Brachial plexus injury after cardiac surgery

The role of internal mammary artery preparation: A prospective study on 1000 consecutive patients

Brachial plexus injury is a typical complication after median sternotomy. A prospective study was performed on 1000 consecutive patients to determine whether preventive actions, including lower position and least possible opening of the sternal retractor, help to reduce the complication rate. Twenty-seven patients were observed with postoperative brachial plexus injury. Nerve conduction measurements and electromyography were performed. Patients without preparation of the internal mammary artery had a complication rate of less than 1%, whereas the complication rate of those patients with preparation of the internal mammary artery was as high as 10.6%. The main symptoms were continuous pain and motor and sensory disturbances. Most frequent were lesions corresponding to the roots C8-T1. Six patients had Horner's syndrome; three had ptosis only with no other signs of Horner's syndrome. Symptoms persisted in eight patients more than 3 months after the operation, and one patient still had intractable pain. Increasing use of internal mammary artery grafts in coronary artery bypass demands measures to protect the brachial plexus.

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Brachial plexus lesion is a typical complication of cardiac operations necessitating median sternotomy. In several published series prevalence varied between 2% and 38% (Table I). Several pathophysiologic concepts and numerous suggestions to reduce the complication rate have been discussed. Most authors agree that the technique of sternotomy and the position and the extent of opening the sternal retractor determine the prevalence of complications by causing a mechanical lesion of the brachial plexus. A prospective study was performed on 1000 patients to check whether these measures are sufficiently helpful to reduce the complications.

Methods

One thousand consecutive adult patients who were undergoing cardiac operations necessitating median sternotomy and extracorporeal circulation were evaluated before and after the operation for symptoms and signs of neurologic deficits related to brachial plexus dysfunction. After specific neurologic training (H.M.-V.) the preoperative assessment was performed (C.F.V. and I.C.). The neurologic examination consisted of a detailed history regarding upper extremity pain, paresthesia, numbness, and weakness and of a thorough examination of all motor and sensory functions supplied by the brachial plexus. Anamnestic and actual signs of central lesions (transient ischemic attacks, periods of hemiplegia) and syncopes were also considered. No patient was included in the study without ultrasonographic preoperative assessment of the cerebral blood supply.

In cases of cannulation of the arteriae radiales the respective arm was abducted; otherwise both arms were adducted. No roll was placed under the shoulders. Internal jugular vein cannulation was generally performed, predominantly on the right side. All operations were performed with bubble oxygenators, aortic and bicaval cannulation, and moderate hypothermia (26°C to 30°C). During the course of the operation an Ankeney retractor placed at a lower than standard position with the least possible opening (9 to 12 cm) was used, unless the internal mammary artery (IMA) was prepared. During that phase of the operation we used the Favaloro retractor. Both blades were used, one
place at the manubrium and the other at the lower part of the body of the sternum. The IMA pedicle was mobilized by sharp dissection immediately after splitting the sternum. A pedicle was dissected consisting of IMA, internal mammary vein, fat, muscle, and pleura. Preparation began in the sixth intercostal space and continued up to the first rib. The dissection was complete when the first intercostal artery was identified and occluded with clips on both sides, artery, and chest wall. After heparinization the pedicle was transected at the distal part, the diameter of the IMA was measured, and the vessel was allowed to bleed freely a few seconds. Harvesting the IMA took 15 to 25 minutes. Only the left IMA was used. After complete mobilization of the pedicle the Favaloro retractor was replaced by an Ankeney retractor. In all cases we tried to achieve symmetric spreading of the sternal halves.

Preoperative, intraoperative, and postoperative data including duration of operation and measurements of extracorporeal circulation (240 items/patient) were analyzed. Within the first 3 days after extubation of the trachea each patient was reexamined according to the preoperative protocol. Whenever a difference between the preoperative and the postoperative state was found, the patients were reviewed with a consulting neurologist to examine the relationship of the assumed neurologic deficits to the brachial plexus. Whenever symptoms persisted for more than 3 days, the patient was examined neurophysiologically. Electromyographic examinations and nerve conduction studies were performed to identify the anatomic pattern of the lesion and to exclude entrapment of an individual nerve in the arm. These examinations were performed 3 to 8 days after the operation and were repeated about 4 weeks postoperatively. Cranial computed tomography was performed to exclude additional central lesions. Only after this set of examinations was completed was the diagnosis of brachial plexus lesion attributed to the patient.

