Costoclavicular Syndrome and the Sitting Position during Anesthesia

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Costoclavicular syndrome causing compression of the neurovascular bundle is a known entity. It can occur in sitting position during anesthesia. The following case report is an example.

REPORT OF A CASE

A 2-year-old female child was admitted to the hospital with right hemiparesis, wide-based gait, increased tone in the right lower extremity, and ataxia. Angiography and computerized axial tomography revealed a large midline tumor extending from the pineal region, over the collicular plate and into the superior vermis of the cerebellum. The patient was brought to the operating room for posterior fossa craniectomy and excision of the tumor, to be done while she was in the sitting position. After placement of proper equipment for monitoring, anesthesia was induced with thiopental, 5 mg/kg, and pancuronium, 0.1 mg/kg, iv. The trachea was intubated and anesthesia was maintained with halothane. A 20-gauge catheter was placed in the right radial artery. Another catheter was placed in the right subclavian vein and its position confirmed radiographically.

The patient was positioned in the sitting position with the neck flexed, the arms by the sides, and the elbows flexed. Her forehead was resting on a "cerebellar head rest," and the head was immobilized with a Mayfield skull lamp. Layers of gauze pieces were placed between the chin and the chest. The cervicotoracic spine was flexed to achieve maximum surgical exposure and comfort for the surgeons. After positioning of the patient, the arterial waveform dampened, with decreased amplitude of the brachial pulse on the right; yet the left radial pulse was unchanged. The radial arterial waveform and amplitude of the right brachial pulse by palpation improved in response to elevating the shoulders and placing towels under the right elbow. The dampening of the waveform was reproducible by removing the towels. Replacement of the towels under the right elbow again improved the amplitude of the pulse and the waveform of the radial artery. Subsequently, towels were also placed under the left elbow. The rest of the procedure was uneventful.

Fig. 1. The neurovascular bundle is compressed between the first rib and the clavicle when the shoulder is dropped downwards.

DISCUSSION

The association of movements of the shoulder joint with reduction or absence of arterial pulse and compression of the brachial plexus is a well-recognized phenomenon. This compression of the neurovascular bundle by the shoulder girdle is known as thoracic outlet syndrome. It includes a large number of syndromes variously described as cervical rib, hyperabduction, costoclavicular syndrome, scalenus anticus, subcoracoid pectoralis minor, first thoracic rib, etc.

In costoclavicular syndrome, backward and downward thrust of the shoulders (assumed by soldiers and back-packers) exerts pressure on the neurovascular bundle between the clavicle and first rib (fig. 1). In our patient, the pulse wave improved when the shoulders were elevated. Since no bony abnormality was evident on the radiogram, the diagnosis of costoclavicular syndrome was made. This syndrome was first described by Falconer and Weddell, in 1943, when they noticed excessive complaints of pain, numbness, congestion in the hands, and weakness of the arms that
soldiers developed while carrying backpacks for prolonged periods. The costoclavicular space is bounded anteriorly by the inner third of the first rib and posterolaterally by the superior border of the scapula. This space is narrowed when there are anomalies—congenital or acquired—of the clavicle, first rib, or a cervical rib. The narrowed costoclavicular space often leads to the costoclavicular syndrome. However, when the shoulders are drawn backwards, dampening of the radial pulse may result in certain normal individuals by compression of the artery between the clavicle and first rib (fig. 1). The treatment of this entity is weight reduction, and physiotherapy to strengthen the elevators of the shoulders, improve posture, and avoid hyperabduction. The majority of the patients will benefit from the above-mentioned conservative management, although a few patients need resection of the first rib to alleviate the symptoms.

Compression of the neurovascular bundle secondary to assuming the sitting position has not been reported before. We recommend that whenever a surgical procedure is to be performed with the patient in the sitting position, both the radial pulses be checked before and after positioning the patient. Even if the pulses are not dampened, the shoulders not be allowed to droop downwards, as there is always the possibility of compression of the brachial plexus without compression of subclavian artery, which could result in paresis or paralysis of the arm during a long surgical procedure.

REFERENCES

Hemorrhage and Cardiac Arrest during Laparoscopic Tubal Ligation
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Two cases of hemorrhage from aortic injury during laparoscopy were recently reported.1 The following case report deals with massive hemorrhage and cardiac arrest during a laparoscopic tubal ligation.

REPORT OF A CASE
A 38-year-old, 60-kg woman, ASA 1, was scheduled for a therapeutic abortion and laparoscopic tubal ligation at an outpatient facility. The fetus was estimated to be of 8 weeks' gestation. History and results of physical examination were otherwise unremarkable. Laboratory data included hemoglobin 12 g/dl and hematocrit 31 per cent.

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Following administration of atropine, 0.6 mg, im, and fentanyl, 0.1 mg, iv, 100 mg methohexital were given iv. Infusion of 0.2 per cent succinylcholine was started while the dilatation and curettage was performed. Following intubation of the trachea, anesthesia was maintained with 66 per cent nitrous oxide. Blood loss during dilatation and curettage was 200 ml. Vital signs remained stable, with blood pressure 100/60 torr and heart rate 75 beats/min, while the Veres laparoscopic needle was inserted and the abdomen was insufflated with carbon dioxide. The heart tones then became less audible. Nitrous oxide administration was discontinued and administration of 100 per cent oxygen was started. Blood pressure and pulse rate were unchanged. The laparoscope was introduced without problem; however, systolic blood pressure then decreased to 60 torr and heart rate increased to 100 beats/min. Neonephine, 0.1 mg, was given ip. The surgeon, when informed of the hypotension, saw no abnormality intradetrusorally through the laparoscope, and found no free blood or perforations of the uterine wall. The blood pressure and heart tones became unobtainable, and cardiorespiratory resuscitation was started, during which lactated Ringer's solution, 3,000 ml, was administered. After stabilization of vital signs, the patient was transferred to our hospital for observation.