine a few weeks before admission. There was nothing of importance in the family history.

Condition on Admission.

The patient was thin; the skin was rather dark and the eyes were perceptibly prominent. The thyroid was not enlarged, but the tonsils were definitely infected.

The pulse was regular, forceful, and rapid (110 to 120 per minute). The radial artery was tough and tortuous, and the blood pressure was 280/210. The apex beat was just out of the nipple line; the sounds were forceful, but no bruits were heard.

The basal metabolism inferred by Read's formula was +8. No abnormalities were found in either the lungs or abdomen. The pupils were equal and the reactions normal. The biceps, triceps, supinator, knee- and ankle-jerks were present and equal; the abdominal reflexes were not obtained, and the plantar reflexes were both flexor. The muscles were small, but their power was good.

The Stroke.

At 6.45 p.m., directly after the blood pressure had been taken and the preceding observations had been made, something amiss was suspected. Whereas a few minutes before the patient had responded to the examination a little nervously perhaps, but otherwise in a perfectly normal manner, an interrogation was now received with eyes open and consciousness apparently undisturbed, but with an abortive effort to speak that resembled the functional aphasia of a nervous person in a witness-box. The words would not form, and the patient smiled her helplessness away. There was confusion, but no anxiety on her face, and believing that she was indeed temporarily unnerved, she was reassured, and the examination was suspended for a few minutes.

Returning after five minutes, the motor aphasia was found to be more definite, and for the first time a facial asymmetry was noticed. The nervous system was then re-examined, and the following facts were observed: a complete right facial palsy; a flaccid paralysis of the right arm and weakness of the right leg; a right-sided blunting of sensations of touch and pain; and an extensor right plantar reflex. The patient was quite conscious, and understood all that was said to her. She was restless, anxious, and sweating.

At 7 p.m. amyl nitrite was inhaled, and she vomited a little. The systolic blood pressure was now 160 mm. Hg. She was drowsy, and later she went to sleep. At 11 p.m. she was still asleep, and the hemiplegia was quite complete. The systolic blood pressure was 240 mm. Hg.

Subsequent Progress.

The following morning the patient was conscious but drowsy; when spoken to she smiled, and said repeatedly, "I see." She was incontinent of urine and faeces. The urine contained a trace of albumin, but no blood was detected by chemical tests. The blood urea was 51 mg. per cent. Both fundi showed many old haemorrhages, with one fresh one on the margin of the right disc; otherwise the discs were normal.

On the fifth day following the stroke the sphincter control returned. The patient understood all that was said to her, and read the daily papers with interest, but she was still only able to reiterate "I see." The right-sided paralysis was still present, but the jerks were not exaggerated, and the paralysis was still flaccid. No clonus was obtained. The right plantar reflex was doubtfully extensor, the left one flexor. Mr. Flemming reported on the eyes as follows: "Eye movements jerky, but no true nystagmus. No evident paralysis or strabismus. The fundi show a slight neuritis, with much haemorrhagic exudation round the discs. Some radiating lines of exudation from the maculae, as in albuminuric retinitis. Arteries thick, and veins dilated and tortuous."

Towards the end of the second week the speech began to improve. She was able to answer questions, concentrating persistently, but at times the word would not come. Both plantar reflexes were definitely flexor.

One month after the stroke the improvement was more marked, the facial palsy, as well as speech and muscle movement, being much improved. The musculature was very flabby, and there was wasting of the right arm and leg, where the jerks were difficult to elicit. The right plantar reflex was doubtfully flexor. Sensation remained diminished. The sight was deteriorating, and there was marked swelling of both optic discs.

On the thirty-sixth day she had severe headache, and vomited twice. Following this the power of speech, except for the same phrase of "I see," was lost, but an hour later it had returned to its former level. Four days later the tonsils were enucleated under a general anaesthetic by Mr. Ibbotson, since it was considered they were a definite toxic factor. Bleeding was minimal, and the patient stood the operation very well.

