Paris, 1846. ⁹ Loc. cit., Section III, p. 290. ¹⁰ *Med. Chir. Trans.* ¹¹ Loc. cit., p. 30. ¹² *Anatomy for Artists.* ¹³ *Anatomy for Art Students*, by Professor Arthur Thomson, 2nd edition, 1899, pp. 37, 121. ¹⁴ *Topographische Anatomie*, Zweiter Band, 1895, p. 185. ¹⁵ *Curvature of the Spine*, 2nd edition, p. 41. ¹⁶ *Anatomie Artistique*, 1890. ¹⁷ Loc. cit., p. 171. ¹⁸ Loc. cit., Part VII, Book I, p. 339. ¹⁹ BRITISH MEDICAL JOURNAL, March 5th, 1892. ²⁰ BRITISH MEDICAL JOURNAL, March 5th, 1892.

THE RAPID ESTIMATION OF THE QUANTITY OF CHLOROFORM VAPOUR PRESENT IN MIXTURES OF CHLOROFORM VAPOUR AND AIR.

BY
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A METHOD for the rapid estimation of CHCl_3 vapour in mixtures of CHCl_3 and air by means of the alteration of pressure produced by absorption of the CHCl_3 vapour by olive oil is described in the BRITISH MEDICAL JOURNAL of July 12th, 1902, and was demonstrated last July to the Physiological Society.¹

It was then stated that the method is applicable to mixtures of chloroform and air between 2 and 10 per 100, and the statement was confirmed by Professor Dunstan for amounts between 3 and 12 per 100.²

The original purpose of the present observations was to test the method for mixtures below 2 per 100. The actual determinations have been made in this laboratory by Dr. Victor Geets, of Brussels, who has, in prosecuting the inquiry, adopted a very simple method of control, which is in itself a very satisfactory laboratory method for the quantitative estimation of the percentage of chloroform vapour in a mixture of chloroform and air.

This method consists in the direct weighing on a chemical balance of a flask of known capacity (*circa* 500 c.cm.), (a) filled with air, (b) filled with the mixture of chloroform and air.

The adequacy of this datum as regards an estimation of percentage will be made clear by the following considerations: 500 c.cm. of a 1 to 10 per 100 mixture of chloroform and air implies 5 to 50 c.cm. of CHCl_3 vapour, weighing (on the assumption that 1 gram of liquid CHCl_3 = 200 c.cm. CHCl_3 vapour) 25 to 250 mg. *minus* the weight of the volume of air displaced by the CHCl_3 vapour.

The weight of a litre of CHCl_3 vapour at 0° C.

$$= \frac{12 + 1 + 106}{2} \times 0.0895 = 5.325 \text{ grams.}$$

Ditto, of a litre of air = 1.293 grams.

So that a litre of chloroform vapour is 4.032 grams heavier than a litre of air. Or each c.cm. of chloroform vapour replacing 1 c.cm. of air gives an increased weight of 4.032 mg.

Taking, then, 4 mg. as the additional weight due to the presence of each 1 c.cm. CHCl_3 in the mixture of air and chloroform (corrections for temperature and pressure not included), we have: In a 500 c.cm. flask each 1 per cent. (= 5 c.cm.) giving an increment of 0.020 gram.; and in a 250 c.cm. flask each 1 per cent. (= 2.5 c.cm.) giving an increment of 0.010 gram.

For practical purposes we can avoid calculation and estimate the percentage of CHCl_3 vapour by a direct reading of the difference of weight of a 250 c.cm. flask, (a) filled with air, (b) filled with the mixture of chloroform and air to be titrated. The excess of the latter, counted in centigrams, gives the percentage of the mixture:

Thus, an excess of 0.010 gram = 1.0 per 100

" " 0.050 " = 5.0 "

" " 0.027 " = 2.7 "

The weighing is easily made within the error of ± 1 mg., equivalent to an absolute error of $\pm \frac{1}{10}$ of 1 per cent., and to a relative error of ± 10 per cent. to ± 2 per cent. for absolute percentages between 1 and 5.

Both the manometric and the gravimetric methods are rapid and mutually control each other, giving estimates in reliable steps of at least 0.1 per cent. The manometric estimate is adapted to hospital use in the absence of a chemical balance; the gravimetric method is more rapid and more exact.

