Paralysis of the Circumflex Nerve Following General Anesthesia for Laparoscopy

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The following is a report of an injury to the circumflex nerve which occurred during general anesthesia for laparoscopy. A review of the literature has failed to reveal a prior report of this specific injury.

REPORT OF A CASE

A 28-year-old woman, mother of three children, was admitted to the hospital for laparoscopic sterilization. Her general condition was excellent and she had had no history of previous illness. Anesthesia was induced with thiopental, 400 mg, and maintained with nitrous oxide—oxygen—halothane, 0.5 per cent, and pancuronium, 6 mg. The patient was in a supine position with 15-degree head-down tilt at the request of the surgeon. The arms were taped at right angles to the body using padded arm supports. An angled iron frame was fixed to a bracket just behind the right shoulder to keep the sterile drapes clear of the patient's face. The operation was finished within half an hour.

An hour after operation the patient was sent back to the ward, where upon arrival she complained that she could not lift her right arm. On examination, the patient was unable to lift her right arm away from the body; could flex the elbow joint only with difficulty, and had an area of paresthesia and loss of sensation on the lateral side of the upper arm. There was flattening of the shoulder and pronounced prominence of the acromion.

Three days postoperatively the patient underwent neurologic and electromyographic examination. Complete paralysis of the deltoid muscle with loss of sensation over the lower part of the deltoid, coinciding with the normal cutaneous distribution of the circumflex nerve, was found. All the other brachial muscles appeared to be intact and showed no sign of weakness. The biceps and triceps jerks were present and symmetrical on the two sides. No other neurologic disturbances could be detected.

The motor nerve conduction velocities of the peripheral nerves of the right arm were all in the normal range. The biceps muscles and other brachial muscles showed normal action potentials 4–10 msec and normal amplitude to 1,500 μV. The right deltoid muscle showed a slight increase of insertion activity and no sign of action potentials.

Treatment consisted of daily physiotherapy and local electrical stimulation. Electromyography 15 days later showed evidence of rapid reinnervation, and by two months innervation was completely normal. Gradually, abduction of the arm to 90 degrees became possible, with atrophy evident only in the posterior part of the deltoid muscle.

DISCUSSION

The time clapping between the end of the operation and the onset of symptoms and signs of neural damage strongly suggests that the lesion occurred while the patient was on the operating table. The lesion occurred during well controlled anesthesia, in which careful attention was given to limb positioning. Lesions of this nature, occurring after operation, are not uncommon. The use of muscle relaxants, techniques of induced hypothermia, or hypotension seems to increase the incidence of such complications.1 Neurologic and electromyographic examination excluded the possibility that the problem was caused by an upper brachial plexus lesion. The commonest neurologic complication met with in anesthesia is a complication of the circumflex nerve.

Based on the above-mentioned facts, we came to the conclusion that the towel support positioned behind the patient's shoulder must have exerted too much pressure on the nerve, despite the presence of shoulder supports. The circumflex nerve probably took a more vulnerable course than usual. Cases of localized paralysis involving single ulnar, median, lateral popliteal, femoral and sciatic nerves have been described, but we can find no mention in the literature of injury to the circumflex nerve occurring as a complication of general anesthesia.2–5

REFERENCES

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Sodium Nitroprusside-induced Improvement in Cardiac Function in Association with Left Ventricular Dilatation

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Patients with severely dilated ventricles occasionally have difficulty generating adequate cardiac output after extracorporeal circulation has been discontinued following open-heart surgery. The low cardiac output is partly due to the physiologic and biochemical alterations imposed by the extracorporeal circulation, the trauma of operation, and the anesthetic agents. However, in dilated ventricles, the abrupt change in geometric dynamics following correction of the causative defect may play a more important role in decreasing the cardiac output. The dimensional changes brought about by correction of the defect are important because of Laplace’s relation, in which pressure is directly related to wall stress and inversely related to the radius of the intraventricular volume. The latter mechanism is especially important when the dilatation is the result of pre-existing mitral insufficiency.

In compensated mitral insufficiency, the values for peak systolic wall stress and the ejection fraction compare favorably with those found in normal hearts. There is a marked difference in forward stroke volumes, since in mitral insufficiency, a fraction of stroke volume regurgitates back into the left atrium. This regurgitant fraction allows the ventricular volume to be decreased. With this smaller volume, the ventricular wall stress can generate sufficient pressure to overcome the afterload and eject an adequate stroke volume into the aorta.

When the regurgitant valve is replaced with a competent valve, the ventricular volume can no longer be vented into the left atrium. As a result of the large volume (radius), the peak systolic wall stress may not be able to generate enough pressure to eject an adequate stroke volume.

REPORT OF A CASE

The patient, a 56-year-old woman weighing 44 kg, had severe mitral insufficiency as a result of chronic rheumatic heart disease. She was intolerant to exercise and completely incapacitated. Physical findings included marked generalized cardiomegaly and pansystolic murmur. The electrocardiogram revealed atrial fibrillation with frequent premature ventricular depolarizations. The patient’s medications were digoxin, furosemide, propranolol, and potassium. Cardiac catheterization revealed: right atrial pressure 7 torr; left atrial pressure 28 torr—V waves to 36 torr; right ventricular pressure 60/8 torr; left ventricular pressure 110/15 torr; pulmonary arterial pressure 60/25 torr; systemic arterial pressure 110/80 torr; pulmonary vascular resistance 329 dynes/cm²/sec-1; systemic vascular resistance 3,670 dynes/cm/sec-1. Cardiac output (Fick) was 1.7 l/min, with a cardiac index of 1.2 l/min/m². Angiography demonstrated mitral-valve regurgitation with a large left atrium. The left ventricle was dilated and hypertrophied, with normal filling compliance but poor contractility. Localized areas of dyskinesia could not be demonstrated. The coronary arteries were normal.

The patient was fully informed and consented to mitral-valve replacement. Anesthesia was induced and maintained with morphine sulfate (1.5 mg/kg) and diazepam (0.5 mg/kg). Muscle relaxation was achieved with pancuronium bromide, and ventilation was mechanically controlled. 

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