sibility for deciding to institute replacement therapy if he (the anaesthetist) considers such therapy to be necessary for anaesthetic safety.

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London

RESPIRATORY OBSTRUCTION DUE TO IMPACTION OF THE EPIGLOTTIS IN AN ADULT

Sir,—Dr. Thomas’s account (Brit. J. Anaesth. (1964), 36, 314) of his case of respiratory obstruction due to impaction of the epiglottis is a very clear description of an important but almost unknown complication. The author failed to find it mentioned in the literature, and as far as I know it has not been reported in any journal devoted to our specialty.

There are, however, three references by laryngologists to respiratory obstruction by the epiglottis in the British Journal of the 1920’s:

(1) Brit. med. J., 1923, 2, 327. The patient became cyanosed under anaesthesia; “... the epiglottis was entirely devoid of cartilage and was of the consistency of a soft piece of wash-leather. It was lying right over the larynx and in tight contact with the posterior pharyngeal wall. ... On hooking the epiglottis forward with my finger, the patient took several deep breaths and became quite pink again. ... I continued the operation, stopping at intervals to hold the epiglottis whenever the respiration became laboured.”

(2) Brit. med. J., 1924, 1, 353. “... under chloroform anaesthesia the respiration fails first owing to the epiglottis sagging back and occluding the upper aperture of the larynx: forcible attempts at inspiration may lead to its impaction. ... The patient seemed to be dead, but I restored her by hooking up the epiglottis, which was definitely impacted in the larynx.”

(3) Brit. med. J., 1927, 2, 238. “I am led to believe that many deaths under anaesthesia may be due to this preventable cause. ... In considering respiratory failure under anaesthesia, occlusion of the larynx by the epiglottis seems to have passed largely unnoticed.”

During the war G. H. Caiger published an important article “The Role of the Epiglottis in Anaesthetic Deaths” in the Journal of Laryngology and Otolaryngology, 1942, 57, 250. In this he wrote: “... under anaesthesia respiration ceased. I found that the epiglottis was firmly impacted in the larynx, but it was easily released, with an audible click. ... Respiration proceeded normally at once. ... In one patient impaction occurred twice during the same anaesthetic.” Caiger made the point that impaction of the epiglottis may well be the cause of mysterious death under anaesthesia. “There are good reasons for its absence of postmortem evidence. Such evidence is not likely to be found unless it is looked for, and unless care is taken that no procedure is carried out that may result in the epiglottis being freed from the larynx before the parts are seen.”

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A COMPLICATION OF THE USE OF SUXAMETHONIUM "EXTENSION" BY TETRAHYDROAMINACRINE

Sir,—I read with interest Dr. Ozorio’s case report (Brit. J. Anaesth. (1964), 36, 322) of a complication arising from the use of tetrahydroaminacrine and suxamethonium. I was under the impression that this was a well-recognized possibility.

I believe it is true that tetrahydroaminacrine given to a patient, without having given atropine 0.6 mg intravenously immediately beforehand, is liable to cause considerable vagal overactivity, presenting a clinical picture such as Dr. Ozorio describes as occurring in his patient. I have seen this happen to one or two of my patients in whom I have omitted giving atropine prior to tetrahydroaminacrine. I think if Dr. Ozorio had given intravenous atropine alone when resuscitating the patient, there would have been an improvement; i.e., I think the methoxamine is of little importance in the treatment of a patient in whom this state develops after the use of tetrahydroaminacrine and suxamethonium.

Some twenty-five or more years ago there was a method practised, which found favour in certain centres, known as "secondary saturation". Briefly, it will be remembered that the essence of this method was to deliberately make the patient a "poor risk" by successive episodes of anaoxia, and it was then found that major surgery could be carried out under nitrous oxide and oxygen anaesthesia alone. Possibly this might be an explanation of the prolonged period of apnoea in Dr. Ozorio’s patient. After the period of hypotension the patient would have become a "poor risk", and this, together with Dr. Ozorio’s efficient ventilation—his figures indicate approximately 36 l./min., a good minute volume for a small patient —I feel might be sufficient to keep the patient apnoeic for the period mentioned.

Another clue stated by Dr. Ozorio was that the patient was undernourished; this makes one wonder if there was some latent electrolyte disturbance, as one has seen bizarre responses to suxamethonium in patients with electrolyte disturbance but a normal blood pseudocholinesterase.

I think the important points in Dr. Ozorio’s case report are that atropine should be given immediately prior to the use of tetrahydroaminacrine and that, provided efficient ventilation is maintained, an apnoeic patient is safe. I do not feel there is a case for using a test dose of suxamethonium.

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