definite pallor of his discs, with little or no visual impairment. There was nystagmus, but no pupillary change, and, although he walked rather feebly, the knee-jerks were present and active, and there was no change in the plantar response. He spoke slowly and with slightly slurred articulation. I regarded the case as one of disseminated sclerosis. He continued in the same condition for five years, when he developed mental symptoms of the grandiose character so often met with in general paralysis; he became very fat and then wasted and died, the mental condition becoming one of profound dementia before he died. In this case, in spite of the early changes in the discs which Mr. Lang had noted, there was no progressive deterioration of vision, but I think the case, although the progress was unusually slow, was really one of general paralysis and not of disseminated sclerosis.

REFERENCES.
¹ BRITISH MEDICAL JOURNAL, February 28th, 1920. ² Ibid., April 3rd, 1920. ³ Ibid., April 3rd, 1920. ⁴ Ibid., June 19th, 1920.

A SIMPLE METHOD OF COMBINING ETHER ADMINISTRATION WITH THE VERNON HARCOURT INHALER.

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SINCE writing previously on the subject of the Vernon Harcourt inhaler,¹ advocating more extensive employment of this dosimetric method of chloroform administration, I have had considerable additional experience with the inhaler, and this has not caused me to modify the opinion expressed in that paper. It seems to me that the prevalent attitude towards chloroform, banning its use as an anaesthetic, is extreme, and that this agent still occupies a valuable place. I agree that it must be given with great care, but think dangers may be very largely discounted by the employment of the Vernon Harcourt inhaler, which limits the vapour strength to $2\frac{1}{2}$ per cent.

It is often asked, Why use a complicated mechanism such as this when a drop bottle and simple mask are so much more convenient? The principle in both cases is identical, air being drawn over the surface of evaporating chloroform, taking up a percentage in passing. In reply to this the British Medical Association Chloroform Committee pointed out many years ago that the percentage of chloroform taken up from the mask and drop-bottle method is difficult of control; it may easily exceed $2\frac{1}{2}$ per cent., and when this happens the danger zone is entered. Levy has attacked this view. In a paper on "Cardiac fibrillation and chloroform syncope"² he says:

"It is now obvious that the present-day teaching—that safety lies in attention to vapour percentages—must be permanently abandoned, but there is no reason for a violent reaction, leading to the extreme opposite practice"; and again: "The theory of death by overdose has, in my opinion, been a most deplorable one for humanity."

The experiments on which his conclusions were based were performed almost exclusively on cats. Embley,³ commenting on Levy's findings, suggests that experiments on cats with regard to chloroform action on the heart are misleading. Talking of ventricular fibrillation, he says:

"I have found it to occur during the administration of chloroform in three out of five successive cats, whilst out of several hundred successive dogs in which death had been caused designedly by chloroform under a great variety of conditions, I have never observed it except as a terminal process when the blood pressure had fallen very low from shock or some other cause—quite a distinct event from that form of fatal syncope in cats." And again: "The control of the cardiac rhythm in the dog is markedly vagal, and it approximates in this respect to that of the human."

Whether or not this be the explanation of Levy's theories, they seem to me to be at variance with clinical experience. In using the Vernon Harcourt apparatus the occurrence of symptoms of cardiac failure have been conspicuously less frequent than when ordinary drop methods have been employed. In those cases in which such symptoms have occurred it has been as an accompaniment of shock-producing surgical procedure, and extremely rarely, as Levy's contention seeks to prove, in the early stages of administration.

It is always advisable, however, to have ether available, and a disadvantage of the Vernon Harcourt inhaler is the impossibility of combining ether, or of changing to ether without a change of apparatus. To overcome this the following simple expedient occurred to me: I employ an ordinary Hewitt wide-bore ether inhaler, without the bag. To fit on in place of the bag attachment Messrs. Coxeter and Son made for me a metal diaphragm fitting to the distal end of the Hewitt reservoir and having a perforation cut through its centre to fit the tube of the Vernon Harcourt inhaler. The Hewitt reservoir is charged with the usual quantity of ether, and, as there is no bag and therefore no rebreathing, administers it by the open method.

In practice I induce anaesthesia by means of chloroform through the Vernon Harcourt inhaler and then introduce ether according to requirements. In cases of cardiac failure the chloroform may be shut off and ether alone administered. In my experience this combination apparatus has proved most useful.

REFERENCES.
¹ Glasgow Medical Journal, January, 1917. ² American Year Book of Anaesthesia and Analgesia, 1915. ³ American Journal of Surgery, January, 1916.

Memoranda: MEDICAL, SURGICAL, OBSTETRICAL.

DEATH AFTER TONSILLECTOMY.

THIS note deals with a case which was under the care of Mr. Alban Evans, who kindly agreed to publication and furnished information about the clinical features of the case:

D. R., male aged 14, at puberty, was first seen at the ear and throat out-patient department of Swansea General Hospital complaining of nasal obstruction and frequent sore throat. Examination showed large and unhealthy tonsils, chronic catarrhal rhinitis, and dull, retracted tympanic membrane. Operation was advised, and the tonsils wereenucleated and a large adenoid mass removed by Mr. Alban Evans. Some thyroid enlargement was noticed before the operation; neither during nor after the operation was there abnormal haemorrhage. The boy was removed to the recovery room and appeared to be doing perfectly well, when about half an hour after the operation he suddenly collapsed and, in spite of prolonged remedial measures, did not recover.

A post-mortem examination made next day revealed no sign of asphyxia or violence; the thyroid gland was very obviously enlarged on external view. The brain appeared normal and showed no haemorrhages of any kind. The larynx was normal, the trachea free of blood, and its lumen between the third and fifth tracheal rings was diminished about one-half by pressure from the enlarged thyroid. The sites of the removed tonsils were clear of blood clot; removal had been complete. The thyroid gland was greatly enlarged, weighing 160 grams; a cut into the gland substance showed presence of colloid secretion and some congestion. The gland had completely enveloped the trachea and compressed the posterior wall so that the lumen was crescentic in shape. Histologically, the thyroid showed an unusually large proportion of secreting cells and congestion of the blood vessels; comparison of this gland with sections of simple goitre removed at a thyroidectomy showed it to contain less colloid. The thymus gland was larger than normal, weight 57 grams; it did not exert pressure on the air passages. Microscopic examination showed apparently normal thymic tissue. The oesophagus was normal, as were also the bronchial and cervical glands. The heart, save for a moderate degree of dilatation of the right auricle, appeared entirely normal; the ventricles were in diastole. The lungs were both well aëriated and free from congestion or oedema; there was no collapse of lung tissue nor other sign of respiratory obstruction or failure. The right upper lobe was slightly adherent to the parietal pleura. In the abdominal viscera the only sign of disease found was mesenteric adenitis of tuberculous type, not advanced. The stomach was empty save for a trace of swallowed blood, and the spleen, pancreas, and suprarenals looked quite normal to the naked eye.

The exact mechanism of death in this case is obscure; the leading features were:

1. Enlargement of thyroid with narrowing of trachea—possibly a late event—and enhanced activity of the gland.
2. Enlargement of thymus.
3. An "adenoid" tendency, not very pronounced.

The boy did not die of asphyxia; apparently there was cardiac failure associated with hyperthyroidism, possibly of acute onset, and with an unusually large thymus.

In the discussion on status lymphaticus held by the Royal Society of Medicine at Bournemouth in June, 1914, the opener, Dr. Hugh Thursfield, laid stress on the