NASAL INTUBATION: SOME POSTNASAL OBSTRUCTIONS AND HOW THEY MAY BE OVERCOME

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During the passage of a tube from nostril to trachea certain difficulties may be encountered. The anterior nasal opening may be unusually narrow, there may be a septal deflection or spur, or an inferior turbinate may be tiresomely oversized. On such occasions, of course, the effect of rotation of the tube is tested, the other nostril is tried, or a narrower tube used.

However, a little beyond the deflected septum or the oversized turbinate there is quite frequently firm resistance to progress of the tube. This has rather vaguely been considered to be due to adenoids or their remnants, to deformities or to scar tissue; and indeed at times such may be the case. But digital examination shows that the commonest causes of obstruction on the posterior wall of the nasopharynx are a prominent anterior arch of the atlas (fig. 1) or the hollow above it (fig. 2). It is at these points that the tip of the endotracheal tube may be resisted in its passage through the nasopharynx. Such prominence of the atlas may be accentuated by Passavant's bar or ridge, which is made up of the fibres of the horizontal part of the palato-pharyngeus muscle where they meet their fellows of the opposite side, forming with the soft palate a sphincter between oro- and nasopharynx (Townshend, 1940). This slim muscular ridge lies on the anterior arch of the atlas but is probably of little significance when relaxants are used and the sphincter is fully relaxed.

Rarely, a tube may be held up on the lateral wall of the pharynx in one or other of the pharyngeal recesses (or fossae of Rosenmüller), hollows of variable depth, one on each side, above and behind the tubal elevations (or Eustachian cushions); but almost all tubes passed nasally take a course medial to these structures.

It is also possible for the tip of the tube to be caught up in the adenoids and perhaps in the

FIG. 1

A nasal tube (filled with radio-opaque substance) shown impacted on a prominent anterior arch of the atlas in a young man anaesthetized through an orotracheal tube.
This enigmatic bursa, to the anaesthetist, is entirely a textbook entity, and to the ear, nose and throat surgeon is only apparent when it gives rise, exceedingly rarely, to a central cyst or abscess (Thornwaldt's disease). Among anatomists there seems to have been little radical change of opinion since Von Luschka (1868) described it as an elongated pocket lined with epithelium and with an orifice situated in the midline at the lower border of the pharyngeal tonsil, extending backwards and attached to the periosteum of the occipital bone. Poirier and Charpy (1901) while admitting the difficulty of assessing its incidence owing to "des accidents pathologiques" thought that in the majority of humans a 1 to 6 mm circular orifice, in the median plane, leads into a tubular recess 1 cm or so deep; while Gray (1954) reminds us that the origin of the bursa is due to persistence of a developmental connection between notocord and pharyngeal endoderm. Others (Jackson and Coates, 1930) are mildly sceptical and consider that the bursa is no more than a recess formed by adhesions between the folds of adenoid tissue.

However this may be, a careful digital examination of many hundreds of nasopharynges as a nasopharyngeal tube was being passed has failed to reveal any central pouch or sac-like depression in the roof or posterior wall of a size likely to cause obstruction; with the exception, of course, of the hollow above a prominent anterior arch of the atlas. It is justifiable, therefore, to conclude that the pharyngeal bursa is something of a rarity, or is very small indeed, and may safely be excluded as a common cause of obstruction to nasotracheal intubation.

Few anaesthetic textbooks do more than give a passing reference to adenoids if they discuss postnasal obstruction at all. Wylie and Churchill-Davidson (1960), however, do favour the
pharyngeal bursa, together with adenoids, as frequent causes of obstruction in this region. They say:

"Lying close to the base of the pharyngeal tonsil (or adenoids) is a small recess—the pharyngeal bursa. These structures often impede the passage of an endotracheal tube; if force is used the tube may penetrate the mucosa and create a false passage which can lead to trouble from sepsis and collection of secretions during the postoperative period."

This description of postnasal obstruction is perhaps occasionally accurate when the adenoids are present. When they have been removed or are atrophic it seems clear that other causes must be sought. Moreover, although it is agreed that firm adenoids may impede a tube in the young, it is thought that they are more often a cause of bleeding than of obstructon.

