became so unsatisfactory that the anaesthetic was discontinued, and the incision rapidly closed. Eight minutes after this artificial respiration became necessary, and after two hours the heart stopped. *Post mortem* there was great dilatation of the ventricles of the brain, and the existence of basal meningitis prevented the opening of the cranial cavity relieving the pressure.

Case x presented some interesting features. The anaesthetist was using the apparatus for the first time, and

Fig. 9.—Case x. Man aged 36. (Ventral hernia, Mr. Clayton-Greene.) Description in text.

found some difficulty in translating his knowledge of anaesthesia into the percentages given by the machine. Induction was too slow (it was before the standard rate had been adopted), and when the patient was moved to the theatre a mask was placed over his face in transit. This evidently gave a higher percentage than was expected, and the result was that after a latent period of one minute the breathing became embarrassed, and artificial respiration had to be performed, ether being used to keep up anaesthesia. The patient came round in a very satisfactory manner, and then was given 1 per cent., and later 0.5 per cent. This latter amount, as can be seen from the other curves, was much too little for the stage of anaesthesia, and the patient began to recover consciousness; 3 per cent. was then hastily given, then 2 per cent., and finally the machine was abandoned and the mask used to finish the operation. The difficulties met with were evidently the result of the use of an unfamiliar method, but one valuable piece of information was disclosed, namely, that the effect of an overdose is not immediately seen, requiring about a minute to become evident.

CONCLUSIONS.

There are many things observed from time to time in this series that fall under the class of unmeasurable phenomena. It is hoped that future work may remove at least some of these into the category of accurate measurements, when they can be considered with more certainty than is now possible. Particularly would one wish that numerical values could be assigned to the individual variations and to the post-chloroform effects that are now so meagrely dealt with. But, from the facts recorded above, the following points may be noted as reasonably certain.

1. Anaesthesia with known percentages of vapour is in man at least as easy and certain as by the ordinary method, even when the latter is carried out with the greatest skill.

2. If it is desired to induce anaesthesia in from eight to ten minutes, the ordinary adult must be given percentages rising to 2 per cent. in two minutes, then to 2½ per cent. in three minutes. This amount usually will suffice. There is no objection to raising the percentage to 3 per cent. in five minutes (*not sooner*) if it is required, *provided due care is exercised*.

3. When induction is completed, 2 per cent. is usually ample, gradual diminution to 1.5 per cent. and 1 following in about twenty to twenty-five minutes, and the anaesthesia can be maintained in this way much more easily and regularly than by the open method.

4. Children generally require less than the above amounts, 2 per cent. for the induction and 1.5 per cent. for the later stages. Some thin adults will also require less than the average, some fat, muscular, or alcoholic adults more, but the data are not sufficient to furnish more precise rules.

5. From the above it is evident that the general features of anaesthesia are the same in man as in the other mammalia. Minor points of difference are seen in the greater individual variations and in the percentages required, 2½ per cent. in man being approximately equal to

2 per cent. in the cat. When due allowance is made for these differences, it is legitimate to apply the conclusions deduced from experiments on animals to the human subject. When it is remembered how conclusively Waller has shown the much greater safety of a known percentage in animals, the importance of the inference can be seen.

Finally, it is again my pleasant duty to convey my thanks to my colleagues at St. Mary's Hospital, who have in every way given me the kindest assistance. Particularly are my acknowledgements due to Dr. Blumfeld, Dr. Collum, and Dr. Beaumont, to whom the credit of the practical success of the method largely belongs.

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ATROPHIC RHINITIS COMPLICATED BY MASTOID ABSCESS AND EXTRA-DURAL ABSCESS.

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So far as I am aware atrophic rhinitis is not a common cause of mastoid suppuration, particularly in the absence of signs of middle-ear suppuration.

Mrs. M. A. S., aged 30 years, came to me on November 5th, 1908, complaining of pain over the mastoid and frontal areas. She had had atrophic rhinitis for twelve years, and had been treated in various ways without any benefit. The right ear had been partially deaf for four years, the deafness coming on gradually. She had had no pain in the ear until lately, and never had otorrhoea according to her own, her mother's, and her husband's testimony. No history or signs of syphilis or tubercle.