The chief practical advantages of a rapid method of estimating chloroform in air differ according as the chloroform question is viewed from the experimental or from the clinical standpoint. Experimentally it is important to possess a ready

means of frequently gauging the percentage of chloroform administered and exhaled. Clinically it is important to possess a ready means of familiarizing students with the smell of mixtures of known percentage. It is an easy matter for a laboratory expert to distinguish by smell whether there is more or less than 1 or 2 per 100 of chloroform vapour in a given atmosphere. Until it is possible to rapidly verify the percentage and to offer to inhalation by his students mixtures of known percentage, even the most experienced teacher of anaesthetics is not entitled to use clear arithmetical language, and to say that chloroform for the safe induction of anaesthesia in ordinary cases should be maintained between the limits of 1 and 2 per 100. But with a rapid method of estimation at hand, this all-important fact can be taught by smell, which after all is, and must remain, the most valuable indicator immediately at command of the practical anaesthetist.

The following table gives the comparative results of a preliminary series of parallel estimates by oil-absorption and by weighing:

Weight Readings and Calculations by Dr. Geets. Manometer Readings by Mr. Shapiro.

Percentage by Manometer.	Percentage by Weight.	Percentage by Manometer.	Percentage by Weight.
5.20	5.95	1.50	1.49
4.50	5.08	1.50	1.49
4.50	4.96	1.50	1.49
3.50	3.72	1.50	1.49
3.50	3.72	1.50	1.44
2.00	3.47	1.50	1.44
2.85	3.10	1.50	1.39
2.80	2.98	1.60	1.36
2.80	2.98	1.50	1.36
2.80	2.73	1.50	1.34
2.15	2.48	—	1.30
2.30	2.28	1.60	1.30
2.60	2.11	1.60	1.30
2.00	2.08	1.45	1.29
2.00	1.98	1.45	1.26
1.50	1.86	1.48	1.12
1.50	1.61	1.30	1.12
1.50	1.59	1.30	0.99
1.50	1.54	—	—

The individual discrepancies in the two series of this table are such as might be expected from experimental errors occurring in a first trial of two new methods by two independent observers. The error has been relatively more considerable at the lower percentages (below 2 per cent.) but is also considerable at the higher percentages. It is more probably attributable to imperfect graduation of the manometer than to incorrect weighing. To determine this point will, however, require further observations.

Corrections for temperature and pressure are of secondary importance in determinations of the present character. The temperature error for $+15^\circ$ is within $\frac{1}{2}\%$ of the nominal percentage, and can be practically eliminated by taking flasks = 260 c.cm capacity, that is, the usual 250 c.cm. flask + 10 c.cm. of its neck above the mark.

The usual corrections for T and H are, however, readily made, in accordance with the following formula, applicable to any flask capacity:

$$\log P = 1.8391 + \log m - \log v + \log T - \log H$$

P being the required percentage, m the observed increment of weight in mgs., v the flask capacity in c.cms., T the absolute temperature, H the barometric pressure, and 1.8391 a constant derived from the density of chloroform vapour at 0° (273° T) and 760 mm. Hg.

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THE PHYSIOLOGICAL ACTION OF ETHYL BROMIDE AND OF SOMNOFORM.

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THE object of the experiments described below was the investigation of a new anaesthetic sold commercially under the name of "somnoform." This is a mixture containing ethyl chloride 65 per cent., methyl chloride 30 per cent., ethyl bromide 5 per cent. It has been largely used in France for producing anaesthesia for minor operations, and the

manufacturers of it claim that it is very readily administered, the anaesthesia is produced very rapidly, is unattended with danger, and that there are no ill after-effects. There is no published account of the physiological action of such a mixture of drugs, and it seemed desirable to investigate their action both alone and in various proportions with a view to discovering, if possible, a reliable and safe anaesthetic for general use. This paper must be regarded as a preliminary account, for at present my experiments on ethyl and methyl chlorides are not complete.

Historical.

Ethyl bromide was first employed as an anaesthetic by Nunnely¹ in 1849. Its use has been rather intermittent, but at the present time it is somewhat extensively used in America and on the Continent as a general anaesthetic, chiefly, however, for minor operations, such as those in the throat. For longer operations it has been employed as a preliminary to ether by Williams,² and to chloroform by Hartmann and Bourbon.³

The chief characteristic of bromide of ethyl as an anaesthetic is its strikingly rapid yet transient action. A suitable dose in about a minute produces unconsciousness lasting on an average one or two minutes, from which the patient awakes and regains almost at once his normal state. (Quoted from A. Brown Kelly. Bromide of Ethyl Anaesthesia in Operations in the Throat, BRITISH MEDICAL JOURNAL, 1902, vol. ii, p. 588.)

