Somatosensory evoked potentials are used for detecting such a complication in selected surgeries. Most reports of intraoperative nerve injuries due to malpositioning are limited to injuries to the peripheral nervous system, and there have been no previously reported cases of somatosensory evoked potential monitoring disturbance attributable to position-related cerebral ischemia in the park-bench position. We present the case of a patient with glioblastoma in the park-bench position whose somatosensory evoked potential waveforms disappeared after head and neck repositioning. A prompt diagnosis of this complication and elimination of the underlying cause led to the return of somatosensory evoked potential waveforms, and there was no relevant neurologic deficit at the end of the surgery.

Keywords: Somatosensory evoked potentials, park-bench position, neuropathy

Introduction

The park-bench position is a variation of the lateral position, wherein the dependent shoulder and arm are positioned outside the surgery table and a sling supports the independent arm. The patient's head may then be positioned at the extreme limits of rotation and flexion. This position is advantageous for approaching low-lying cranial lesions and provides the surgeon with access to the anterior brainstem and foramen magnum as well as the cerebellopontine angle for the resection of acoustic neuromas (1).

There are some reports of intra- or postoperative complications of surgeries, such as brachial plexopathy, cervical cord injury and massive tongue swelling, performed in the park-bench position. Somatosensory evoked potentials (SSEP) are used in selected high-risk brain surgeries.

Case Presentation

A 62-year-old man with ASA class 2 was scheduled for craniotomy and removal of a glioblastoma of the posterior horn of the left ventricle. After the induction of anaesthesia and intubation, in addition to standard monitoring, invasive blood pressure monitoring was applied. With the patient in the supine position, bilateral median nerve SSEP monitoring was applied. The peak-to-peak amplitudes of the primary cortical response N19/P22 complex and the latency were measured online and basal measurements were conducted. After placing the Mayfield clamp, the patient was repositioned to right the park-bench position. The amplitude and latency of the right and left median nerve SSEP were normal. Before starting the surgery, optimal surgical exposure was achieved by right-sided head rotation and flexion (Figure 1a). Three minutes after this maneuver, the left median SSEP waveforms abruptly disappeared, while the amplitude and latency of the right-side median nerve SSEP remained normal (Figure 2). A thorough investigation for discovering the cause of SSEP disturbance started. The vital signs were as heart 65/min, BP 110/65 mmHg and BT 36.3°C (vital signs on arrival were HR 82/min, BP 110/65 mmHg and BT 36.3°C). There was no evidence of the upper extremity ischemia nor of facial edema or cyanosis. Haemodynamic changes, disturbance of head and neck venous re-
turn and upper extremity malpositioning were eliminated as the cause of SSEP disturbance.

Head and neck malpositioning was considered to be the reason of SSEP malfunctioning. The lateral head flexion was reduced approximately 30°, and the head and neck position changed to the anatomical position with only the right-sided head rotation (Figure 1b). After 2 min, SSEP waveforms reappeared with the normal latency and amplitude (Figure 1). Surgery was performed, and craniotomy and tumor removal proceeded with no further disturbance in the SSEP waveforms.

Discussion

Peripheral nerve injuries are commonly attributed to improper positioning (2). SSEP is a valuable modality to monitor somatosensory pathways and to prevent cerebral ischemia in the operating room (3-5). In our patient, the sequence of events led us to identify cerebral ischemia due to the ipsilateral carotid artery compression as the cause of SSEP waveforms disappearance. The complete return of the SSEP waveforms after correcting the position of the head and neck corroborated that there was no permanent neurological deficit because of the short duration of malpositioning as the neurological exam after arousal revealed no deficit on the left extremities. To the best of our knowledge, to date, cerebral ischemia as a complication of the park-bench position has not been reported.

Conclusion

Extreme caution should be exercised to prevent cerebral ischemia in patients with the park-bench position. Our recommendations for preventing cerebral ischemia in the park-bench position are as follows:

1) Identification of the baseline head and neck range of motion during preoperative visit.

2) Close cooperation between anaesthesia and surgical teams to obtain the best surgical exposure with minimum head and neck lateral flexion and/or rotation.

3) Use of cerebral monitoring, such as SSEP or cerebral oximetry, for an early diagnosis of cerebral perfusion impairment.

Informed Consent: Written informed consent was obtained from patient who participated in this case.

Peer-review: Externally peer-reviewed.


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