de 30-35 cm H₂O pendant 0,5 à 1 sec. La radiographie du thorax est très importante pour le diagnostic de la détresse respiratoire chez le nouveau-né. Suivant la sévérité du pneumothorax en cause le traitement peut aller des mesures conservatrices jusqu’au drainage sérieux du pneumothorax en cause le traitement peut aller des mesures conservatrices jusqu’au drainage aspirateur intercostal sous l’eau.

**PNEUMOTHORAX BEIM NEUGEBORENEN**

**ZUSAMMENFASSUNG**


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**CORRESPONDENCE**

**RE-ANAESTHETIZING CASES OF TONSILLECTOMY AND ADENOIDECTOMY BECAUSE OF PERSISTENT POSTOPERATIVE HAEAMORRHAGE**

Sir,—There are certain points in Dr. Denison Davies’ article on this subject (Brit. J. Anaesth., 36, 244) which merit challenging! With reference to the advocated removal of clots before inducing anaesthesia, I find that clots which are really loose are expectorated or swallowed by all but heavily narcotized patients, while the usual semi-adherent clots in tonsil fossae remain in position during anaesthetic induction and have to be swabbed away by the surgeon: when this is done, fresh and maybe brisk bleeding from the vessel concerned (there is invariably only one such) occurs. Surely the last thing desirable would be fresh and fast bleeding during induction of anaesthesia?

Having personally so far effectuated upwards of 10,000 tonsillectomies with or without adenoidectomy, and likewise dealt with something like forty cases of postoperative haemorrhage, it seems most remarkable that over 200 cases should have been quoted as returned to the operating theatre for postnasal bleeding: I have known only one such case where another operator had, at a guillotine operation, left half the adenoids behind. Such bleeding, to my mind, implies imperfect adenoidectomy, which can be avoided altogether in the open “dissection” operation which is nowadays, one hopes, the routine procedure elsewhere as hereabouts, where anaesthetists feel quite free to use nasally introduced intratracheal tubes.

By all means empty the stomach of swallowed blood if possible; but blood-volume diminution of serious degree should never occur in primary postoperative haemorrhage if promptly dealt with, and in this connection I think the surgeon’s opinion ought always to prevail. I teach juniors that primary haemorrhages should never be allowed to reach the stage of having to contemplate blood replacement. Secondary haemorrhages admitted from outside hospital may, of course, be in a condition to need blood replacement before operation.

**M. HAYDON-BAILIE**

Worksop

A copy of this letter was forwarded to Dr. Denison Davies, who replied as follows:

Sir,—I should like to comment on one or two points raised by Mr. Haydon-Baillie.

Respiratory obstruction by inhaled blood clots is a very real hazard in these cases and one cannot rely on all loose clots being expectorated or swallowed before induction of anaesthesia. Naturally one does not want to start fresh bleeding, and therefore only clots which are obviously loose should be removed. It is certainly remarkable that over 200 cases should be quoted as having returned to the theatre because of postnasal bleeding. These cases were from a series of 21,500 children, the majority of whom had had adenoidectomy performed; this, certainly, must represent an abnormally high return rate. In this connection it is significant that out of a total of five deaths in this series of 21,500 cases, four were directly related to postnasal bleeding. This high return rate is probably related to the fact that most of the tonsillectomies and adenoidectomies in the series were performed by junior house surgeons in training. It is possible that postoperative bleeding is occasioned by imperfect adenoidectomy, but one wonders whether the too radical use of an over-sharp curette may not be a more common cause.

Mr. Haydon-Baillie states that in cases of primary postoperative haemorrhage serious blood volume depletion can always be avoided. This, most certainly, is an unattainable ideal and must represent a dangerously unrealistic approach to the problem. Blood can be swallowed by the patient and a considerable blood loss may have occurred before the ward staff are aware that the patient is bleeding. In fact in many cases postoperative bleeding may be actually heralded by the patient, quite unexpectedly, vomiting a dangerously large quantity of blood.

One wonders what is meant by a “serious” degree of blood-volume diminution. A deficit which is not serious in a patient lying in bed in a ward may be very serious if that patient is anaesthetized and his airway becomes temporarily obstructed by blood clots. Naturally the anaesthetist must accept respon-
sibility for deciding to institute replacement therapy if he (the anaesthetist) considers such therapy to be necessary for anaesthetic safety.

D. Denison Davies
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 Medical Journal of the 1920's:

(1) Brit. med. J., 1923, 2, 327. The patient became cyanosed under anaesthesia and 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 Otology, 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."

It would appear unlikely that Dr. Thomas's case had any reference, as he thought, to the thyroglossal cyst which had been excised thirty years previously. It is more likely to have been due to the epiglottis which was described as being unusually floppy.

ROBERT MACINTOSH
Oxford

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 anoxia, 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 9 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.

G. A. Rawlins
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