Ginsburg⁴ gives the best account of the physiological action of ethyl bromide, his experiments being performed on rabbits and dogs. He finds that with small doses, yet sufficient to produce anaesthesia, the blood pressure is not lowered, the pulse becoming more rapid, and not irregular. With larger doses there is a fall in blood pressure, an irregular, rapid pulse, and a quickening of respiration. Death is produced by cessation of respiration, which precedes that of the heart. He ascribes the quickening of the heart to stimulation of automatic cardiac centres or of peripheral accelerator nerves. The fall in blood pressure with large doses is due, according to Ginsburg, to paralysis of peripheral vasomotor nerves. He states that ethyl bromide has no effect on the vagus, vasodilator centres, or peripheral vasodilator nerves. Thornton and Meixell⁵ obtained similar results, the chief source of danger being stoppage of respiration. Ethyl chloride has been used locally as an anaesthetic in dentistry, and also as a general anaesthetic for minor operations (W. J. McCardie)⁶. Its physiological action seems to be similar to that of ether, but it is liable to produce death by paralysis of respiration⁷.

As far as I am aware methyl chloride has not been used as a general anaesthetic.

Experimental.

My own experiments are 20 in number, 12 being on rabbits, 3 on cats, and 5 on pithed frogs.

The animal was first anaesthetised, by ether in the case of rabbits, and chloroform followed by the A.C.E. mixture in the case of cats. The effect of the somnoform or ethyl bromide was tried whilst the animal was only lightly under the influence of the preliminary anaesthetic. Tracheotomy

was performed in all cases, and the anaesthetic administered by the tracheal tube.

The constituents of somnoform are all highly volatile, the boiling points being: Methyl chloride, -23°C .; ethyl chloride, 12°C .; ethyl bromide, 40.7°C . For this reason it is difficult to regulate nicely the amount that it is desired to administer.

The drug is sold in hermetically-sealed bottles that can be fitted with a valved nozzle. I generally allowed about 5 c.cm. to boil over into a bottle containing some cotton wool, and into this bottle I introduced one limb of the Y-shaped tracheal tube that was used in all my experiments. The other end of the tracheal tube was left open for the first few seconds, being closed when I desired to give the animal a larger dose. The objection to this method of administration is that the air reaching the lungs contains a high percentage of the drug. The advantage is that it allows the various effects to be well seen, owing to their rapid development.

I will now proceed to describe the results I have obtained, dealing separately with the effects observed on the several functions of the body.

Effect on Respiration.

The effect of somnoform on respiration was studied on 6 rabbits and 2 cats.

Methods.

On Rabbits.—A record was taken of the contractions of the slip of the diaphragm passing to the xiphisternum and isolated after the method of Head.⁷

On Cats.—The movements of the sternum were recorded by means of two tambours.

Somnoform has an immediate and pronounced stimulating effect on the respiratory centre, which is manifested in three ways: 1. An increase in the size of contraction; 2. an increase in the rate of contraction; 3. an increase in the tone of the diaphragm.

With large doses this stimulation of the centre is so great that the diaphragm is finally brought to a standstill, remaining in a state of strong tonic contraction until death ensues, the heart still beating strongly.

After section of the vagi inhalation of the drug shows its effect on the centre by an increased height of contraction, the rate and tone being not so markedly affected as in an animal with intact vagi. Also after section of the vagi it is difficult, in fact almost impossible, to cause cessation of respiration with the diaphragm in a state of tonic contraction. Under these circumstances the diaphragm becomes relaxed owing to an apparent exhaustion of the respiratory centre.

An important point in connexion with the use of somnoform as an anaesthetic is that this cessation of respiration does not take place until some considerable time after the causation of complete insensibility. Also that with all but very large doses it is very easy to re-establish breathing by artificial respiration. This is probably due to the fact that the heart is so little impaired by even prolonged inhalation of the drug, so the medullary centres do not become anaemic.

The effect of somnoform on respiration is shown in Figs. 1, 2, and 3.

(a) Somnoform.

