Spinal Cord Stimulation Evoked Potentials during Thoracoabdominal Aortic Aneurysm Surgery

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Although monitoring of somatosensory evoked potentials elicited from stimulation of lower extremity peripheral nerves has been suggested as a method for assessing neural function during thoracoabdominal aortic aneurysm surgery, this technique has been reported to yield a large number of false positives. It was believed that direct stimulation of the spinal cord would eliminate some of the problems associated with peripheral evoked potentials. The present study compared in 18 patients the use of scalp recorded evoked potentials following stimulation of either the posterior tibial nerve via percutaneous needles or the spinal cord via an epidural electrode previously placed fluoroscopically. In 10 patients in whom distal bypass or shunt was not used, peripheral evoked potentials totally disappeared within 5–30 min of aortic clamping. Spinal cord stimulation evoked potentials disappeared permanently in 2 patients shortly after aortic cross-clamping; 1 died shortly after the procedure, and the other awoke densely paraplegic and died the next day. When distal perfusion was maintained by shunt or bypass, the disappearance of both peripheral and spinal evoked potentials accurately predicted the neurologic outcome of 1 paralyzed patient. Loss of spinal cord stimulation evoked potentials was found to be correlated with adverse neurologic outcome. Over the period of aortic clamping a gradual decrease in mean amplitude (50% at 45 min [P < 0.05]) and a 20% increase in mean latency time were observed. Maintenance of adequate distal perfusion may permit the use of peripheral evoked potentials in the assessment of spinal cord ischemia during aortic cross-clamping. Our results indicate that the persistence of spinal cord stimulation evoked potentials portends good neurologic outcome in this high-risk group of patients. (Key words: Complications: paralysis; spinal cord injury. Monitoring, spinal cord: somatosensory evoked potentials; spinal cord stimulation evoked potentials. Surgery: thoracoabdominal aortic aneurysm. Techniques, spinal: cerebrospinal fluid drainage; epidural electrode.)

PARAPLEGIA AND PARAPARESIS are among the most devastating and unpredictable complications of cross-clamping the descending thoracic aorta for the resection of thoracoabdominal aneurysms. Ischemia of the spinal cord is related to inadequacy of blood flow in the anterior spinal artery and may be further compromised by an acute increase in spinal fluid pressure during aortic cross-clamping.1-5 Improvements in operative techniques and in perioperative management have ameliorated overall mortality and morbidity, but the incidence of spinal cord injury remains substantial6-7 and is related most clearly with the extent of aorta requiring repair.

Ischemic damage to the spinal cord during aortic surgery commonly remains undetected for several hours after the procedure, pending return of spontaneous or voluntary movements. Any real-time assessment of spinal cord function during the operation might be valuable if used to guide therapy or alter surgical procedures. Cortical evoked potentials are signals produced by stimulation of visual, auditory, or other neural input to the brain that increasingly are being used to assess neural integrity in a variety of surgical procedures. Somatosensory evoked potentials elicited by posterior tibial nerve stimulation are used routinely for intraoperative monitoring of spinal cord function during spinal surgery.8 Rapid loss of somatosensory potentials evoked in the posterior tibial nerve occurs soon after aortic clamping9,10 but may be due to peripheral hypoperfusion, temperature changes, anesthesia, and other factors.11,12

The purpose of the present study was to determine the efficacy of posterior column evoked potential monitoring in predicting the development of subsequent paralysis or paresis in patients undergoing repair of thoracoabdominal aortic aneurysms. We used both peripheral somatosensory evoked potentials and spinal stimulation evoked potentials in parallel. The surgery was performed both with and without the assistance of distal perfusion using partial cardiopulmonary bypass or shunt.

Materials and Methods

The study was approved by the institutional committee on clinical investigation, and written informed consent was obtained from each patient.

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Eighteen consecutive patients scheduled for elective repair of large descending thoracic aneurysms or thoracoabdominal aneurysms were eligible to participate in the study. A distal bypass or shunt was used at the discretion of the surgeon. The decision to use distal bypass or shunt was unrelated to the patient’s medical condition; instead, it reflected a change in surgical practice undergone during the study period.