Before suggesting a means of overcoming these forms of postnasal obstruction, it is relevant to quote Thomson and Negus (1955) as they describe the end of an operation for removal of the adenoids: "Finally, the curette is laid aside and the forefinger is again introduced to ascertain that no thickening remains, that there is no obstruction in the posterior choanae, and that the fossae of Rosenmüller have been cleared." It is this manoeuvre with the forefinger, so familiar to the ear, nose and throat surgeon, which may be helpful to the anaesthetist and it is described for any not already aware of it.

Thus, a tube passed through the right nostril which has met such an obstructon, either laterally in the pharyngeal recess or at any point on the posterior wall against a prominent anterior arch of the atlas or mass of adenoid tissue, should be left in place. The lubricated forefinger of the right hand is passed directly back to touch the posterior wall of the oropharynx, thence laterally behind the right posterior fauces pillar. It is then in the plane of the nasopharynx and an upward sweep carries it behind the soft palate without folding that structure upwards (to fold the soft palate upwards is damaging in itself and limits movement of the finger in the nasopharynx). Often, the tip of the endotracheal tube is felt, obstructed at one of the points mentioned above, usually the anterior arch of the atlas. The body of the tube should be moved slightly to and fro with the other hand, while the tip is pushed over the obstruction with the finger which is behind the soft palate (fig. 3). When the left nostril has been used the position of the hands should be reversed. If bleeding occurs this can be coped with by suction, pack or posture. It must be stressed that the nasal route should only be used when its advantages clearly outweigh its recognized dangers and difficulties.

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REFERENCES

This monograph on disturbances in the gas exchange of the lung aims at differentiation between four causes of hypoxia, viz. overall ventilation deficiency (called global), disturbances of oxygen diffusion, irregular distribution of gases within the lung, and vascular short-circuit. The author tries to correlate clinical signs and symptoms with the results of respiratory function tests and gas-analysis measurements and he expects that these will provide additional clues for the diagnosis of complicated cases and for their prognosis.

In the theoretical part he describes his methods of investigation and gives various equations and formulae (19) for calculation and evaluation of oxygen saturation and tension, carbon dioxide tension, oxygen diffusion capacity, venous admixture to the arterialized blood of the pulmonary circulation, and, in a later section, the two components of oxygen diffusion from alveolar air to haemoglobin. Measurements are taken at varying oxygen contents of inspired gases, at rest, and during work on an ergometer bicycle.

Interesting results were obtained in patients suffering from obstructive emphysema due to either chronic bronchial asthma on an allergic basis, or to recurrent purulent asthmatic bronchitis, with or without cor pulmonale. In allergic asthma the distribution of gases becomes faulty while, even after years, the oxygen diffusion capacity remains unimpaired. In contrast, it is decreased in chronic bacterial asthma bronchitis due to inflammatory changes in the interstitial tissue caused by repeated infections. If the disease continues unchecked progressive destruction of interalveolar septa and pulmonary capillaries results in further deterioration of the oxygen diffusion capacity and leads, together with increasing insufficiency of the right ventricle, to a dangerously high admixture of venous to the arterialized blood. Besides, an overall ventilation deficiency is found, and these three causes of hypoxia combine to reduce the oxygen saturation to values incompatible with life.

As the tests are difficult and time-consuming the number of patients in each group is understandably small, but under these circumstances the author should not have given averages derived from five, four, or even two cases whose examinations some-times yielded widely divergent figures. It does not make for easy reading that while studying one table one has frequently to refer to several others in order to identify the symbols used. Although the timed vital capacity test is carried out in all investigations the range of what are considered to be normal values is not given. The figure of 83 per cent (Wylie and Churchill-Davidson, 1960) is hardly ever reached, not even in extrapulmonary diseases.

The booklet is too specialized for most anaesthetists but may interest those who undertake research on respiratory or circulatory problems.

Luise Wislicki

REFERENCE


The therapeutic value of injections of local anaesthetics is accepted. Few, however, think of the damage which can result, not only from the injection of a large volume of fluid but also from the local toxicity of the active agents. This new volume in the American Lecture Series is of considerable interest to all anaesthetists. Not only has it reviewed the literature (ninety-seven references) but it also reports the results of experiments on nerve, muscle and skin, comparing the local toxicity of twelve local anaesthetics and other solutions injected occasionally, either intentionally or by accident. Full-page illustrations of the histopathologic effects are given and enable the reader to assess for himself the damage caused in the different tissues.

This monograph brings our knowledge of this subject up to date and gives a guide to the further studies needed to give a complete picture of the histopathology following local injections.

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