Fig. 1.—The effect of somnoform on the contractions of the diaphragm and on the carotid blood pressure of a rabbit. Contraction of diaphragm slip caused upward movement of lever. At point indicated somnoform was administered by tracheal tube, the administration lasting for 25 seconds. The blood pressure rose very slightly during the administration, falling to its previous height after discontinuing somnoform. Respiration was almost immediately affected, the contractions of the diaphragm becoming larger and more rapid, the tone of the diaphragm also increasing considerably. After discontinuing the drug the contractions decreased in size and rapidity, the tone, however, remaining high for over 3 minutes, when it gradually fell to its previous state (8 minutes).

Fig. 2.—The effect of somnoform on the carotid blood pressure and on the contractions of the diaphragm of a rabbit after ether narcosis. Both vagi had been previously cut high up in the neck. Blood pressure fell slightly and slowly during the inhalation of somnoform, rising rapidly afterwards, the heart becoming irregular in its action. At the point marked "a" the heart beats were suddenly reduced to exactly half their previous number, this effect passing off with equal suddenness at the point marked "b." The respiratory contractions were increased in size at the commencement, but returned to their original magnitude before the drug was discontinued. There was no increase in rate or tone.

Fig. 3.—The effect of somnoform on the carotid blood pressure and on the respiratory movements of a cat, anaesthetised by chloroform. Both vagi had been cut high up in the neck. The animal had previously had a rather large dose of chloroform, and its blood pressure had not fully recovered when somnoform inhalation began at the point shown. The blood pressure fell very slightly during the inhalation of somnoform. The drum was stopped for two minutes, during which time the pressure rose considerably. The peripheral end of the left vagus was stimulated at A during the inhalation, at A' just after, and at B, C and D some time after stopping the inhalation, in all cases with the coil at 7 c.cm. At A and A' no slowing of the heart or fall of pressure took place. At B, C and D there was a distinct slowing of the heart, the pressure rising at B and falling at C and D. The experiment thus shows that somnoform produces a temporary paralysis of the peripheral endings of the vagus. The respirations became faster and shallower.

Fig. 4.—Effect of somnoform on the carotid blood pressure of a rabbit. Ether was given as a preliminary anaesthetic. Both vagi divided in the neck. At V8 the peripheral end of the right vagus was stimulated with the secondary coil at 8 cm. It can be seen that during and just after the inhalation of somnoform stimulation of the vagus is without effect on the heart.

Effect on the Heart.

Methods.—1. The examination of the blood-pressure tracings. 2. Direct records were taken of the contraction of the auricle and ventricle in rabbits. This was done in five rabbits. In all cases the thorax was opened and artificial respiration maintained either by the hand bellows or by the apparatus made by the Cambridge Scientific Instrument Company. In opening the thorax care was taken to ligature the intercostal and internal mammary arteries of both sides. The loss of blood was usually small. The pericardium was opened by a median incision, and the cut edges stitched to the parietes of the chest. In this way the movements of the

heart were but little affected by the inflations of the lung. In four cases the contractions were recorded by levers attached to a thread passing over a pulley, the further end of the thread being fastened to a small hook inserted in the ventricle and auricle. In two cases I chose the left auricle and ventricle, and in the other two the right. 3. In the fifth case I recorded the contractions by means of the ingenious myocardiograph described by Roy and Adami.⁸ The results obtained were the same as with the simpler method.

The effects of somnoform are shown on: (1) The rhythm; (2) the strength of contraction; (3) the conduction.

1. As regards the rhythm, I find that the rate is increased

Fig. 5. The effect of ethyl bromide on the endings of the vagus in a pithed frog. Normal saline saturated with ethyl bromide was dropped on the heart after the first tracing had been taken.

with small doses both with intact and cut vagi. With larger doses a slowing is produced in both cases.

This increase of rate with intact vagi is due to all or some of the three following causes: (1) The paralysis of the vagal nerve endings; (2) a direct stimulation of the cardio-accelerator and augmentor fibres; (3) a direct action on the heart itself. I describe later experiments that show conclusively that the preganglionic endings in the cardiac ganglia are paralysed or depressed by somnoform. This would to a certain extent account for the quickening of the heart with the intact vagi. I have frequently observed an acceleration of the heart during administration of the drug, even after section of the vagi. This is probably due to a stimulation of the cardiac accelerator centre or nerves.

2. The strength of contraction is always decreased, both before and after section of the vagi. This is particularly noticeable with large doses. The effect is produced by ethyl bromide alone, as well as by somnoform.

