

## Continuous Monitoring of Jugular Bulb Venous Oxygen Saturation for Evaluation of Cerebral Perfusion during Carotid Endarterectomy

Hiroshi NIINAI<sup>1)</sup>, Itsuo NAKAGAWA<sup>1)</sup>, Takeshi SHIMA<sup>2)</sup>, Masashi KAWAMOTO<sup>3)</sup>  
 and Osafumi YUGE<sup>3)</sup>

1) Department of Anesthesia, Chugoku Rosai General Hospital, Kure, Hiroshima 737-0193, Japan

2) Department of Neurosurgery, Chugoku Rosai General Hospital, Kure, Hiroshima 737-0193, Japan

3) Department of Anesthesiology and Critical Care Medicine, Hiroshima University School of Medicine, Hiroshima 734-8551, Japan

### ABSTRACT

In this study we examined whether continuous monitoring of jugular bulb venous oxygen saturation (SjO<sub>2</sub>) is applicable for the evaluation of cerebral hypoperfusion during carotid endarterectomy (CEA). The subjects were 25 patients who underwent elective CEA under general anaesthesia. After the carotid stump pressure (SP) was measured, SjO<sub>2</sub> and the somatosensory evoked potentials (SEP) were monitored during the carotid test clamping for 10 min.

There was no alteration in cardiovascular and respiratory status during the test clamping. No correlation was observed between SEP amplitude and SP ( $r=0.16$ ,  $p=0.25$ ). However, at clamping, SjO<sub>2</sub> decreased from 70 to 64% ( $p<0.01$ ) with a reduction in SEP amplitude from 2.0 to 1.6  $\mu V$  ( $p<0.01$ ). After declamping, SjO<sub>2</sub> increased from 65 to 70% ( $p<0.01$ ) with a recovery in SEP from 1.6 to 1.9  $\mu V$  ( $p<0.01$ ). The changes in SEP amplitude and SjO<sub>2</sub> correlated ( $r=0.66$ ,  $p<0.001$ ).

These results suggest that continuous monitoring of SjO<sub>2</sub> is superior to SP measurement in the prediction of cerebral hypoperfusion caused by carotid clamping and applicable to CEA.

**Key words:** Jugular bulb venous oxygen saturation, Cerebral ischaemia, Carotid clamp, Carotid endarterectomy

Carotid endarterectomy (CEA) as a prophylactic surgical treatment for the prevention of stroke in patients with symptomatic carotid stenosis has been widely performed, and its efficacy has been confirmed<sup>2,13</sup>. Although anaesthetic techniques have been improving<sup>11,16</sup>, cerebral infarction from inadequate cerebral blood flow during carotid cross clamping is still a problem. Carotid stump pressure (SP) has been employed to evaluate the necessity of internal shunts, with intraoperative monitoring of an electroencephalogram (EEG) succeeding SP. In addition, monitoring of somatosensory evoked potentials (SEP) have been proposed as a reliable tool. However, as long as we have to evaluate the change in cerebral perfusion for a relatively short time, monitoring should preferably be continuous, quantitative, prompt and easy to interpret by the anaesthetists who manage the cardiovascular, respiratory and anaesthetic conditions just beside the patients. Therefore, none of the above monitor would be ideal on its own.

Fiberoptic continuous measurement of jugular bulb venous oxygen saturation (SjO<sub>2</sub>) has recently become available, and it may allow us to observe

the real-time balance of cerebral blood flow and metabolism. Under general anaesthesia, given a stabilized cerebral metabolism, changes in SjO<sub>2</sub> would represent changes in the cerebral blood flow. The aims of the present study were to evaluate the efficacy of continuous SjO<sub>2</sub> monitoring for the prediction of cerebral hypoperfusion during carotid cross clamping and to demonstrate its applicability to CEA in patients being operated under general anaesthesia.

### MATERIALS AND METHODS

We studied 25 patients (20 men and 5 women; mean age 63 yr, weight 64 kg, and height 161 cm) who underwent elective CEA following transient ischaemic attack or minor stroke due to severe carotid stenosis (unilateral; 21 patients, bilateral; 4 patients). After approval of the hospital ethical committee, informed consent was obtained from each patient.

