MEDICAL MEMORANDA

Extensive Neurological Damage after Cannulation of Internal Jugular Vein

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Internal jugular vein cannulation is reputed to be a safe procedure. The only neurological complication reported after it is a Horner's syndrome (Parikh, 1972). We report a case in which lesions of the left cervical sympathetic nerve; the ninth, tenth, eleventh, and twelfth cranial nerves; and the anterior branches of the second, third, and fourth cervical nerves occurred after left internal jugular cannulation.

Case Report

A 64-year-old man with an anaplastic carcinoma of the bladder was admitted to hospital for cystectomy. Preoperative examination of the patient showed no neurological abnormality.

As heavy blood loss was expected during the operation an arterial line was inserted into his left radial artery, an intravenous infusion into his right arm, and a central venous line into his left internal jugular vein. The latter consisted of a 16-gauge Medicut needle placed in the left internal jugular vein by the elective method described by English *et al.* (1969). This was then attached to a central venous pressure recording line containing 5% dextrose solution.

During the operation severe haemorrhage occurred, and eight litres of a mixture of blood, plasma, and dextran were given through the arm. Also 100 ml of 8·4% sodium bicarbonate and 10 ml of 20% calcium chloride were given through the jugular vein, which was used for the rest of the operation to monitor central venous pressure (Briscoe, 1973).

The transfusion in the neck was continued until the next day when a large swelling was noticed and the cannula was therefore removed. Intravenous tetracycline and 200 ml of 10% mannitol were given during this period but it is not certain through which line.

The patient's postoperative progress was marred by an ileus during the first week, and he was not given fluids by mouth for 10 days. He was then found to have some difficulty in swallowing and it was noticed that he had a left Horner's syndrome.

More neurological abnormalities appeared during the next week. A barium swallow in the third week showed obstruction at the level of the pyriform fossa and some spill of the barium into the trachea. Neurological examination now showed evidence of lesions of the left ninth, tenth, eleventh, and twelfth cranial nerves, and of the anterior primary divisions of the left second, third, and fourth cervical nerves; the left Horner's syndrome was still present.

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In the fourth week the patient developed renal failure followed by bronchopneumonia and died.

Necropsy (Dr. R. C. B. Pugh) was limited to the head and neck at the request of the patient's relatives. This showed a thickened left sternomastoid muscle with necrotic patches and scarring deep to it, closely related to the lower two-thirds of the left internal jugular vein. A few crystals were seen in the scarred region. No metastases were found in the brain, skull-base, or neck, but some cerebral oedema was noted.

Sections through the scarred region showed necrosis of muscle, fat, and large nerves, with a surrounding inflammatory reaction but with no evidence of bacterial infection. The necrosed tissues adjoined a plaque of thrombus on the wall of the internal jugular vein. There were no iron deposits to suggest that a large haematoma had been present.

Comment

The problem raised by this case is how such extensive neurological damage could result from an intravenous infusion in the neck. The injured nerves all pass close to the left internal jugular vein and necropsy confirmed that necrosis had occurred in this area.

There was no evidence that damage had been caused directly by insertion of the needle. It appeared, therefore, that injury had been due to pressure from a haematoma or extravasated fluid, or to chemical damage from this fluid and the drugs contained in it.

Haematomata occasionally may produce peripheral nerve palsies, and recovery may be delayed for days or weeks after subsidence of the swelling (Parkes, 1945), presumably because of reversible structural damage to the compressed nerve fibres (Fowler *et al.*, 1972). Nevertheless, in this case the delay in onset of the neurological abnormalities and the absence of iron deposits in the neck suggest that compression by a haematoma was not a major factor.

The most important contribution to the damage was probably a direct chemical action of the drugs and fluids on the nerves. The swelling in the neck suggests that the cannula became at least partially dislodged from the vein, and that fluids and drugs passed into the tissues of the neck causing chemical damage to the neighbouring tissues. This mechanism would be in keeping with the necropsy findings of local muscle and nerve necrosis and of crystals at the site of cannulation. The actual substance was not identified but mannitol and tetracycline are the two most likely offenders.

We suggest that it is advisable to avoid injecting drugs into the neck veins unless the catheter used is long enough to prevent any danger of dislodgement. It is also important to realize that extensive neurological damage can result from local injury after cannulation of the internal jugular vein.

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