3. In Fig. 2 I show a blood-pressure tracing from a rabbit, in which the oscillations were suddenly reduced to half their number 30 seconds after somnoform was given. This lasted until 15 seconds after the somnoform was removed. I have seen a similar effect in three rabbits. In all cases the vagi were cut, and the only explanation I can offer is that it

is due to the ventricle failing to contract owing to a depression of conduction in the auriculo-ventricular ring. I have not been fortunate enough to obtain the effect with the heart exposed. A similar result has been described by Gaskell and Shore during chloroform administration. With ethyl bromide alone I have observed a quickening of the heart with intact vagi, and also once with cut vagi. Ginsburg, who failed to notice the depression of the vagal nerve endings, ascribed the acceleration of the heart under ethyl bromide as entirely due to stimulation of the accelerators, either peripherally or centrally.

Effect on the Blood Pressure.

With small doses of somnoform the blood pressure is not usually affected to any great extent. Any alteration that does occur is generally in the upward direction. With large doses the pressure remains high for a good length of time, and then gradually falls. This is especially the case if the vagi are intact. After section of the vagi a rise has never occurred in my experiments. Usually a gradual yet distinct fall in pressure takes place.

A very common effect is that during the administration of the drug the blood pressure remains unaltered. After

Fig. 6.—Record of the contractions of the ventricle and auricle of a rabbit, with effect of somnoform on peripheral ends of vagus. (Ether was given by tracheal tube during the preliminary dissection.) Stimulation of cut end of left vagus (coil at 13) at A produced stoppage of heart. Somnoform was inhaled for 25 seconds, the drum started, and the vagus stimulated again at B and C without producing any effect. The drum was stopped for one minute (somnoform having been discontinued). On stimulating at D a distinct inhibitory effect was produced. Stoppage of the heart was produced after the expiration of another half minute (E). Auricular tracing above.

cessation of the inhalation it rises considerably. I have not determined the cause of this after-rise.

The rise in pressure seen in an animal with intact vagi is due, I think, to the removal of the inhibitory vagal impulses, the rise being usually accompanied by an acceleration of the heart. The subsequent fall is due to the direct action of the drug on the heart.

Effect on the Vagal Nerve Endings in the Heart.

I find that during the inhalation of somnoform or of ethyl bromide, the endings of the vagus in the ganglia of the heart are paralysed. I have observed this in 2 cats and 7 rabbits, in every case in fact in which I have looked for such an effect.

In the frog I have found that the post-ganglionic endings in the heart itself are not paralysed by ethyl bromide, stimulation of the sino-auricular junction by a weak interrupted current producing marked inhibition, whilst stimulation of the trunk of the vagus is entirely without effect. This is shown in Fig. 5.

Effect on the Cardio-inhibitory Centre.

Owing to the depression of the vagal nerve endings I have been unable to ascertain the effect of the drug on the cardiac centre in the medulla. And owing to this peripheral depression it seemed unnecessary to investigate this point, for any impulses that would be originated in the medulla could not affect the heart's action.

Effect on the Vasomotor Functions.

I have performed two experiments on cats with an intestinal plethysmograph to ascertain the effect on the blood vessels, using somnoform and ethyl bromide. I was quite unable to detect any alteration. The intestinal volume increased with a rise in blood pressure, and decreased with a fall. I found this to be the case even with a very large dose, and am therefore unable to agree with Ginsberg, who states that ethyl bromide paralyses the peripheral vaso-constrictor fibres.

Effect on the Skeletal Muscles.

A striking effect of the drug is the peculiar rhythmic movements of the limbs, jaws, and tail that nearly always supervene with somnoform and ethyl bromide inhalation. The muscles are quite firm, even during deep coma. The struggling movements occur when it is quite impossible to obtain the corneal reflex, and are due to an excitation of the cord or higher centres. They cease immediately on section of the motor nerves to the parts.

Effect on the Non-skeletal Muscle.

In the case of the bladder I have observed a most distinct and rapid rise of pressure in a cat that was being experimented on by Mr. T. R. Elliott, of Trinity College, Cambridge. Since the increase in pressure could only be obtained when the pressure in the bladder was high, we concluded that it was due to a direct effect on the musculature of the viscus.

Effect on the Pupil.

It is difficult to estimate the action on the pupil owing to the effect of the previous anaesthetic. Whenever the pupil was small under the influence of ether, somnoform caused a dilatation, passing into a constriction with large doses.

Conclusions.