Patients were premedicated with 5 to 10 mg of oral diazepam 1 hr prior to the induction. General anaesthesia was induced with 3 to 4 mg/kg of thi-amylal and 0.2 to 0.3 mg of fentanyl, then endotra-

cheal intubation was facilitated with 0.1 mg/kg of vecuronium. Anaesthesia was maintained with nitrous oxide and 0.5 to 1.0% isoflurane in oxygen. Incremental doses of fentanyl and vecuronium were added as needed. Radial arterial pressure, electrocardiogram, end-tidal carbon dioxide tension (EtCO<sub>2</sub>) and pulse oximetry were continuously monitored (Model 66S, Hewlett Packard, Andover, USA). EtCO<sub>2</sub> was maintained as close as possible to 35 mmHg. For the evaluation of intra-operative cerebral perfusion, SjO<sub>2</sub>, SP and SEP were monitored.

#### SjO<sub>2</sub> measurement

We confirmed that the right and the left transverse sinus were equally observed in the venous phase of cerebral angiography of each patient. After exposure of the internal jugular vein and the common facial vein, a 4F fiber optic catheter (OPTICATH, Abbott Laboratories, North Chicago, USA) was inserted into the transected end of the facial vein and threaded up the internal jugular vein as far as it could be advanced to the jugular bulb. The position of the catheter tip was confirmed by X-ray. The catheter was connected to the oximetry for continuous SjO<sub>2</sub> measurement (Oximetrix 3, Abbott Laboratories, North Chicago, USA). Jugular venous blood was sampled very slowly, to avoid extracranial blood contamination, through the catheter and oxygen saturation was measured for *in vivo* calibration (ABL 510, Radiometer, Copenhagen, Denmark).

#### Stump pressure measurement

After a pressure transducer was calibrated at the level of the mid-axillary line in the supine position, a 26-gauge needle connected to the manometer was inserted into the common carotid artery. The external carotid artery and the common

carotid artery just proximal to the needle were clamped to measure SP (RMT-6004, Nihon Kohden, Tokyo, JAPAN).

#### SEP monitoring

Following induction of general anaesthesia, electrodes for SEP monitoring were set up: two surface electrodes for stimulation were placed on the wrist, contralateral to the operation side, along the median nerve at a distance of 4 cm. A square wave stimulus of 0.1 msec at a rate of 5 Hz was delivered to the median nerve and the intensity was adjusted to produce a minimal muscle contraction. According to the 10–20 International System, a referential electrode was placed on Fz and an exploring electrode was placed on the scalp overlying the sensory cortex contralateral to the stimulated side (C3' or C4'). Sampled waves were processed by the SEP monitor (Cadwell 5200A, Cadwell Laboratories, Kennewick, USA) and the data were recorded by a personal computer (PC-9801, NEC, Tokyo, JAPAN). Changes in the amplitude of the short-latency SEP (N<sub>20</sub>-P<sub>25</sub>) were evaluated to detect cerebral ischaemia.

#### Study protocol

After SP was measured, a test clamping of the external carotid artery and the common carotid artery was performed for 10 min to evaluate cerebral tissue tolerance against ipsilateral carotid occlusion. During the test clamping, neither the inspired oxygen tension nor isoflurane concentration was altered.

Mean arterial pressure (MAP), arterial blood oxygen saturation (SpO<sub>2</sub>), EtCO<sub>2</sub>, SjO<sub>2</sub>, and SEP were recorded at the following times: (1) immediately before clamping; (2) 1 min after clamping; (3) immediately before declamping; and (4) 1 min after declamping. SEP was recorded consecutively

**Table 1.** Cardiovascular and respiratory status during 10 min carotid test clamping

	pre-clamping	clamping 1 min	pre-declamping	declamping 1 min
MAP (mmHg)	87±11	88±13	90±10	85±10
EtCO <sub>2</sub> (mmHg)	35±5	35±5	35±5	35±5
SpO <sub>2</sub> (%)	99±1	99±1	99±1	99±1

(mean±SD)

Changes in mean arterial pressure (MAP), end-tidal carbon dioxide tension (EtCO<sub>2</sub>) and arterial blood oxygen saturation (SpO<sub>2</sub>) at the time of pre-clamping, clamping 1min, pre-declamping and declamping 1 min. There was no significant difference between groups (ANOVA)

