hypo-aesthesia in cases of pruritus is very satisfactory. The percaïne content in 10 c.cm. of this solution (0.05 gram) is well within the limits of safety for adults, and I have only once had any general reaction: this was in the case of a man with pruritus, who, on a hot summer's day recently, became slightly pale and faint after the injection, but quickly recovered.

Anal Fissures.

In the case of anal fissures, A.B.A., or the percaïne in oil solution, can be trusted to produce sphincteric relaxation, 5 c.cm. being the usual amount which I now inject into the sphincter. A superficial fissure will heal readily: a fissure with a sentinel tag will heal when the tag has been snipped off, but it is not possible to secure healing of a deeply undermined fissure without a drainage operation. A fissure of this last type is really a submucous pocket or a blind internal fistula, and must be drained back by a surgical operation. Injection of A.B.A. in such a case may relieve the symptoms, and will permit a thorough examination to be made, but it will not cure the condition.

The operation for drainage of a deep undermined fissure is best performed under a low spinal anaesthesia by the technique described by my colleague at St. Mark's Hospital, Mr. J. K. Hasler.³ With the patient in a sitting position 0.4 or 0.5 c.cm. of stovaine billon in saline solution is injected intradurally through any convenient lumbar interspace. The patient remains sitting for a few minutes before being placed in the lithotomy position; he is then found to have a saddle-shaped area of anaesthesia in the perineum corresponding to the distribution to the skin of the fifth, fourth, third, and sometimes the second sacral nerves. The operation is performed as follows. The sphincter is gradually stretched until five or six fingers can be admitted readily. The undermined pocket is then drained posteriorly, and the sentinel tag is excised. Instead of the usual "lozenge" or diamond-shaped incision which has hitherto been recommended, I now make an incision with square ends, which has great advantages; it gives better drainage, and renders healing of the wound in the anal canal quite inevitable (Fig. 3).

FIG. 3.—Indicating incision with square ends, recommended by the author for drainage of a deeply undermined fissure.

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ANAESTHETIC EXPLOSIONS.

BY

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Two cases of alleged explosion of chloroform vapour have recently come to my notice. Chloroform is not inflammable; in fact, it cannot be made to burn by itself. A mixture of 2 parts of chloroform with 1 part of ether, or a mixture of chloroform, alcohol, and ether will, however, burn, giving off strong acrid fumes containing chlorine.

I have tried to discover whether in favourable circumstances the vapour of chloroform can be caused to ignite, for in one case there was an explosion when an electric cautery (Cameron's "cauterodyne") was being used in the mouth. I first passed a stream of oxygen through chloroform in a Junker's bottle, and failed to ignite the vapour issuing from it. I thought that the extremely high temperature of the electric spark might in some way render the oxygenated vapour combustible, so I prepared an experiment in which chloroform was boiled in a tin canister with a stream of oxygen bubbling through. The flame of

an oxy-acetylene blowpipe was applied to the vapour, and, subsequently, a fragment of steel was kindled in the vapour with the aid of a jet of oxygen. This procedure resulted in such brilliant scintillations being given off that the heat must have been well over 4,000° C.—the temperature of the flame—and at least as high as the sparks from the electric cautery.

In both cases the chloroform vapour decomposed at once into billows of pungent and acrid black fumes; these did not catch fire, and the decomposition did not continue when the flame was removed, indicating that it was not the chloroform vapour that became ignited in the two cases. Since there is no doubt at all about the facts in the two cases of explosions at operations, I can only say that the occurrences must have been caused either by the inadvertent addition of ether, or by the burning of combustible gases arising from hot and charred tissues in such a confined space as the mouth. This is a probable explanation in the second case, and accounts, of course, for the flickering always seen when using such cauteries.

At least a hundred explosions probably occur every year with ether in this country, causing trifling or severe burns of the eyebrows, lips, and pharynx, but not extending into the lungs, because the mixture is too weak to burn there. The explosions are caused by the ignition of the richer mixture introduced into the mouth by a tube or occurring in the immediate proximity of the soaked coverings of the mask. I feel sure that two or three minutes after stopping the administration of ether the expired air contains too little of the gas to ignite.

For several years I have continually used the ether bomb apparatus, often within two or six feet of an open fire, and frequently much nearer a cautery, without any ignition taking place. The quantity of ether is too small, or too quickly diluted, although, where there is an electric radiator in the room, the pungent fumes of slow combustion can always be smelt above it within a few minutes of the vaporization of even a small quantity. An explosion was reported to have occurred from the introduction of the apparently sound lamp of an oesophagoscope into the pharynx; it must be concluded that the connexion in the lamp socket was sufficiently imperfect to cause minute sparkings, because I have found that a needle introduced into an air-ether mixture has to be at least red-hot to ignite it.

The ordinary optimum mixture of petrol vapour and air in a motor car engine is 1 in 13; in the case of alcohol it is, I believe, 1 in 14, and in that of ether the proportion is probably the same. A weaker mixture would, however, ignite, and so would a stronger, but the limits (without compression) are not wide and the ignition point is considerably raised. When a combustible gaseous mixture is raised to a certain temperature it "fires"; this is its "ignition point." The term "flash point" refers to a liquid, being that temperature at which it gives off inflammable vapour. The temperature at which a hot body introduced into an inflammable mixture will cause ignition depends partly upon the nature of the body.

In this connexion a case was recorded in the *British Medical Journal* of October 17th, 1925, where a dental warm air syringe set fire to ether vapour in the mouth of a patient and resulted in fatal haemorrhage. The nearest flame was said to have been six feet away, and the nozzle of the syringe was certainly not red-hot. In the correspondence which followed this report the possibility of catalytic action, similar to that induced by spongy platinum, was mooted, and also the chance of burning spirit being carried on to the nozzle from the lamp employed to heat it. I can find very few references to explosions.

By far the most serious fires with ether have been caused by bottles being dropped in rooms with open fires or electric radiators. Ethyl chloride will not ignite in air, but it burns with an apple-green flame on the addition of a little extra heat, and more freely with oxygen; curiously enough, however, the condensed substance burns freely with a luminous and smoky flame, which is coloured green near the liquid surface. The explanation of this may be that the jet of gas becomes too rapidly diluted with air, partly owing to the velocity at which it passes out of the nozzle.

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