FATAL HAEMORRHAGE FROM THE INNOMINATE ARTERY
AFTER TRACHEOSTOMY

A Case Report
BY
A. D. IVANKOVIC, SHARON THOMSEN AND C. C. RATTENBORG

SUMMARY
A case of fatal haemorrhage from the innominate artery occurred a week after tracheostomy was performed. Control of the bleeding was tried unsuccessfully by hyperinflation of the cuff of the tracheostomy tube. The mechanism is erosion of the artery by the inferior border of the tracheostomy tube. Since the treatment of massive haemorrhage from the innominate artery is extremely difficult, the avoidance of low tracheostomies and the use of short and properly curved tracheostomy tubes may be effective in prevention of this fatal complication.

Tracheostomy, usually regarded as a minor surgical procedure, is not without hazards. Numerous operative and postoperative complications have been reported with incidences ranging from 13 to 100 per cent. Also, mortality due to the procedure itself is surprisingly high, from 0.9 to 5.3 per cent, according to different authors (Meade, 1961; Head, 1961; Davis, Kretchmer and Bryce-Smith, 1953).

The first case of ruptured innominate artery after tracheostomy was reported by Korte in 1879. Schlaepfer reviewed 115 cases of fatal haemorrhage after tracheostomy that had been reported prior to 1924. He found that the highest incidence of fatal haemorrhage was due to rupture of the innominate artery. In 1956, Davis and Southwick reported two new cases of fatal rupture of the innominate artery. They described pulsation of the tracheostomy tube as a pathognomonic warning sign of possible impending rupture of one intrathoracic artery. Recently, Silen and Spieker (1965) presented four proved and two suspected instances of massive haemorrhage from the innominate artery after tracheostomy.

CASE REPORT
A 21-year-old female was admitted to the hospital because of progressive muscular weakness, which developed five days prior to admission after a slight upper respiratory infection and a sore throat. The tentative diagnosis was the Guillain-Barré syndrome. On the third hospital day, the patient experienced steady worsening of her weakness and sensory loss. By the fourth hospital day, she had marked weakness, a left positive Babinski sign, and decreased ability to cough and to swallow. On the fifth hospital day, she had increased difficulty with secretions and respiration and was transferred to the Intensive Care Unit, where tracheostomy was performed under local anaesthesia; a size 12 Portex tracheostomy tube with cuff was inserted. On the same day, her respiratory paralysis increased, and, at that time, an arterial sample was drawn and the following results obtained: $P_{O_2}$ 58 mm Hg, $P_{CO_2}$ 27 mm Hg, and pH 7.48. Assisted ventilation with a Bird ventilator was begun at that time. Because of her downhill course, steroid therapy was started. After one day of slight improvement, however, her weakness became progressively worse. She was unable to move her extremities, had a bilateral facial paralysis, and was unable to swallow. She exhibited a complete lack of sensation except for some discomfort at the site of the tracheostomy tube. However, her spirits remained good, and she complained only of moderate pain in the tracheostomy area. No pulsation of the tracheostomy tube was noticed. On the twelfth hospital day, her respiratory effort was unable to trigger the respirator, and she was placed on controlled ventilation. On the same day, she suddenly developed massive bleeding both through and around the tracheostomy tube. It was tried immediately to control haemorrhage by hyperinflating the cuff of the tracheostomy tube and by suctioning of the trachea. However, it was not possible to stop this enormous outpouring of blood, and it was almost impossible to ventilate the patient's lungs. Her pupils dilated within a minute and, shortly after that, the blood pressure became unobtainable. At that point, it became possible to clear the trachea of blood and to restore ventilation. Resuscitation was continued with external cardiac massage and intra-
FATAL HAEMORRHAGE AFTER TRACHEOSTOMY

The arrow points to the perforation of the innominate artery with the inferior border of the tracheostomy tube approximately 1 cm from aorta.

Venous administration of plasma, lactated Ringer's solution, sodium bicarbonate, isoprenaline, and calcium chloride. Although electrical activity of the heart did not cease, no pulse could be palpated, and resuscitative measures were discontinued approximately 30 minutes after the bleeding started.

Autopsy showed the tracheostomy opening at the seventh tracheal ring. A marked dilatation of the trachea was noted below the tracheostomy and was associated with granulation tissue and atrophy. The innominate artery showed a hole approximately 1 ml in diameter in direct communication with the tracheal lumen. Demyelination, oedema, and cellular infiltrates involving bulbar and spinal nerves were consistent with a diagnosis of Guillain-Barré syndrome.