All patients had a baseline neurologic examination by a neurosurgeon who provided continuing assessment perioperatively and who was responsible for placing the epidural stimulating electrode.

Selective spinal angiography was performed preoperatively to demonstrate the point of origin, patency, and degree of supply to the cord of the “critical” intercostal or lumbar artery. Such an artery, arising from the aerysmal wall, signifies a markedly increased risk for postoperative paralysis. Selective spinal angiography was performed preoperatively to demonstrate the point of origin, patency, and degree of supply to the cord of the “critical” intercostal or lumbar artery. Such an artery, arising from the aneurysmal wall, signifies a markedly increased risk for postoperative paralysis.

Electrode placement for spinal cord stimulation evoked potential monitoring was adapted from routine methods of screening patients for implantation of permanent spinal cord stimulators for the management of intractable pain. On the evening prior to surgery, the patient was positioned prone on the fluoroscopy table. In sterile fashion and under local anesthesia, a 15-G Tuohy needle was advanced to and through the T12-L1 intralaminar space via a paramedian approach. Loss of resistance to a Seldinger wire signified entry into the epidural space. A monopolar stimulating electrode (Pisces Sigma 3483S, Medtronic, Inc., Minneapolis, MN) was advanced to the midline position at T9 under fluoroscopic guidance. The electrode was slowly withdrawn and positioned so as to produce symmetrical lower extremity paresthesias in a sciatic distribution (consistent with standard posterior tibial nerve peripheral stimulation); the electrode was then secured to the skin with a temporary suture. Stimulation parameters were adjusted to an amplitude that produced uncomfortable paresthesias in the awake, prone patient: at a pulse width of 200 µs and a pulse repetition rate of 50 per second, this was typically 6–8 mA (3–4 V, across a nominal electrode impedance of 500 ohms). These thresholds were tested again in the supine patient to obtain baseline reference values before surgery.

Before arrival in the operating room, all patients received morphine 0.15 mg/kg intramuscularly, diazepam (Valium) 0.1 mg/kg orally, and scopolamine 0.005 mg/kg intramuscularly.

Blood pressure was monitored continuously via radial and femoral arterial catheters. Central venous and pulmonary artery pressures were also continuously measured. Following sterile preparation, a 4-Fr whistle-tip ureteral catheter was advanced through a 14-G Tuohy needle 15 cm into the subarachnoid space at the L4–L5 interspace for drainage of cerebrospinal fluid during the procedure. Large-bore intravenous access was established, and fentanyl (25–30 µg/kg), midazolam, and pancuronium bromide were used for induction of anesthesia. In all patients a double-lumen endotracheal tube was inserted with fiberoptic guidance, and ventilation was monitored with O2 and CO2 analyzers and a pulse oximeter. Inhalational agents, isoflurane or enflurane, were administered as required in concentrations of 0.5–0.75 MAC to minimize their depressant effect on the signal quality of the evoked potentials. All cardiovascular parameters were recorded continuously on a nine-channel recorder (model 7758A, Hewlett-Packard).

The signal generation and averaging of the peripheral and spinal stimulation evoked potentials was performed using the Nicolet Compact Four Apparatus (Nicolet Biomedical, Madison, WI). For posterior tibial stimulation, sterile 23-G needle electrodes were placed immediately after induction. Measurements were performed at that time and repeated every 30 min until aortic cross-clamping. During clamping, the peripheral and spinal evoked potentials were performed at least every 10 min and often more frequently. For both spinal and peripheral evoked potentials, 256 stimuli were generated (3.9 per second) and averaged; the stimulus intensity was 20 mA for the posterior tibial nerve and 3–8 mA for spinal stimulation. In case of loss of the spinal stimulation evoked potentials, the stimulus intensity was gradually increased to a maximum of 20 mA. The recording cup electrodes were located on the forehead (Fpz), above the cervical spine (C2), and at the vertex (Cz) (fig. 1). The same recording montage was used for both posterior tibial and spinal stimulation, and all measurements reported were recorded from the head. All data were stored on magnetic disk for later analysis.

When spinal stimulation evoked potentials were noted to markedly decrease or disappear, measures were taken to improve spinal cord perfusion pressure. The surgical team was informed; blood pressure proximal to the upper aortic clamp was further increased to improve perfusion to the cord via cervical collaterals; and additional CSF was aspirated in an attempt to improve spinal cord perfusion pressure.