On the forty-seventh day the patient felt faint quite suddenly, consciousness, however, being undisturbed. The pulse rate fell from its usual figure of 100-120 to 70 per minute, and the heart sounds were faint. The pupils were small, and reacted to light sluggishly, but no further changes were noted in the central nervous system. The patient lay quietly, with her eyes closed, but speech was undisturbed. The systolic blood pressure had dropped from its usual height to 105 mm. Hg. One hour later it was 170; three hours later 210; five hours later 220; and eight hours after the onset it was 230 systolic. A similar attack had been noticed by the nurses two days previously.

For nearly two more months the patient continued to progress slowly. The pulse rate seldom varied further than the range of

110-120, the systolic blood pressure kept around 220 mm. Hg, and the temperature remained between 98° and 99° F. Repeated examinations of the urine showed little or no albumin; the daily output averaged about 60 ounces during the first three weeks of her stay, and then gradually dropped to about 30 ounces. The blood urea test was repeated several times, but always gave a figure well within the normal. The Wassermann reaction was negative in the blood and in the cerebro-spinal fluid, a lumbar puncture having been performed ten days after the stroke. The fluid was not under pressure, and no blood pigment was found. The sugar estimation was 0.087 per cent., and the chlorides 0.74 per cent. The right-sided paralysis improved steadily, and towards the end of the period a fair amount of power had returned. The arm recovered sufficiently to enable her to feed herself. Passive and active movements were given daily.

Three months after the stroke the general condition began to deteriorate, and evidence of a failing heart appeared. A low-grade pneumonia set in, the patient became delirious, and the blood pressure gradually fell, so that within a week it had dropped to 140. The urine flow diminished, albumin was found in appreciable amounts, and the blood urea increased to 190 mg. per cent. There was a polymorphonuclear leucocytosis, but the temperature remained subnormal. Death ensued nineteen weeks after the initial stroke.

Post-mortem Report.

The body was thin, and there was wasting of the musculature, especially of the right upper and lower limbs. The lungs showed a terminal congestion, and there were some pneumonic patches at the right base. The heart was small and atrophied; aortic arterio-sclerosis was not marked. The liver showed nutmeg change, and the spleen was small and fibrotic. The kidneys were also small and fibrotic, and there was terminal congestion; the cortex was fairly well preserved.

A large old haemorrhage occupied practically the whole of the basal ganglia of the left lobe of the cerebrum, with the original site in the anterior part, about the level of the genu of the internal capsule. The arteries were not unduly changed, and thickening was not a feature.

Hypertension to the extent exhibited by the patient is of rare occurrence at that age, the more so when it does not fall into one of the three common groups of cardiac, renal, or arterial disease. The kidneys were damaged, it is true, but the changes were consistent with what would be considered a normal response to the high blood pressure; the arteries were surprisingly healthy, and their changes cannot be accepted as primary in origin. Moreover, the two attacks during which the blood pressure fell to normal limits strengthen the opinion that the arterio-sclerosis was slight, and that the contractions and relaxations in these vessels were active movements bespeaking an intact musculature. Most diseases of doubtful origin have been ascribed at various times to infections, both focal and acute; this patient had infected tonsils, and she had had diphtheria four years previously. However, Walker and Hare consider that there is no evidence of pressors in infections, and their figures show that a history of diphtheria is more common in normal people than in hypertonics.

There remains, consequently, the consideration whether the case is to be regarded as one of "essential hypertension," due, as some observers believe, to a constitutional state, inherited as a dominant Mendelian characteristic, and rendered active in response to varied stimuli. Having regard to this possibility, permission was given lately to determine the blood pressures of other members of the family, though further examinations were not permissible. The following were the readings obtained: father, aged 59, 155/90; mother, aged 60, 215/140; sister, aged 29, 130/90; sister, aged 21, 155/80. The mother has had rheumatic fever on two occasions, but is in perfect health, and is not supposed to have any heart involvement. The younger sister was quite well until two years ago, when she was in a mental home for a few months, suffering from depression and delusions of unworthiness which had followed an unfortunate love affair. Her recovery has been complete. There is a brother who was not available for examination. Whereas such a casual examination does not permit of a definite conclusion being stated, there seems to be fair evidence in favour of the view that the patient came from a hypertonic stock, and that her case can be placed in the class of "essential hypertonics" mentioned above.