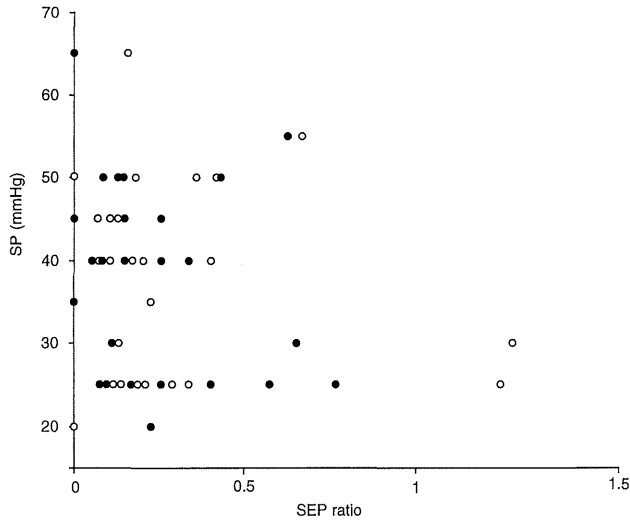
**Table 2.** Changes in jugular bulb venous oxygen saturation and somatosensory evoked potential

	pre-clamping	clamping 1 min	pre-declamping	declamping 1 min
SjO <sub>2</sub> (%)	70±12	64±12 *	65±11	70±10 †
SEP amplitude (μV)	2.0±0.5	1.6±1.0 *	1.6±1.0	1.9±1.0 †

(mean±SD)

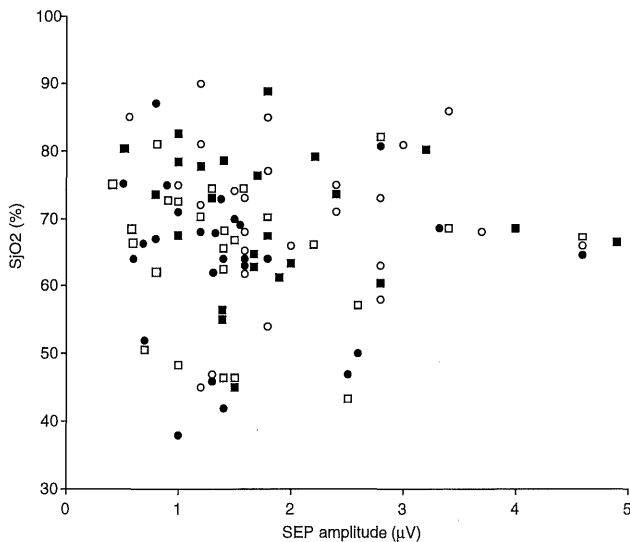
Changes in jugular bulb venous oxygen saturation (SjO<sub>2</sub>) and somatosensory evoked potential (SEP) amplitude (N<sub>20</sub>-P<sub>25</sub>) at the time of carotid cross clamping and declamping.

\*p<0.01 compared with pre-clamping, †p<0.01 compared with pre-declamping (paired t test)



**Fig. 1.** Relationship between somatosensory evoked potential (SEP) ratio at clamping, declamping procedure and stump pressure (SP).

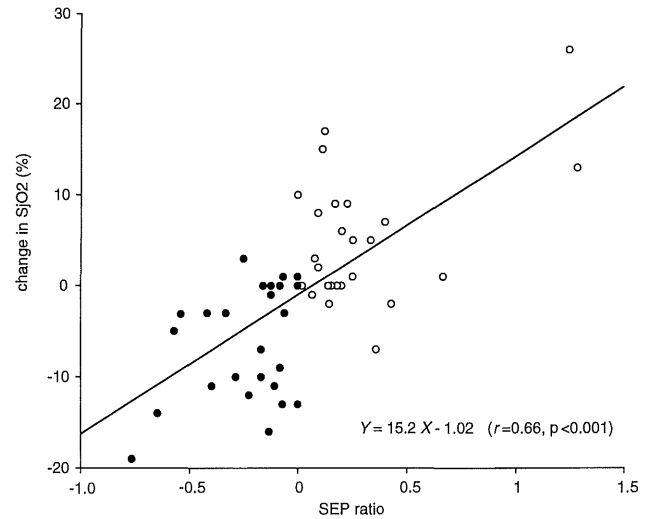
The ratio of [decrease in amplitude ( $\mu\text{V}$ ) / pre-clamping amplitude ( $\mu\text{V}$ )] (●) and the ratio of [increase in amplitude ( $\mu\text{V}$ ) / pre-declamping amplitude ( $\mu\text{V}$ )] (○). There was no significant correlation. ( $n=50$ ,  $r=0.16$ ,  $p=0.25$ )