COMMENT

Although haemorrhage is one of the most common complications of tracheostomy, it is fortunate that haemorrhage caused by a rupture of a major vessel is extremely rare. Davis and Southwick (1956) stated that the tip of a long tracheostomy tube was the cause of fatal erosion in their two cases. Silen and Spieker (1965) suggested that vascular erosion of an artery results from pounding of the vessel against the inferior edge of the tracheostomy tube placed low in the trachea. This mechanism was suggested by Korte (1879) as the cause of erosion of an innominate artery. All the cases reported by Silen and Spieker (1965) had low-placed tracheostomies. Apparently the two cases reported by Davis and Southwick also had low-placed tracheostomies.

It is obvious that low tracheostomy is the main cause of this fatal complication. Inadvertent low tracheostomy is more common than is usually appreciated. When the neck is hyperextended to facilitate exposure of the trachea, several tracheal rings are moved upward causing the tracheal incision to be made lower than is realized. To prevent this complication, tracheostomy should be made no lower than the third ring. However, occasionally the identical situation could occur with normally placed tracheostomy openings when the innominate artery is situated in an abnormally high position. Davis and Southwick suggested that a soft tube of plastic or rubber is less likely to invade vessels but such tubes have several disadvantages and have been abandoned in many clinics (Yanagisawa and Kirchner, 1964).

If haemorrhage is not massive, the bleeding could be temporarily controlled by inflation of the cuff of the endotracheal or tracheostomy tube. In most cases, this would allow enough time to perform emergency thoracotomy. However, in this case, the authors were unable to control the bleeding by inflation of the cuff of the tracheostomy tube. Since treatment of massive haemorrhage in the respiratory tract is extremely difficult, the best remedy is prevention of the erosion.

Although pulsation of the endotracheal tube by the adjacent artery was not observed, this pathognomonic sign should be looked for, especially in patients with low tracheostomy. If pulsations are observed, the tube should be replaced with a shorter one until pulsations have disappeared. This rare and fatal complication could be prevented by avoidance of low tracheostomies and by use of short and properly curved tracheostomy tubes.

REFERENCES


HEMORRHAGIE FATALE DE L'ARTERIE INNOMINEE APRES TRACHEOSTOMIE: RAPPORT D'UN CAS

SUMMARY
Un cas d'hémorragie fatale de l'artère innominée s'est produit une semaine après une trachéotomie.

BOOK REVIEWS


The word "synopsis" has come to mean a summary. In fact, however, the original Greek words mean "looking at everything together" and it is really this sort of synopsis that Dr. Lee and Dr. Atkinson have provided for anaesthetists. Their presentation is, of course, still synoptic but within the 876 pages of their book they have covered most matters of interest to students of the specialty. The recent growing points of anaesthesia have been dental anaesthesia, resuscitation, and intensive therapy units, and short chapters have been added to draw attention to fundamental work in these fields. There is, also, a most interesting and valuable new chapter on anaesthesia in abnormal situations such as under unusual ambient pressure (high altitudes and hyperbaric chambers), in field situations, and not least a problem which besets every anaesthetist at one time or another, anaesthesia in the dark.

The authors are to be congratulated, too, on another major change which adds greatly to the value and a little to the bulk of the book. In previous editions, references were printed for only a limited number of the statements, though, as the reviewer himself can testify, Dr. Lee was always willing and able to give anyone who wrote for it a reference to any specific point. In the present edition the number of references has been greatly increased and this has added a new aspect to the usefulness of the Synopsis. For it is now a most adequate starting point for reading on any aspect of anaesthesia. It is particularly helpful in relation to the history of the specialty.

Once again, therefore, we commend this excellent volume, not only to those studying for higher examinations but also to every anaesthetist who wishes to have on his bookshelf an exhaustive work of reference.

A. R. Hunter


This is the sixth volume in a series which has been appearing since 1961. Earlier contributors have been drawn from both sides of the Atlantic though the present work is of purely European authorship, comprising two review articles and an experimental report. Bengmark outlines the present state of liver surgery including the indications, technique and anatomical basis of liver resection. Subsequent regeneration and the accompanying biochemical disturbances are described. Other possible ways of treating liver tumours, such as de-arterialization and intra-arterial infusion, are also discussed. The whole subject is exhaustively covered in 48 pages of lucid, concentrated writing followed by 12 pages of bibliography. Gruenagel then describes some promising experiments on dogs, suggesting that intrapulmonary transposition of the spleen may offer a satisfactory way of lowering raised portal venous pressure. The last section, by Bergentz, deals with fat embolism. The many puzzles and paradoxes of this condition are fairly presented and both theoretical and clinical aspects of the subject are brilliantly summarized. Altogether, the book is a model of completeness and lucid compression.

Roger Brearley