CORRESPONDENCE

A DEVICE FOR MECHANICAL VENTILATION SUITABLE FOR NEWBORN AND INFANTS DURING ANAESTHESIA

Sir,—There is a much simpler approach to the problem of mechanical ventilation of the newborn than the one Dr. Doctor describes in his article (Brit. J. Anaesth., 36, 259). Although superficially similar, its mode of operation is clear and straightforward, and we feel your readers might be interested in a brief preliminary description.

![Diagram of ventilation device]

The addition of a small open-ended bag to a standard Ayre's T-piece (1) is a well-established modification. This bag (2) is placed in a jar (3) of about 1 litre volume. The adult ventilator is connected to a small bag (4) of variable capacity in parallel with the jar. Expiration reaches the atmosphere through an automatic inflating valve (5), such as the Fink or Beaver, with the compression gas from the jar. (The Newcastle ventilator is pressure cycled and the pressure tapping is shown (6) for completeness.) Ventilation of the smallest patient, from 3 lb. upwards, can be achieved in this manner.

Although this arrangement has been used for more than 1,200 cases over a period of three and a half years, we have not yet completed full investigations on its exact performance. We agree with Dr. Doctor about the complete inadequacy of the Wright meter in those patients about whom information is most required.

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TRANQUILLIZERS AND HALOTHANE

Sir,—During the past few months there has been much correspondence on the subject of the risks of combining halothane and adrenaline. Again, the hazards to the patient of treatment with the amine oxidase inhibitor group of tranquillizers have become increasingly evident. The responses to pethidine and the amphetamines, and some unidentified factor in cheese, of patients under such treatment are now well documented. It has been suggested that hypertensive crises and possibly cardiac arrhythmias may occur spontaneously and perhaps with fatal result.

These effects resemble those to be expected from a sudden increase in circulating adrenaline. If this be true it would be reasonable to expect an adverse response if halothane were employed to anaesthetize a patient under treatment with one of this group of tranquillizers.

The following experience appears to support this argument.

Case report.

A physically healthy male fitter, 36 years old, presented with a history of six weeks' dental pain and one day's increased pain and facial swelling associated with carious and abscessed maxillary first and second molars.

For the past two years he had been under psychiatric treatment with tranylcyromine (Parnate) 10 mg t.d.s., and trifluoperazine (Stelazine) 5 mg t.d.s. until six weeks previously when he had started to take Spansules of Stelazine 15 mg (instead of tablets of trifluoperazine) and tranylcyromine 10 mg b.d.

On examination prior to anaesthesia his temperature was 99°F, resting pulse 76 beats/min, and blood pressure 130/80 mm Hg.

Suppuration precluded local analgesia and the patient's pathological fear of injections made him object to intravenous induction, so inhalation anaesthesia was employed. Induction was with nitrous oxide, oxygen and halothane, using 15-20 per cent oxygen from a Walton Mk. 5 and a Goldman Mk. 2 halothane vaporizer. This was smooth and uneventful and the halothane was cautiously advanced to the full "on" position. After six or seven breaths of this concentration and just as surgery was about to begin, the pulse rate suddenly rose to 110-120 beats/min and the pulse became grossly irregular in a manner indicative of extrasystoles. The halothane was at once withdrawn and surgery was cautiously started under nitrous oxide and oxygen alone, the offending tooth being extracted. During the subsequent 5-7 minutes the patient recovered consciousness and the pulse became regular again and the rate settled down to 90 beats/min. Recovery was normal and when he was discharged 1 hour later his pulse rate was 90 beats/min and his blood pressure 130/95 mm Hg.

Monitoring with a blood-pressure follower before beginning anaesthesia was contemplated, but to avoid further excitement in an already nervous patient this was not done.