Preoperative exclusion criteria included emergency operations, preoperative central neurologic dysfunction, amputation or mechanical dysfunction of the arm, polyneuropathy, and inability to understand the German language. Postoperative exclusion criteria included a prolonged intubation period with reintubation, uncooperative patients (postoperative psychiatric disorders), death within 14 days, and postoperative central neurologic dysfunction. The follow-up period included a 3-month interval. For statistical comparisons parametric (Student's t) and nonparametric (χ²) tests were used appropriately.

Results

Of the 1000 patients entered in the study, the majority underwent coronary artery bypass grafting (CABG) (719), whereas heart valve replacements (205), combined heart valve replacements plus CABG (47), and miscellaneous procedures (29) defined the other groups. Twenty-eight of these patients showed transient and unspecific symptoms such as heaviness and weakness of the arms or intermittent numbness. Whenever these symptoms of a predominantly sensory character disappeared completely within 3 days, the diagnosis of a plexus lesion could not be substantiated and no further analysis was performed (Tables II and III).

The 33 patients with persistent neurologic symptoms underwent careful neurologic and neurophysiologic analysis. Twenty-seven patients had a brachial plexus lesion, whereas it was seen in four of the 205 patients with single valve replacements and once in the combined valve replacement/CABG group.

Of the 719 patients with CABG 22 sustained a brachial plexus lesion, whereas it was seen in four of the 205 patients with single valve replacements and once in the combined valve replacement/CABG group.

Of the CABG patients, 198 received the IMA as
Table II. Basic data of patients

<table>
<thead>
<tr>
<th>Factor</th>
<th>With BPL (n = 27)</th>
<th>Without BPL (n = 973)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>58.31 ± 2.76</td>
<td>58.53 ± 0.36</td>
<td>0.92</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>77.80 ± 2.51</td>
<td>73.70 ± 0.61</td>
<td>0.27</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>175.39 ± 2.88</td>
<td>169.82 ± 0.42</td>
<td>0.03</td>
</tr>
<tr>
<td>Duration of ECC (min)</td>
<td>96.76 ± 5.48</td>
<td>100.39 ± 1.21</td>
<td>0.62</td>
</tr>
<tr>
<td>ACC (min)</td>
<td>61.34 ± 3.88</td>
<td>63.67 ± 0.89</td>
<td>0.67</td>
</tr>
<tr>
<td>Reperfusion (min)</td>
<td>32.16 ± 3.41</td>
<td>34.01 ± 0.62</td>
<td>0.62</td>
</tr>
<tr>
<td>Duration of OP (min)</td>
<td>264.53 ± 12.62</td>
<td>242.05 ± 2.21</td>
<td>0.09</td>
</tr>
</tbody>
</table>

A statistically significant difference (p < 0.01) between the groups was not found for any of the measurements. BPL. Brachial plexus lesion; ECC, extracorporeal circulation; ACC, aortic crossclamp time; OP, operative procedure.

Table III. Type of operation and occurrence of postoperative neurologic symptoms of the upper extremity*

<table>
<thead>
<tr>
<th>Operation</th>
<th>n</th>
<th>Sensory deficit &lt;3 days†</th>
<th>Brachial plexus lesion‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>CABG</td>
<td>521</td>
<td>4 (0.8%)</td>
<td>1 (0.2%)</td>
</tr>
<tr>
<td>CABG + IMA</td>
<td>198</td>
<td>15 (7.6%)</td>
<td>21 (10.6%)</td>
</tr>
<tr>
<td>VR</td>
<td>205</td>
<td>6 (2.9%)</td>
<td>4 (2.0%)</td>
</tr>
<tr>
<td>Comb</td>
<td>47</td>
<td>3 (6.4%)</td>
<td>1 (2.1%)</td>
</tr>
<tr>
<td>Misc</td>
<td>27</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>1000</td>
<td>28</td>
<td>27</td>
</tr>
</tbody>
</table>

The absolute numbers of patients and the proportion of patients with respect to the operative treatment group are given. CABG, Coronary bypass graft; VR, valve replacement; Comb, valve replacement combined with CABG; Misc, miscellaneous operative procedure.

