Left vocal cord paralysis after mobilisation of the internal mammary artery

ROBERT R JEFFRY, BRIAN M FABRI, MARK A FOX

From the Regional Adult Cardiothoracic Unit, Broadgreen Hospital, Liverpool

ABSTRACT A case of vocal cord paralysis following mobilisation of the internal mammary artery is described. Of the various possible mechanisms, the most likely in this case is damage to the vagus nerve in the root of the neck, where it passes close to the origin of the internal mammary artery. Diathermy may have caused the damage, and this may be avoided by using a disposable automatic clip applier on any branches of the mammary artery in the area.

The left internal mammary artery is widely used in suitable patients as the conduit for coronary revascularisation. We report a case of left recurrent laryngeal neuropraxis that followed mobilisation of the left internal mammary artery.

Case report

A 47 year old manual worker was admitted for coronary revascularisation. He had sustained two previous myocardial infarctions and had an eight year history of severe angina, which persisted despite treatment with nifedipine, atenolol, and isosorbide dinitrate. He had never had surgery or an anaesthetic previously. On physical examination there were no abnormal findings and all peripheral pulses were intact. The chest radiograph was unremarkable. Coronary angiography showed appreciable triple vessel disease. Left ventricular function was normal with an end diastolic pressure of 10 mm Hg. Five weeks before his projected date for surgery the patient sustained a further myocardial infarction, so surgery was postponed for 12 weeks. Repeat angiography showed further disease in one vessel only

After premedication with oral diazepam anaesthesia was induced with midazolam (0·1 mg/kg) and fentanyl (0·03 mg/kg) and paralysis with pancuronium (0·15 mg/kg). Laryngoscopy showed nothing remarkable and a 9·5 mm low pressure, cuffed endotracheal tube (Portex Profile) was inserted without difficulty. Anaesthesia was maintained with isoflurane in an air-oxygen mixture. Access to the central venous system was by cannulation of the right internal jugular vein.

The left internal mammary artery was mobilised before

Address for reprint requests: Mr R R Jeffrey, RACTU, Broadgreen Hospital, Liverpool L14 3LB.

Accepted 1 September 1988

heparinisation from its origin at the left subclavian artery to its bifurcation with low current diathermy. Exposure was facilitated with a Favoloro sternal retractor. The internal mammary artery was anastomosed to the left anterior descending artery with 7/0 prolene and reversed saphenous vein was used for a further two grafts.

His immediate postoperative condition was good and a postoperative chest radiograph showed that the end of the endotracheal tube was just above the tracheal bifurcation. After an atraumatic extubation 16 hours after operation the patient was noted to be hoarse with a bovine cough. He developed a chest infection and sputum culture grew Haemophilus influenzae. Indirect laryngoscopy on the 11th postoperative day showed paralysis of the left vocal cord. His chest infection responsed to appropriate antibiotics and physiotherapy. After five weeks his voice was stronger and repeat laryngoscopy showed normal movement of both vocal cords.

Discussion

Vocal cord paralysis is rare after surgical procedures that do not affect the neck or the course of the recurrent laryngeal nerves. We are aware of one case report of cord paralysis after mobilisation of both internal mammary arteries.

Ellis and Pallister² have proposed that a pressure neuropraxia can occur as a consequence of inflation of the cuff of the endotracheal tube in the larynx rather than the trachea. As the postoperative chest radiograph confirmed that the end of the endotracheal tube was just above the carina this explanation would not be tenable in our patient. Stanley et al³ showed that nitrous oxide diffuses into the cuff of the endotracheal tube more rapidly than nitrogen diffuses out and suggested that this may cause increased pressure in the cuff, which would be transmitted to the mucosa of the larynx. Nitrous oxide was not given to our patient and an endotracheal tube with a low pressure cuff was used, so pressure neuropraxia from the tube cuff is a very unlikely explanation for the vocal cord paralysis in this case.

Right vocal cord paralysis and Horner's syndrome has been reported after internal jugular venous cannulation, but no attempt at venous cannulation was made on the left side of our patient's neck.

We think that the most likely cause of the vocal cord paralysis in our patient was damage to the vagus nerve in the root of the neck where it passes close to the origin of the internal mammary artery from the subclavian artery.⁵ Diathermy may have damaged the nerve and we now deal

Jeffry, Fabri, Fox 942

with any branches of the mammary artery in this area with a disposable automatic clip applier (Surgiclip, Auto Suture

References

- 1 Phillips TG, Green GE. Left recurrent laryngeal nerve injury following internal mammary artery bypass. Ann Thorac Surg 1987;43:440.
- 2 Ellis PDM, Pallister WK. Recurrent laryngeal nerve palsy and

- endotracheal intubation. J Laryngol Otol 1975;89:823-6.

 3 Stanley HT, Kawamura R, Graves C. Effects of nitrous oxide onto volume and pressure of endotracheal tube cuffs. Anesthesiology 1974;41:256-62.

 4 Davis P, Watson D. Horner's syndrome and vocal cord paralysis as a complication of percutaneous internal jugular vein catheterisation in adults. Anaesthesia 1982:37:587-8. eterisation in adults. Anaesthesia 1982;37:587-8.
- 5 Romanes GJ. The peripheral nervous system. In: Romanes J, ed. O Cunningham's Textbook of Anatomy. 11th ed. London: Oxford University Press, 1972:730.