The chief indications that my experiments afford as to the use of somnoform as an anaesthetic are:

1. The chief danger being a paralysis of respiration; careful watch must be kept over the respiratory movements.
2. Owing to the depression or paralysis of the peripheral endings of the cardio-inhibitory fibres, and the comparative slight action on the heart itself, there is no danger of heart failure provided respiration has not ceased.
3. After cessation of the respiratory movements it is easy to restore the animal by artificial respiration.

I am of opinion that the paralysis of the cardio-inhibitory fibres by ethyl bromide renders that drug a very valuable anaesthetic. Embley⁹ has recently shown that the sudden death that not infrequently occurs in chloroform anaesthesia is due to inhibition of the heart through stimulation of the cardio-inhibitory centre. This is usually only seen in the early stages of chloroform administration, the danger passing off when the medullary centres are exhausted.

I venture to suggest that a valuable anaesthetic for pro-

longed operations could possibly be obtained by combining ethyl bromide with chloroform. During anaesthesia produced by ethyl bromide the vagi are paralysed; chloroform would then be deprived of one of its great dangers. It might be possible to get well past the stage of excitation into that of depression of the medulla before ceasing to give ethyl bromide and maintaining anaesthesia by chloroform alone, ethyl bromide being discontinued owing to its paralytic effect on respiration. I hope shortly to give an account of experiments I am performing to test this possibility.

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THE INFLUENCE OF ETHER ADMINISTRATION ON NITROGENOUS METABOLISM.

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THE question upon which the following research was made has already received a certain amount of attention. In the larger number of cases chloroform has been the anaesthetic employed, but since my own observations have been conducted with ether as the anaesthetic that work does not strictly come within the scope of the present paper.

It may be incidentally remarked, however, that the results of Terrier¹ (whose work on the nitrogen excretion of rabbits and man after chloroform is careful and exhaustive) bear a general resemblance to the results to be detailed below.

Thompson,² working with the human subject, found that ether anaesthesia produces diuresis and an increase in the total nitrogen and urea excretions.

Haig³ found, under similar circumstances, that "the rise of urea was enormous."

I have been unable to find any literature dealing with the excretion of uric acid and xanthin bodies under similar circumstances. Previous writers have for the most part been contented with estimating variations in urea, few of them entering even into the question of total nitrogen excretion.

METHODS.

The patients whose urine was investigated were in the wards of the Westminster Hospital. It will be seen that in most cases the patient were young adults suffering from surgical ailments which did not greatly interfere with their general health, and whose kidneys were, with one exception, absolutely healthy.

Below are given short details of the cases themselves:

CASE I.—J. G., female, aged 17. Laparotomy for tuberculous peritonitis. Temperature raised irregularly. Milk diet before and after the operation.

CASE II.—Mrs. K., aged 50. Removal of deltoid bursa. Fish before operation; milk to twenty-four hours after, then fish. No temperature.

CASE III.—R. T., male, aged 18. Radical cure of varicocele. Temperature to 100° on third day after operation, then normal. Diet ordinary before, milk to third day after, then fish.

CASE IV.—T. G., male, aged 18. Radical cure inguinal hernia. Temperature 99.8° third day after, then normal. Ordinary diet before, water to twelve hours after, then milk.

CASE V.—S. M., male, soldier, aged 24. Bilharzia. No cutting operation, only cystoscopy. No temperature. Ordinary diet before anaesthesia, and from twelve hours after. The complete table of estimations of this case is quoted in Tables I and II.

CASE VI.—K. W., female, aged 23. Tuberculous abscess of breast. No temperature. Ordinary diet before; water twelve hours after, then milk.

CASE VII.—T. H., male, aged 28. Wiring of fracture. No temperature. Ordinary diet before; milk and beef-tea to third day, then fish.

CASE VIII.—C. C., male, aged 25. Radical cure of inguinal hernia. Ordinary diet before; milk and beef-tea after operation.

CASE IX.—R. T., male, aged 18. Removal of sequestrum. No temperature. Ordinary diet before operation; fish after.

CASE X.—A. S., male, aged 22. Removal of sequestrum. No temperature. Ordinary diet before; milk and beef-tea afterwards.

CASE XI.—T. S., male, aged 15. Removal of tumour from sphenoid. Much haemorrhage. Temperature 100° second day, after then declined. Ordinary diet before operation; milk and beef-tea after.

CASE XII.—Mrs. P., female, aged 30. Radical cure femoral hernia. No temperature. Fish diet to day before, then milk.

CASE XIII.—Mrs. H., female, aged 25. Dilatation of cervix uteri under