On November 5th she complained of intense pain over the right mastoid and frontal areas, tenderness over the same and on pressure on the right tragus. The post-auricular gland was enlarged, but as her head was infested with pediculi great weight was not attached to this, particularly as she said it had been in much the same condition for months. Temperature 103.5°, pulse 120. No rigors or signs of intracranial mischief. Both nostrils were wide and partly covered with stinking crusts. The left inferior turbinal had been removed. The septum was deflected to the right. By posterior rhinoscopy the Eustachian orifices were seen to be dilated and the walls and roof of the naso-pharynx dry, glazed, and covered with adherent crusts. On examining the right ear no perforation could be seen in the membrane, which was opaque, thick, white, and immobile with Siegle's speculum. Rinne's test was negative; the watch could not be heard at all; Weber's test was referred to the right side. There was no redness, swelling, or oedema over the mastoid process nor was the auricle displaced.

Leeches were applied in front of and behind the auricle, followed by large fomentations. Drops of a solution of cocaine and ac. carbolic. (āā gr. v in glycerine (j)) were instilled into the ear and a purgative given.

The patient felt much relieved, had a fairly good night, and next morning (November 6th) the temperature had fallen to normal, the pulse to 96. She still, however, had a great deal of pain and tenderness. The same night she became much worse, and a swelling became apparent above and behind the auricle, displacing it downwards and outwards. Operation was advised, and early next morning (November 7th) the mastoid antrum was opened, giving exit to over a drachm of thick yellow pus. Considerable bone destruction had gone on posteriorly, therefore the lateral sinus was exposed, and although there were some granulations on its external aspect, it was not thrombosed. The mastoid cells were thoroughly opened up, the mastoid antrum, almost as large as a Barcelona nut, was curetted, the patency of the aditus assured, and the cavity packed with gauze. The temperature, taken immediately before operating, was 100° F.; after the operation it was normal.

From this time until November 13th, she did well generally, except that the pulse was rapid (see chart), but still complained of intermittent frontal pain and tenderness. On this date facial paralysis of the right side was noticed, but it was by no means marked. I considered it might be due to inflammatory thickening near the stylo-mastoid foramen in the depth of other symptoms, and could not decide that the head pain was due to intracranial mischief, as she had no other signs of such trouble, such as rigors, vomiting, optic neuritis, spasms of limbs, vertigo, or the pulse or temperature of brain or meningeal disease, although the two last symptoms are known to be uncertain quantities. Her mental condition was clear and alert, but she was very restless.

On November 14th the facial paralysis was worse, and at night the pain was intense, and had extended over the right side of the head. There was tenderness on pressure over the squamous portion of temporal bone. The temperature was normal, and the pulse 110. There was no vertigo or other signs of involvement of the intracranial portion of the auditory

nerve, therefore it was concluded that the paralysis was due to pressure on the facial nerve in the middle ear, where possibly the bony wall of the aqueduct of Fallopius was deficient. Extradural abscess over the tegmen tympani was considered as a probable solution of the cause of pain, and on November 15th the previous operation was extended and the complete mastoid operation performed. The middle ear was filled with granulations, and no trace of the ossicles could be seen. The cavity in the bone was enlarged by cutting upwards and the middle fossa opened. The dura was covered with granulations but did not bulge. A probe passed under the dura in a forward and inward direction entered an abscess cavity $\frac{3}{4}$ in. from the opening, and about $1\frac{1}{2}$ drachms of thick, yellow, foul pus escaped. Good drainage was secured and the wound gently packed with gauze and drained posteriorly. The mastoid wound was also packed and drained through the auricle, from which a flap was cut. The temperature and pulse before operation were 99° and 100 respectively; after it they fell to 98.2° and 72. The patient became exceedingly restless in a few hours, having to be held down in bed; $\frac{1}{4}$ grain of morphine was given hypodermically and secured sound sleep for eight and a half hours.

November 16th. Much quieter, but still restless; slow cerebration; no pain in head; pupils equal and active; some stiff-

O, Operation. D, Death.

ness of back of neck; both knee-jerks very active. No other signs. Basal septic meningitis diagnosed and bad prognosis given; pulse 84, temperature 97.9°; facial paralysis much improved.

November 17th. Pulse 104, temperature 100°. Drowsy, restless, and irritable, reminding one very much of the picture of the first stage of cerebral irritation. Has vomited a little (one mouthful) once without retching. No pupil disturbance or other cranial symptom, except those present yesterday. Facial paralysis scarcely perceptible; taking nourishment fairly well.

November 18th. Cannot be roused. Conjunctiva insensitive. No squint, pupils equal. Pulse 132, respirations 46, no stertor.