*Frequency of patients with fluctuant postoperative disturbances after median sternotomy and those with definite brachial plexus lesion.
†Patients with fluctuant symptoms of the upper extremity, which might be caused by irritation of the brachial plexus. Because these symptoms disappeared completely within 3 days, no further neurophysiologic examination regarding the origin of the disturbances was performed.
‡Patients with a definite brachial plexus lesion and the respective neurophysiologic assessment.

bypass graft. However, 21 of the 23 patients (including 1 patient with combined valve replacement/CABG) with brachial plexus lesion after CABG had an IMA preparation. Thirteen of these 21 patients had symptoms ipsilateral to the IMA preparation, five on both sides and the remaining three on the opposite side (Table IV).

The main symptoms (in decreasing order) were sensory deficits (27 patients), motor deficits (21 patients), pain (10 patients), complete Horner’s syndrome (6 patients), and ptosis (3 patients). Twenty-one patients had a lesion of the lower trunk and/or medial cord of the brachial plexus (C7-T1); in 6 patients the symptoms were attributable to an upper trunk lesion. Neurophysiologic analysis did not reveal signs of an isolated peripheral nerve lesion in the arm of any patient.

The severity of the symptoms varied. Mild symptoms did not impede the patient’s motor activity and were observed in 10 patients. Moderate symptoms occasionally combined with pain were found in 10 patients. Severe symptoms included pain and/or motor disturbances that dramatically impeded the patient’s ability to perform basic daily life activities such as eating or writing (seven patients).

There was no correlation between preoperative, intraoperative, and postoperative data (age, preoperative drug administration including platelet aggregation inhibitors, hypertrophy of the left side of the heart, internal jugular vein cannulation, abduction of the arm, bypass time, minimal temperature, minimal flow, minimal pressure) of the patient and the prevalence of brachial plexus lesions.

Complete recovery required 1 month in four patients, 2 months in seven patients, and 3 months in eight patients. After 3 months symptoms still persisted in eight patients. One patient continued to have intractable pain and was referred for neurosurgical intervention.

Discussion

Two groups of patients with a different risk of brachial plexus injury can be distinguished on the basis of our analysis. The first group without preparation of the IMA had a remarkably low complication rate of less than 1% (6/802), whereas patients with IMA preparation displayed a complication rate of 10.6% (21/198). This suggests that the measures to prevent brachial plexus damage (lower position and least possible opening of sternal retractor) worked for one group but not for the other. Our observations that median sternotomy predominantly affected the lower parts of the plexus and that most lesions recovered spontaneously over several weeks or months are in accordance with the literature.2–4, 11–14 However, in
some patients (eight in our study) symptoms persisted after 3 months. Thus the frequency and severity of this complication of median sternotomy requires preventive measures.

Postoperative ulnar nerve lesions are widely known\textsuperscript{15, 16} and also have been reported as a complication of operations with median sternotomy.\textsuperscript{6, 8, 17} Compression of the ulnar nerve at the elbow is thought to be the pathogenetic factor. According to the “double crush” hypothesis,\textsuperscript{18, 19} a mild subclinical cubital tunnel entrainment may be converted into a clinically overt ulnar neuropathy by a minor traction injury to the lower trunk of the brachial plexus during median sternotomy. Thus two relatively minor lesions, one proximal and one distal, neither of which by itself is sufficient to block axoplasmic transport below a critical level, may jointly produce a significant clinical deficit. In none of our patients, however, did electrophysiologic measurements reveal local compression of any nerve in the arm.

Symptoms of the 28 patients with transient sensory disturbances, which were not classified as brachial plexus lesion, may be at least partly caused by slight mechanical irritation of the plexus. This is corroborated by the data of Marganitt and colleagues,\textsuperscript{5} who electrophysiologically (F-wave latency) detected a subclinical brachial plexus lesion in 13 of 15 patients with median sternotomy without clinical signs of a brachial plexus lesion.

The factors involved in the pathogenesis of postoperative brachial plexus injury after cardiac operations are still under discussion. Graham and colleagues\textsuperscript{20} underscored the influence of the intraoperative position of both arm (abducted/adducted) and head (rotation of the head to opposite side). This finding was not replicated by other authors.\textsuperscript{6, 7, 13} Recently an intraoperative “hands-up” position was recommended as a preventive measure.\textsuperscript{11} In our patients positioning of the arm did not correlate with the prevalence of brachial plexus lesions. Further evidence against such a causal relationship can be derived from the fact that brachial plexus lesions resulting from malpositioning during operation predominantly involve the upper parts of the plexus.\textsuperscript{21-23}

Other groups\textsuperscript{3, 4} attributed lesions of the plexus to
catheterization of the internal jugular vein, which was not (in line with our experience) verified by other researchers. The fact that most of the lesions involved the segments C8 and T1 argues against catheterization-related pathogenesis. The few reports of brachial plexus lesions caused by catheterization of the jugular vein again described only damage to the upper parts of the plexus. In agreement with Sotaniemi, a specific influence of mode and duration of operation and extracorporeal circulation could not be found in our patients, although a dependence on hypothermia and operation time had been discussed in the literature in support of the concept of ischemic neuropathy.