Fisher’s exact test was used in analyzing the relationship between loss of the spinal stimulation evoked potentials and postoperative neurologic outcome. The same method was also used for the peripheral somatosensory evoked potentials. An analysis of variance for repeated measures was used to evaluate the changes occurring in amplitude and latency time of the evoked potentials following aortic cross-clamping, compared to the preclamping values. Group means were compared by the method of least significant differences; P < 0.05 was considered significant.
SPINAL CORD EVOKED POTENTIALS IN AORTIC ANEURYSM SURGERY

None of the patients who underwent the selective spinal angiography suffered a complication or residual effect from the radiologic procedure. Evoked potential monitoring predicted all neurologic sequelae.

NO DISTAL PERFUSION (n = 10)

In the ten patients in whom distal perfusion was not used, the peripheral evoked potentials disappeared within 5–30 min of aortic cross-clamping (table 2) and did not reappear during the operation. In six patients the potentials disappeared within 5–10 min, in two within 15–20 min, and in another two after 30 min.

In six patients who were neurologically intact postoperatively, the spinal stimulation evoked potentials remained intact. In another patient who did well neurologically (indicated “+” in table 2), there were technical difficulties in obtaining the spinal stimulation evoked potentials, despite normal threshold in the test stimulation during placement of the electrode preceding day. Presumably, the electrode migrated to a very high threshold zone in the epidural fat layer and/or adjacent to a blood vessel (low impedance shunt). Two patients died before regaining consciousness. One died intraoperatively of uncontrolled bleeding, but spinal stimulation evoked potentials remained intact until systolic blood pressure decreased to less than 60 mmHg, whereas in the other patient, spinal evoked potentials were lost during aortic cross-clamping. In this patient, the “critical” intercostal artery was reattached to the graft, an action that was time-consuming and evidently unhelpful. A third patient awoke with a dense paraplegia but was otherwise alert. In this patient the aortic cross-clamp was released 2 min after the disappearance of the spinal evoked potentials, but the potentials did not reappear.

DISTAL PERFUSION (n = 8)

Of the patients in whom distal perfusion was used, the peripheral evoked potentials remained unchanged during aortic cross-clamping in seven. In these patients, spinal stimulation evoked potentials were not lost, and the patients were neurologically intact postoperatively. However, in one patient both spinal and peripheral evoked potentials were lost. This patient awoke with flaccid paraplegia and subsequently died on the fourth postoperative day of multiorgan system failure. Post mortem analysis of the spinal cord showed long sections of softening and edema from the midthoracic cord down.

The correlation between loss of the spinal stimulation evoked potentials and neurologic outcome in the 15 patients in whom the neurologic outcome could be evaluated was found to be significant (P < 0.01). In this small group of patients there were no false negatives, in that spinal

Results

The 18 patients who participated in the present study represent the highest risk groups of thoracoabdominal aortic aneurysm. All had type I or type III aneurysm, signifying substantial involvement of the thoracic and abdominal aorta. In 16 of the 18 patients, either one or both of two important risk factors for the occurrence of postoperative paraplegia were present; chronic dissection and/or a patent “critical” intercostal artery supplying the spinal cord (table 1). Chronic dissection or prior repair of ascending or abdominal aortic aneurysm was present in 10 patients. Of the 15 patients investigated angiographically for localization of the spinal artery, the critical artery was visualized in 8 (53%), arising between T7 and T12, usually on the left. In all except 1 patient, this artery arose from the aneurysm, signifying an especially high risk.
Table 1. General Data