Later. Pulse 144, respirations 48 and of Cheyne-Stokes type at intervals, temperature 100° F. Perfectly quiet, with an occasional gentle movement of the hands and arms, not at all convulsive in character.

November 19th. Died quietly at 8.30 a.m.; no autopsy obtained.

The infective process in this case had evidently passed from the naso-pharynx to the middle ear and destroyed the ossicles, and had also invaded the antrum and gradually rarefied the mastoid bone. No symptoms occurred, however, until the aditus became blocked with granulations or swollen mucous membrane, as until then a free road was available into the naso-pharynx for the products of inflammation. When the block occurred the symptoms appeared—the result of tension.

When the signs and symptoms set down as characteristic of intracranial disease, such as rigors, vomiting, optic neuritis, pupil changes, convulsions, hydrocephalic cry, etc., are absent, one is liable to be lulled into a false sense of security. Until the last two days of life of the patient whose case has been described, *restlessness* was the most noteworthy symptom. It was characterized chiefly by the continual efforts of the patient to get up on her hands and knees; she would stay in that position for a moment and lie down again. This sequence would sometimes occur five times in as many minutes.

It would undoubtedly have been better in this case to have explored the middle fossa at the time of the first operation, as well as the lateral sinus. This procedure would not have unduly prolonged the operation, and much valuable time would have been saved.

It has since appeared to me that a more effective route for draining the floor of the middle fossa might be obtained where the complete mastoid operation is done, if the infero-lateral opening usually made was continued inwards and forwards by removing the roofs of the external meatus, middle ear, attic, and antrum. The external semicircular canal would not be in danger if a bent probe were kept over it, and the others could be safeguarded by cutting, as before noted, inwards and forwards. (See figure.)

In the above diagram 1 corresponds to area over glenoid cavity; 2, to area over external auditory meatus; 3, to area over mastoid antrum; 4, to area over middle ear; E T, to area over Eustachian tube; B, to area over internal ear; 5, to position of lateral sinus. The area outlined in coarse is that to be removed.

A CASE OF OEDEMA WITH RESOLUTION BY URINARY CRISIS.

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THIS case of what may be called a urinary crisis in a man with chronic parenchymatous nephritis is worth recording, if only on account of the phenomenal loss of 4 st. 3 lb. in four days coincident with the disappearance of oedema.

The patient, a man 27 years of age, got wet through twelve days before admission; nine days before admission he had abdominal pain, diarrhoea, sickness, and malaise; a few days later oedema was noted, and one day before admission phlebitis of the left thigh appeared. Eight years ago he had had an attack of kidney disease with oedema. On admission on July 11th, 1907, he had oedema of the legs and lower part of the abdomen, and thrombo-phlebitis of the left femoral vein. The urine—specific gravity 1028—contained a considerable amount of albumin, and showed diminution of chlorides, but there were no casts or blood. There was no hypertrophy of the heart, and the blood pressure was 100 mm. of mercury.

The phlebitis soon subsided, but the general oedema increased. On July 26th he weighed 11 st. 12½ lb.; on September 9th, 12 st. 1 lb.; on September 14th, 12 st. 10 lb.; on October 10th, 13 st. 11½ lb.; on October 16th, 14 st.; and on October 23rd, 15 st. 7½ lb. The average amount of urine passed was about 30 oz., and the intake of fluid was restricted for most of the time to 60 oz. From the time of his admission various means of reducing the oedema were employed, but without effect. He had successively and in different combinations theocin, caffeine, hot air baths, pil. digitalis co., puncture of the legs. For a period of four weeks, when in the summer Dr. Spriggs looked after the patient, the amount of fluid taken was not restricted. These measures did not have any beneficial effect. On October 5th he developed an attack of cellulitis of the left groin and lower part of the abdomen on the left side, which soon passed off. On October 16th his weight, as already stated, was 14 st.; it then slowly decreased, and a week later (on October 23rd), when it was 13 st. 7½ lb., the amount of urine suddenly increased from 48 oz. in twenty-four hours to 98 oz., and on the succeeding days was 112, 166, 180, 160, 204, 112, 138 oz. The hot-air baths were then stopped (on October 30th), and the amount then fell to 84 and 60 oz. on October 31st and November 1st; by this time the oedema had completely disappeared. On October 31st he weighed 9 st. 4 lb., having lost 4 st. 3 lb. since October 27th, and, after losing 2 lb. more, his weight increased to 9 st. 6 lb., when he left the hospital free from oedema on November 12th. After the sudden disappearance of oedema he