Sir Thomas Horder, in a recent discussion on high blood pressure at the Harveian Society, spoke of "essential hyperpiesia" as being possibly the expression of an endocrine disturbance, adrenal, pituitary, or both. The

patient certainly exhibited symptoms attributable to a thyro-adrenal complex—the persistent tachycardia, the dilated pupils, and the moderately prominent eyes. Read's formula, which is considered to be a reliable inference of the basal metabolism, was in this patient slightly raised above the normal figure, and, consequently, it seems justifiable to conclude that the condition was one of congenital hyperpiesia of endocrine origin.

In right-sided hemiplegia, when the speech centre is involved, the aphasia may be complete except for "automatic speech," which may be the first hint of the returning function. In this connexion Kinnier Wilson relates how a patient, on witnessing the spectacle of a Zeppelin in flames, exclaimed "Hallelujah," and then lapsed into a silence as complete as before. In the case under discussion the words "I see" occurred repeatedly after the initial stroke, and subsequently, when speech was lost again. It may well be that the words were occasioned often during her avocation as a school mistress, and that, becoming spontaneous by constant use, they formed the whole extent of her expletives, and at times after her stroke, the sole intact connexion between her speech centre and the exterior.

I wish to thank Dr. Browning Alexander for his permission to publish this case, and Dr. Trounce for his report on the previous history of the family.

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A CASE OF OLD-STANDING TUBERCULOUS KIDNEY.

BY

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THE case here recorded is of interest as showing that even advanced tuberculous changes in the kidney are compatible with prolonged life and comparative good health.

A man, aged 47, was admitted to Guy's Hospital in June, 1930. A brother had died from consumption at the age of 21; the patient lived in the same house with him during most of his illness. At the age of 18 the patient noticed that his urine was turbid, and he consulted the late Professor Julius Dreschfeld in Manchester, who made a diagnosis of tuberculous kidney. For some years the patient was kept under observation, and on several occasions had attacks of haematuria, often severe. At this time he had occasional fever and urinary symptoms such as frequency, but his general health remained sufficiently good for him to work for engineering examinations, and by about the age of 21 he was quite fit, apart from some frequency of micturition and occasional burning sensations in the region of the bladder. From this time until the outbreak of war he led an active life as a civil engineer; in 1914 he joined the Royal Engineers and went to France, serving in a Field Company.

In 1915, while on active service, he passed a few very minute stones, but did not have to report sick. In 1916, while still in France, he had a severe attack of pain in the bladder, and passed very large quantities of pus in the urine. He was invalided to England, and suprapubic cystotomy was performed in a military hospital, but no stones were discovered. The wound healed satisfactorily; the patient made an apparently complete recovery, and was able to return to active service in France in 1917. While on service he was exposed to conditions of great hardship; after six months his symptoms recurred and he was again invalided home. With medical treatment and a vaccine he again recovered completely, and was able to join the Royal Air Force in 1918. After the war he led an active life as an engineer and lived many years in the East. During this period his general health remained good and he put on a great deal of weight, eventually reaching close on 20 st. From time to time there was pyuria and slight frequency.

On May 1st, 1930, he suddenly became very ill; the urine became scanty and extremely purulent, and there was practically anuria for four days. The temperature was high—up to 104°—with severe diarrhoea and vomiting. With transient periods of improvement his illness lasted for six weeks, and he was often thought to be moribund; on several occasions he had severe rigors with high fever. Until he was transferred to Guy's Hospital there were no facilities for investigation, but clinically he was considered to be uraemic, possibly as a result of calculous anuria.