Neurologic complications of open heart surgery

Computer-assisted analysis of 531 patients

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Neurologic problems occurring in patients undergoing open heart surgery are infrequent, but nevertheless tangible. Problems have included prolonged "metabolic" encephalopathies, cerebrovascular infarcts, seizures, peroneal palsies, and brachial plexus injuries, to cite the main examples. Although there is a considerable literature concerning the neurologic findings associated with cardiac valve surgery and some data are available on the subject of the neurologic phenomenology associated with cardiac transplantation, there is virtually no information on the subject of neurologic difficulties encountered in the patient population undergoing coronary artery bypass graft procedures.[1–6] With the large experience in open heart surgery at The Cleveland Clinic Foundation, it was decided to study prospectively a series of 531 patients who underwent various open heart procedures in an effort to identify the types of neurologic difficulties encountered, their frequency, and risk factors for neurologic compromise. The patients were studied by neurologists before and one or more times after open heart surgery. Four hundred fifty-one preoperative, operative, and postoperative variables were assessed. In the myocardial revascularization group, 380 patients underwent bypass grafting for the first time and 38 for the second time.

Beginning on the fourth postoperative day in the myocardial revascularization group, new central nervous system (CNS) deficits were found in 68 of 418 patients (16%). However, only nine patients (2%) experienced significant functional debilitation. Twenty-two of 68 (37%) of all CNS complications were focal, and in ten (45%) of these a possible cause was documented. All remaining CNS complications, 46 of 68 (68%), were nonfocal or diffuse encephalopathies and in only six (13%) of these was a possible cause identified. There were seven deaths (1.7%) in the series of 418 patients with an associated severe CNS deficit in three.

Also in the revascularization group, 55 of 418 patients (13%) developed 64 new postoperative peripheral nervous system deficits. Of these complications, 28 of 64 (44%) involved the upper extremities, and 21 of these were injuries to the brachial plexus. There were 24 of 64 (38%) lower extremity mononeuropathies involving the saphenous nerve in 16 and the peroneal nerve in eight. Other complications included prolonged hoarseness or documented vocal cord paresis or both in seven, a peripheral seventh cranial nerve deficit in one, an isolated Horner's syndrome in one, and prolonged singultus possibly related to phrenic nerve trauma in four. Although a number of these problems proved initially disturbing to patients, follow-up data thus far indicate that most were transitory.

In the total group of 531 patients (including patients who had undergone valve surgery) 26 (5%) sustained injury to the brachial plexus (alluded to above), and in 19 of 26 (73%) of these the lesion involved the lower trunk, seven on the left and 12 on the right. An electromyogram confirmed the clinical impression in 12 patients, but electrical findings were inconclusive in two others.

In 18 of 26 patients (69%), the side of the plexus lesion correlated with the side of the internal jugular vein cannulation. Because of the preponderance of lower trunk lesions, the anatomic proximity of the lower trunk to the internal jugular vein, and the significant correlation of ipsilateral vein cannulation and plexus injury, traumatic cannulation may be a mechanism of plexus injury. The resulting clinical syndrome of severe pain and dysesthesias lasting for weeks or months may thus be preventable.

References

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