CORRESPONDENCE

INCREASE IN HALOTHANE CONCENTRATION FOLLOWING REFILLING OF CERTAIN VAPORIZERS

Sir,—I am sure that many anaesthetists on reading the excellent article by Drs. Jennings and Hersant (Brit. J. Anaesth., 37, 137) on the increased concentration of halothane vapour following the refilling of the halothane bottle, will recall to mind a procedure that was adopted pretty routinely by anaesthetists in the days before relaxants.

The routine anaesthesia was thiopentone, nitrous oxide, oxygen and ether, and one soon learned that after refilling the ether bottle it was essential to remove the cork from the ether vaporizing bottle, and to flick the lever fully over, otherwise an excess of ether vapour was produced on a partial opening of the lever, which resulted in coughing and spasm. This was particularly troublesome following thiopentone. One accepted this as routine procedure but, unfortunately, did not investigate it, hence it is gratifying to see scientific facts replacing blind faith after many years, and Drs. Jennings and Hersant are to be congratulated on bringing to light this potential danger of increased halothane concentration.

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PERIPHERAL PULSE AMPLITUDE IN HALOTHANE ANAESTHESIA

Sir,—In case any of your readers would like to practise amplitude observation, I have the following practical comments to make.

With regard to the pulse indicator, those I have used have all been home-made, using Dr. Bishop's circuit. I have tried with an early type Sonopulse (MIE) but found that its amplification always gave a maximum reading. I have also tried with a Cotel Keating pulse indicator but failed to obtain satisfactory results. I do not know whether this was due to the particular instrument or whether this is common to all Cotel Keating indicators. An oscilloscope display of the peripheral pulse wave would be a more erudite method and inertia would be minimal; however, this would be subject to interference from radio-frequencies such as short-wave diathermy.

Perhaps a few words should be said about the end of the anaesthetic and re-establishment of spontaneous respiration. Apnoea may be due to the following anaesthetic causes: (a) relaxants which are still active; (b) deep halothane anaesthesia; (c) hypocapnia.

Paralysis from relaxants can be excluded by the history and the use of a nerve stimulator. Deep halothane anaesthesia is not the cause if the systolic blood pressure is greater than 80 mm Hg. Having excluded (a) and (b), carbon dioxide added to the oxygen or nitrous oxide-oxygen mixture will invariably start spontaneous respiration, but excess carbon dioxide must, of course, be avoided.

My usual practice is to continue IPPR with an oxygen flow of 5 l./min on a partially open circuit about 15 minutes before the end of the operation. When the systolic blood pressure has risen to 80 mm Hg I then continue with 50 per cent oxygen and 50 per cent nitrous oxide with the soda lime canister switched off. If at the end of the operation good spontaneous respiration, with early signs of regaining consciousness, is not occurring, I administer carbon dioxide until there is a small degree of hyperpnoea. Finally I maintain the patency of the patient's air passages while deep breaths of air are taken.

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ARTERIAL OXYGEN TENSION AND AGE

Sir,—In their paper "Arterial oxygen tensions of patients awaiting surgery", Conway, Payne and Tomlin (Brit. J. Anaesth., 1965, 37, 405) confirmed the earlier findings of Raine and Bishop (1963), who found an inverse correlation of arterial Po2 with age. It is fair to point out that very similar results were presented at the October 1964 meeting of the Association of Anaesthetists at the Royal College of Surgeons. The abstract, which was published in January 1965, contains the following: "A preliminary study of 50 unanaesthetized, healthy, supine adults showed a highly significant negative correlation of Po2 with age, Pco2, remaining constant" (Marshall, 1965).

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REFERENCES