Commenting upon the sudden death of a boy of 16 under treatment with an amine oxidase inhibitor drug, Womack (1963) suggests that the drug provokes hypertensive crisis and possibly cardiac arrhythmia. Such a crisis may be precipitated by emotional reactions and is presumably mediated by release of catecholamines. The unpremedicated patient in this report may well have responded to the emotional stress of his impending operation in this manner and may, in fact, have been on the verge of a hypertensive crisis before induction.
METHOXYFLURANE AND BLOOD PRESSURE

Sir,—I was interested to read the paper by Black and Rea (Brit. J. Anaesth., 36, 26) on the effects of methoxyflurane in paediatric anaesthesia, and, in particular, their comment upon the lack of hypotension. However, they go on to say "... in the adult, hypotension is frequently seen when methoxyflurane is inhaled." In support, they quote the findings of Walker, Eggers and Allen (1962) who, from haemodynamic studies on five males admitted for elective surgery, state "... the cardiovascular effects of methoxyflurane anaesthesia resemble those of halothane rather than diethyl ether ... ", which agreed with their clinical experience. They quote Bagwell and Woods (1962), who, in thirty-five dogs found "... the degree of cardiovascular depression during induction and light levels of anaesthesia with methoxyflurane are similar".

In the past ten months I have been using methoxyflurane in various fields, and it is my clinical experience that one of its advantages is that blood pressure is maintained close to pre-induction levels during anaesthesia—in fact, this stabilizing cardiovascular effect makes it especially valuable in selected cases.

In this, our results are more in line with Knox, North and Stephen (1962), who, in a study using methoxyflurane and controlled ventilation, found that "... since the conventional signs for determining the depth of anaesthesia were absent with methoxyflurane, the level of anaesthesia was followed clinically by response of blood pressure to increase and decrease of concentration. In this series, almost cadaveric muscle relaxation could be obtained while maintaining a clinically adequate systolic pressure—during surgical procedures the average reduction in systolic pressures from pre-operative reading was 12.3 mm Hg". The maximum concentration available from their vapourizer was 1.6 vols per cent; induction was with a minimum of 1 per cent and maintenance from 0.2 to 0.5 per cent. Without attempting to produce muscle relaxation (I do not think methoxyflurane used alone is suitable for abdominal surgery) and using higher concentrations than those of the American workers—viz., 2.8 per cent for induction and approximately 1 per cent for maintenance—I have seen very little cardiovascular depression. The fact that systolic pressure remains high during administration makes it the agent of choice for procedures where avoidance of hypotension is either obligatory or preferable, where relaxation is not required. I use it for peripheral arterial surgery, arteriography, especially carotid, the pinning of hips in the elderly and so on, and hope soon to publish a paper with a fuller report on my findings.

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REFERENCES


ACETYCHOLINE—HYDROLYSIS OR DESTRUCTION

Sir,—Thank you very much indeed for inviting me (Brit. J. Anaesth., 36, 66) to explain my views on the terminology of acetylcholine hydrolysis.

It is stated (Evans and Gray, 1959) that "Acetylcholine depolarises the region and is almost immediately hydrolysed by a tissue enzyme cholinesterase, to acetic acid and choline.

Other authorities (Wylie and Churchill-Davidson, 1960) state: "... acetylcholine molecules only have a minute distance to travel before reaching their target... their subsequent fate, however, is a little more obscure. The majority are certainly hydrolysed by the specific enzyme-cholinesterase to acetic acid and choline. The molecules of choline are then available for resynthesis by the nerve terminal"

Another authority (Foldes, 1957) states "acetylcholine is attracted to... the acetylcholinesterase and is rapidly hydrolyzed to acetic acid and choline... subsequently the hydrolysis products of acetylcholine, choline and acetic acid are resynthetised to acetylcholine..."

Another authority (Samson Wright, 1963) says: "... cholinesterase splits acetylcholine to choline and acetic acid. The choline is available for synthesis of more acetylcholine...

Thus it can be seen that there is general agreement that after the hydrolysis of acetylcholine by acetylcholinesterase into acetic acid and choline, this latter becomes again available and is used for resynthesis into acetylcholine.

On the other hand, the term "to destroy" is defined (Pocket Oxford Dictionary, 1955) as "reduce to nothing or to uselessness" and by another source (Concise Oxford Dictionary, 1952) "make useless, annihilate"

From all the foregoing it would, therefore, appear that, when describing the action of an enzyme in general, and, in this case, of acetylcholinesterase in particular, the term "to hydrolyse" or "to split" would be the term of choice and that the term "to destroy" ought not to be used in this context.

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