<table>
<thead>
<tr>
<th>Age (y)</th>
<th>Aortic Lesion</th>
<th>Spinal Artery</th>
<th>Distal Perfusion</th>
<th>SEPs</th>
<th>PEPs</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>61</td>
<td>SC-celiac, ch. dissection</td>
<td>T9</td>
<td>None</td>
<td>P</td>
<td>L</td>
<td>Uncomplicated</td>
</tr>
<tr>
<td>51</td>
<td>SC-iliaic</td>
<td>T10</td>
<td>None</td>
<td>L</td>
<td>L</td>
<td>Died 2 h postop.</td>
</tr>
<tr>
<td>29</td>
<td>SC-iliaic, S/P AsAA repair</td>
<td>ND</td>
<td>None</td>
<td>P</td>
<td>L</td>
<td>Uncomplicated, headache (4 days)</td>
</tr>
<tr>
<td>67</td>
<td>T10-renal</td>
<td>T10</td>
<td>None</td>
<td>P</td>
<td>L</td>
<td>Uncomplicated</td>
</tr>
<tr>
<td>69</td>
<td>SC-celiac</td>
<td>T9</td>
<td>None</td>
<td>P</td>
<td>L</td>
<td>Died in OR</td>
</tr>
<tr>
<td>63</td>
<td>SC-femoral, ch. dissection</td>
<td>T7</td>
<td>None</td>
<td>P</td>
<td>L</td>
<td>Uncomplicated, headache (1 day)</td>
</tr>
<tr>
<td>70</td>
<td>SC-femoral, ch. dissection</td>
<td>NI</td>
<td>None</td>
<td>L</td>
<td>L</td>
<td>Paralyzed; died after 36 h</td>
</tr>
<tr>
<td>61</td>
<td>T9-iliaic</td>
<td>ND</td>
<td>None</td>
<td>P</td>
<td>L</td>
<td>Uncomplicated</td>
</tr>
<tr>
<td>67</td>
<td>SC-renal, S/P AsAA repair, ch. dissection</td>
<td>ND</td>
<td>Fem-Fem bypass</td>
<td>P</td>
<td>P</td>
<td>Uncomplicated</td>
</tr>
<tr>
<td>69</td>
<td>SC-celiac</td>
<td>T8</td>
<td>LV-FA</td>
<td>P</td>
<td>P</td>
<td>Uncomplicated</td>
</tr>
<tr>
<td>67</td>
<td>SC-AAA repair, ch. dissection</td>
<td>ND</td>
<td>None</td>
<td>*</td>
<td>L</td>
<td>Uncomplicated</td>
</tr>
<tr>
<td>76</td>
<td>SC-celiac, ch. dissection</td>
<td>NI</td>
<td>LV-FA</td>
<td>P</td>
<td>P</td>
<td>Uncomplicated, headache (3 days)</td>
</tr>
<tr>
<td>70</td>
<td>SC-renal</td>
<td>N1</td>
<td>LV-FA</td>
<td>P</td>
<td>P</td>
<td>Uncomplicated</td>
</tr>
<tr>
<td>71</td>
<td>Between DsAA and AAA repair</td>
<td>ND</td>
<td>LV-FA</td>
<td>P</td>
<td>P</td>
<td>Uncomplicated</td>
</tr>
<tr>
<td>76</td>
<td>T10 renal</td>
<td>T12</td>
<td>None</td>
<td>P</td>
<td>L</td>
<td>Uncomplicated</td>
</tr>
<tr>
<td>71</td>
<td>SC-renal, ch. dissection</td>
<td>ND</td>
<td>LA-FA</td>
<td>L</td>
<td>L</td>
<td>Paralyzed, died after 4 days</td>
</tr>
<tr>
<td>71</td>
<td>T10-renal</td>
<td>T9</td>
<td>AO-FA</td>
<td>P</td>
<td>P</td>
<td>Uncomplicated</td>
</tr>
<tr>
<td>59</td>
<td>SC-renal, S/P AAA, CABG</td>
<td>ND</td>
<td>LV-FA</td>
<td>P</td>
<td>P</td>
<td>Uncomplicated</td>
</tr>
</tbody>
</table>

SEP = spinal stimulation evoked potentials; PEP = peripheral evoked potentials; P = preserved; L = lost; SC = subclavian artery; ch. = chronic; NI = not investigated; Fem = femoral; ND = not demonstrated; AsAA = ascending aortic aneurysm; DsAA = descending aortic aneurysm; AAA = abdominal aortic aneurysm; OR = operating room; S/P = status post; LV = left ventricle; FA = femoral artery; LA = left atrium; AO = aorta; CABG = coronary artery bypass grafting.