Strong evidence exists that the techniques of incision and retraction of the sternum are crucial in the pathogenesis of brachial plexus injury after cardiac operations. The constituent nerve roots of the brachial plexus are fixed proximally at their points of exit from the vertebral canal. Distally the nerves are tethered to the axillary fascia. Any mechanical force that increases the distance between these points may cause a stretching injury of the nerves. Kirsh and colleagues conducted an autopsy study showing that excessively spreading the retractor pushed the clavicles into the retroclavicular space and rotated the first ribs upward. This increased the distance between the points of fixation of the nerves, and the brachial plexus was obviously stretched. In another autopsy study Vander Salm and colleagues demonstrated fractured first ribs penetrating the brachial plexus in 11 of 15 cadavers. Because the cervical sympathetic chain lies just medial to the fracture site, its injury (causing Horner's syndrome) can be explained by either a trauma resulting from the fractured rib and/or by fracture hematoma compressing the nerve. Vander Salm and colleagues showed that the risk of rib fracture can be minimized by opening the sternal retractor as little as necessary and by placing it as caudally as possible.

These pathogenetic concepts are supported by the most striking result of our study: the coincidence between IMA preparation as graft for CABG and the subsequent prevalence of brachial plexus lesions.

IMA preparation requires both a wide opening of the retractor and asymmetric traction to allow visualization of the costosternal junctions. The Favaloro retractor fulfills this requirement by constant traction of the left sternal half during the IMA preparation. However, this period of constant traction may lead to brachial plexus lesions. Vander Salm and colleagues showed that fractures at the costotransverse articulations occurred when the sternum was opened asymmetrically with the lesion at that side where the sternal half was higher. In those cases the lateral end of the rib fracture transfixed the lower trunk of the brachial plexus.

In our patients with a brachial plexus lesion x-ray studies did not show fractures of the first two ribs. However, this does not exclude fractures of the ribs being a pathogenetic factor. It has been reported that rib fracture, as detected by radionuclide bone scans, are a common complication after cardiac operations with median sternotomy, although routine roentgenograms did not detect them in any patient. It was hypothesized that mild symptoms related to injury of the brachial plexus may be caused by pressure or traction, whereas rib fractures associated with protrusion into the nerves may cause severe symptoms. The probability of such a pathogenetic mechanism is supported by the prevalence of Horner's syndrome resulting from a lesion of the cervical sympathetic chain, which is located near the costotransverse articulation of the first rib.

The striking difference between the complication rates of patients with (more than 10%) and without IMA preparation (<1%) argues in favor of the direct influence of the preparation technique. Using the Favaloro retractor during IMA preparation is a common and comfortable practice. Considering that IMA preparation takes about 20 minutes, constant traction during this period may well cause an injury. A less traumatic mode of IMA preparation with reduced traction, although less convenient, is therefore recommended. In contrast to the present series, Roy and colleagues and Tomlinson and associates found only a slight and nonsignificant difference in the prevalence of plexus injury for patients with and without preparation of the IMA. It is difficult to reconcile the differences between their results and our study. It may well be that the differences are the result of smaller patient groups or the fact that these authors accepted transient or uncharacteristic symptoms as evidence for a nerve injury without verifying them by the appropriate neurophysiologic measures.

The increasing frequency of IMA preparation for CABG stresses the need to be aware of this complication, which may lead to a long-lasting disability. In light of the present study and the literature we suggest the following preventive measures.

Exact median sternotomy should be performed. Caudal localization of the retractor should be attempted.

Constant traction on the sternal halves should be reduced.

Asymmetric traction should be avoided when possible.

Retractors designed for asymmetric traction (e.g., the Favaloro type) should be used with extreme caution.

Postoperative neurologic assessment should be performed in every patient to allow early detection and therapy of nerve lesions.
REFERENCES


