Laryngospasm and bronchospasm are recognized as hazards of anaesthesia in both man (Gillespie, 1950) and animals (Wright and Hall, 1961). Laryngeal spasm has been produced experimentally in cats (Harrison and Vanik, 1963) and more recently there has been a report of the sensitization of laryngeal sensory receptors by a variety of chemical agents (Dirnhuber, Green and Tregear, 1965).

The experiments on which the present report is based were concerned with both the experimental production of laryngeal spasm and with a definition of the regions of the respiratory tract from which the spasm may be stimulated. Twenty-seven decerebrate preparations (intercollicular sections) of cats, and five cats under chloralose anaesthesia (65 mg/kg injected intravenously) were the experimental preparations used. Conventional techniques of electromyographic recording were used to follow the activity of the cricothyroid muscle and of the diaphragm (Basmajian, 1962). The detecting electrodes were concentric needle electrodes (Adrian and Bronk, 1929).

M. cricothyroideus has been identified as an adductor of the vocal cords (Faaborg-Andersen, 1957); its activity was found, in the present study, to be representative of the group of muscles characterized as the vocal cord adductors. Two types of resting activity of the cricothyroid muscle were identified in the preparations used. One of these consisted of a continuous low frequency discharge of a relatively few motor units; the other was of phasic activity, again of relatively few motor units — this phasic activity was related to the inspiratory activity of the diaphragm (fig. 1, upper trace).

Harrison and Vanik (1963) showed that inhalation of ether by cats may stimulate laryngospasm. They depended on direct observation of the vocal cords. Their observations have been confirmed in the present study by direct detection of the responses of vocal cord adductor muscles (usually M. cricothyroideus, but also M. vocalis, M. cricoarytenoideus lateralis and M. arytenoideus). In addition it has been shown that inhalation of halothane vapour at a concentration of 6-8 per cent stimulates laryngospasm.

In the present study, ether was administered from a standard Boyle ether vaporizer with the lever in the full-on position and the plunger up. Macintosh, Mushin and Epstein (1963) showed that the concentration of ether obtained from the vaporizer under these conditions was between 10 and 20 per cent, and in clinical practice concentrations of up to 20 per cent are required for induction of anaesthesia (Wood-Smith and Stewart, 1962). Halothane was administered from a modified trichloroethylene vaporizer (Hillard, 1957) and the concentration, according to the calibration curve (British Oxygen Co.), was in the range 6-8 per cent. This is within the range used by Johnstone (1961) in his high percentage induction technique.

The inhalation of 10-20 per cent ether or 6-8 per cent halothane administered via a facepiece (Hall, 1957) was followed within 0.6 sec by a high frequency discharge from a greater number of motor units of the cricothyroid muscle. With the administration of these anaesthetic agents for 24-30 sec, this discharge continued for 24-45 sec. The initial period of administration of the anaesthetic agents was also accompanied by apnoea which was recorded as a cessation of the rhythmic contraction of the diaphragm (fig. 1). After withdrawal of the anaesthetic agent the discharge from the cricothyroid muscle returned to normal and a normal respiratory rhythm was resumed.
The sites of stimulation by ether or halothane of laryngospasm have been identified not only as larynx, but also as cervical trachea and lower respiratory tract. This was demonstrated by appropriate cannulation, or isolation, of the trachea and larynx. In the case of the larynx, the effects were reduced by spraying its anterior regions with 2 per cent lignocaine hydrochloride, and abolished when the internal branch of the superior laryngeal nerves was cut on both sides. After both these procedures, laryngospasm still occurred when the cervical trachea or lower regions of the respiratory tract were exposed to 10–20 per cent ether or 6–8 per cent halothane.

The present experiments confirm observations made by Harrison and Vanik (1963). They also show that halothane stimulates laryngospasm. Ether and halothane were administered at concentrations employed in clinical anaesthesia with apparatus also used in clinical anaesthesia. Stimulation of laryngospasm may occur with administration of these agents via a facepiece or endotracheal tube.

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STIMULATION OF LARYNGOSPASM BY VOLATILE ANAESTHETICS


BOOK REVIEWS


Within the space of four years this book has become one of the best known in the German language. The new edition with its many revisions and additions will without doubt maintain that position. Both authors some part of their early training in Britain, and this influence can be recognized even by those without a good knowledge of German from the illustrations and general layout of the text—so very different from books on anaesthesia in former years written by surgeons. Professor Barth and Dr. Meyer have once again presented their colleagues with an up-to-date, concise, practical and, above all, scientific treatise on modern anaesthesia, which will be a credit to themselves, to German anaesthesia, and to their British teachers and friends.

W. W. Mushin


The earliest major cardiac operations both with and without bypass were carried out in Philadelphia. It is therefore not entirely inappropriate that a Symposium on Anaesthesia for Open Heart Surgery should recently have been held there. This book is an edited account of the proceedings of the Symposium. It has, however, been so well edited that though quite a few articles are largely written in the first person, the ultimate result would really be best entitled "The Beginner's Guide to Anaesthesia for Cardiac Surgery". For each of twenty contributors provide 1000–2000 words on an aspect of anaesthesia for cardiac surgery. In addition to the detail of anaesthesia subjects such as estimation of blood loss, monitoring and even operative position and draping are dealt with. There is a brief account of the postoperative problems and their management. The vast majority of the contributions have no references.

One or two of the contributors give an indication of the authorities they quote, Inevitably there is a considerable amount of overlap; for example, tracheostomy and artificial ventilation are mentioned in three different sections of the book. To one whose experience of anaesthesia for cardiac surgery is limited it would appear that this book would provide a useful introduction to the subject.

A. R. Hunter

CORRESPONDENCE

BLIND INTUBATION

Sir,—I am indebted to Dr. P. Chandra for his excellent account on the subject of blind intubation of the trachea (*Brit. J. Anaesth.*, 1966, 38, 207). The article serves to add weight to my argument that junior anaesthetists today should be fully familiar with this technique.

Dr. Chandra points out that there are few surgical patients on whom direct laryngoscopy and intubation cannot be performed, and it may be that the number of such cases he reports is in excess of that likely to be encountered in an average general hospital in this country. However, this should not deter each anaesthetist from being adept at a technique which may on occasion prevent an embarrassing situation or may even be life-saving.

At a very early stage in my career as an anaesthetist I was taught a standard method of blind intubation under general anaesthesia, and encouraged to use and practise it whenever possible. I am extremely grateful to the consultant who first taught me and have since been keen to pass on this good advice to other Junior anaesthetists.

PETER B. HARDWICK

London