* Technical difficulties in data recording.

evoked potentials were never preserved when postoperative paraplegia developed. The peripheral evoked potentials, however, showed poor correlation with the neurologic outcome when distal perfusion was not in use (table 2). Figure 2 shows the evolution of both somatosensory and spinal cord stimulation evoked potentials for two patients, one of whom had distal perfusion maintained with a bypass technique and one of whom did not.

An overall decrease in amplitude of both spinal stimulation and peripheral evoked potentials was observed in patients in whom spinal stimulation evoked potentials were not lost; amplitude decreased 50% at 45 min of cross-clamping (P < 0.05) whereas latency was not significantly changed compared to preclamping values (figs. 3 and 4). In all three patients in whom spinal evoked potentials were lost, an early decrease in amplitude (of more than 50%) was observed 5–10 min after aortic cross-clamping. However, there was no correlation between this decrease in amplitude and the time at which the evoked potentials totally disappeared.

Table 2. Spinal Stimulation and Peripheral Evoked Potentials Correlation with Neurologic Outcome

<table>
<thead>
<tr>
<th>Neurologic outcome</th>
<th>Evoked Potentials</th>
<th>Spinal Evoked Potentials</th>
<th>Peripheral Evoked Potentials</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Exist</td>
<td>Lost</td>
<td>Exist</td>
</tr>
<tr>
<td>Without distal perfusion (n = 10)</td>
<td>Complete</td>
<td>6</td>
<td>*</td>
</tr>
<tr>
<td></td>
<td>Early death</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Paralysis and death</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>With distal perfusion (n = 8)</td>
<td>Complete</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Early death</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Paralysis</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Technical difficulties in data recording (in one patient).
FIG. 2. Spinal cord stimulation and somatosensory evoked potentials for two patients. Right: Patient without bypass. Somatosensory evoked potentials disappear promptly with cross-clamp application while spinal evoked potentials are maintained throughout (with some decrease in amplitude). Left: Patient with bypass. Both spinal and somatosensory evoked potentials are maintained, although both demonstrate some transient degradation of amplitude immediately after clamp release. Both patients were neurologically intact postoperatively.

Discussion

Efforts continue in the complex field of thoracoabdominal aortic aneurysm surgery to find new modalities for detection and reduction of the ischemic insult to the spinal cord and abdominal organs. Early detection of ischemic changes in neural tissue may be important both as a warning sign to initiate protective interventions and as a means of better modifying therapy for subsequent patients.

A reliable method of detecting cord ischemia would be invaluable in furthering efforts to evaluate methods to improve blood supply to the spinal cord during aortic cross-clamping. Monitoring spinal cord function during the period of aortic cross-clamping has been proposed as a method for attempting to reduce the incidence of postoperative neurologic deficits. Somatosensory evoked potentials elicited by lower extremity nerve stimulation (peripheral evoked potentials) are routinely used for real-time, intraoperative monitoring of spinal cord function during spinal surgery. Using peripheral evoked potentials, Cunningham et al. studied 33 patients undergoing operation on the descending thoracic or thoracoabdominal aorta. In patients in whom distal aortic perfusion pressure was maintained at greater than 60 mmHg by either shunt or partial bypass techniques, the disappearance of peripheral evoked potentials accurately predicted

FIG. 3. Change in amplitude of spinal and peripheral evoked potentials during the aortic cross-clamping period. Each data point in the spinal tracing is a mean value (±SEM) from 12 patients and from 5 patients in the peripheral tracing. *Significant (P < 0.05 compared to PRE depression in mean amplitude was observed at 45 min of aortic clamping in the spinal data. Only patients who did not lose spinal evoked potentials are included.

FIG. 4. Change in latency time of spinal and peripheral evoked potentials during the aortic cross-clamping period. Only minimal changes in latency were observed.
postoperative paraplegia. In patients in whom distal perfusion was inadequately maintained, however, peripheral evoked potentials were not helpful. Crawford et al., using the same technique,9 reported 13% false negatives and 67% false positives using peripheral evoked potentials in 99 patients undergoing repair of thoracoabdominal aortic aneurysm.

In our small series, spinal cord stimulation evoked potentials were able to detect neurologic injury and predict neurologic outcome. Using this technique there were no false positives (i.e., loss of spinal evoked potentials with no neurologic injury). In general, we found changes in amplitude to be more marked in response to cross-clamping than changes in latency for both spinal and peripheral evoked potentials. In the present study, the development of an immediate postclamping large decrease in amplitude in the spinal stimulation evoked potentials was associated with poor outcome.

Several studies have evaluated peripheral somatosensory evoked potential monitoring during aortic surgery and have reported mixed results.24,19 We found a consistent distinction between patients who received adequate distal bypass shunting and those who did not (table 2). In all patients without adequate lower extremity perfusion, peripheral somatosensory evoked potentials disappeared within 5–30 min of aortic cross-clamping. Some authors suggest that disappearance of the peripheral evoked potentials within the first 20 min of cross-clamping is very suggestive of spinal cord ischemia, whereas a slower disappearance is typical for peripheral nerve ischemia.19 Our findings, however, show no correlation between timing of peripheral evoked potential disappearance and neurologic outcome; i.e., in 66% of patients with normal outcome, evoked potentials disappeared in less than 20 min, and in all within 30 min, when distal perfusion was not in use. In the remaining eight patients, with adequate distal perfusion, the behavior of peripheral somatosensory evoked potentials was commensurate with spinal evoked potentials (figs. 3 and 4). These findings are contrary to those of Crawford et al.,9 who reported a high incidence of false positives.

Due to vascular insufficiency and peripheral neuropathy, peripheral evoked potentials may be technically difficult to obtain and require expertise in interpretation even in optimal circumstances. We have noted an additional problem that arises if somatosensory evoked potentials are attempted in the extremity in which bypass or shunt cannulae are placed. Cannulae are directed cephalad at the femoral cutdown site, and the stabilization techniques used to secure the cannulae inhibit distal flow. Therefore, consideration should be given to placing posterior tibial electrodes contralateral to the extremity in which bypass or shunt cannulae are placed, as was done in the present study.

Spinal cord stimulation evoked potentials have advantages over peripheral somatosensory evoked potentials because of the factors discussed above (lower stimulus intensity and less dependence on peripheral perfusion). However, they do not directly monitor the motor tracts of the spinal cord. Spinal stimulation evoked potentials monitor the integrity of conduction in ascending fibers of the posterior and lateral columns of the spinal cord, whereas true motor evoked potentials are those that are generated by stimulation of the motor cortex and are recorded from spinal or peripheral electrodes.20,21 Therefore, it is possible that epidural stimulation of the posterior columns could result in continuation of recorded potentials despite inadequate perfusion of the anterior motor tracts, leading to paralysis. This outcome has been reported in nonvascular thoracic surgery with the use of peripheral evoked potentials.22–24 Laschinger et al.25 reported an investigation in dogs using intraoperative recording of motor evoked potentials. Their data suggest that distinctive changes in motor evoked potentials indicative of reversible ischemia of spinal cord motor tracts occur after thoracic aortic cross-clamping. The authors suggest the use of motor evoked potential monitoring during thoracic aneurysmectomy, but to our knowledge this technique has not been applied clinically.

The technique of epidural electrode insertion is not especially complicated when performed under fluoroscopy and has been described for patients with chronic pain, for whom it is in widespread clinical use.16 It is theoretically possible that direct stimulation of the spinal cord with excessive intensity could result in cord damage, although we have noted no complications of placement or stimulation in the patients in our series. Compared to peripheral evoked potentials monitoring, the spinal cord stimulation evoked potentials require no additional recording apparatus.

We conclude that peripheral somatosensory evoked potentials are not of value in the assessment of spinal cord ischemia during aortic cross-clamping if the extremity from which the evoked potentials is monitored is not perfused. Maintenance of adequate distal perfusion by partial bypass or shunt may permit the use of peripheral evoked potentials in assessment of spinal cord ischemia. Our results are consistent with the hypothesis that maintenance of spinal stimulation evoked potentials during aortic cross-clamping portends good neurologic outcome. If the reliability of spinal cord stimulation evoked potentials is confirmed in larger series, this technique may serve as an important tool not only for early detection of ischemia but also for the development and evaluation of surgical techniques that may possibly improve outcome.

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References