**Fig. 2.** Relationship between somatosensory evoked potential (SEP) amplitude and jugular bulb venous oxygen saturation ( $\text{SjO}_2$ ) at the time of pre-clamping (○), clamping 1 min (●), pre-declamping (□) and declamping 1 min (■). There was no significant correlation. ( $n=100$ ,  $r=0.003$ ,  $p=0.97$ )

during carotid clamping and every 5 min until the end of the operation. The serial changes in SEP amplitude were calculated as the ratio of [decrease in amplitude ( $\mu\text{V}$ ) / pre-clamping amplitude ( $\mu\text{V}$ )] and [increase in amplitude ( $\mu\text{V}$ ) / pre-declamping amplitude ( $\mu\text{V}$ )].

The changes of MAP,  $\text{EtCO}_2$  and  $\text{SpO}_2$  were analyzed using analysis of variance. The relationships



**Fig. 3.** Relationship between somatosensory evoked potential (SEP) ratio and change in jugular bulb venous oxygen saturation ( $\text{SjO}_2$ ) at clamping, declamping.

The ratio of [decrease in amplitude ( $\mu\text{V}$ ) / pre-clamping amplitude ( $\mu\text{V}$ )] (●) and the ratio of [increase in amplitude ( $\mu\text{V}$ ) / pre-declamping amplitude ( $\mu\text{V}$ )] (○). There was significant correlation. ( $n=50$ ,  $r=0.66$ ,  $p<0.001$ )

amongst SEP, SP and  $\text{SjO}_2$  were evaluated using Pearson's correlation coefficient. The changes in SEP and  $\text{SjO}_2$  at the time of carotid cross clamping and declamping were compared using a paired t test. Values are presented as mean  $\pm$  SD.  $p<0.05$  was considered statistically significant.

## RESULTS

No significant difference was observed between haemoglobin concentration measured before and after test clamping. There was no significant change in MAP,  $\text{EtCO}_2$  and  $\text{SpO}_2$  throughout the 10 min carotid test clamping (Table 1). No significant correlation was observed between SEP and SP ( $r=0.16$ ,  $p=0.25$ ) (Fig. 1). There was no significant correlation between SEP amplitude and  $\text{SjO}_2$  value in 100 points recorded throughout the carotid cross clamping and declamping procedure ( $r=0.003$ ,  $p=0.97$ ) (Fig. 2). On application of the carotid clamping,  $\text{SjO}_2$  decreased from 70 to 64% ( $p<0.01$ ). At the same time, SEP amplitude decreased from 2.0 to 1.6  $\mu\text{V}$  ( $p<0.01$ ). After declamping,  $\text{SjO}_2$  increased from 65 to 70% ( $p<0.01$ ) in association with a recovery in SEP from 1.6 to 1.9  $\mu\text{V}$  ( $p<0.01$ ) (Table 2). There was a significant correlation between the changes in SEP shown by the ratio and the changes in  $\text{SjO}_2$  ( $r=0.66$ ,  $p<0.001$ ) (Fig. 3).

## DISCUSSION

We demonstrated the quick response of  $\text{SjO}_2$  reading and the superiority of the continuous mea-

surement of  $SjO_2$  over SP for evaluation of cerebral perfusion during carotid cross clamping for CEA. The changes in SEP and  $SjO_2$  were supposed to be produced by changes in the cerebral blood flow during ipsilateral carotid cross clamping, and not by anaesthesia, because MAP,  $EtCO_2$  and  $SpO_2$  were maintained consistently throughout the procedure.

Patients undergoing CEA demonstrate various sclerotic cerebrovascular changes according to the stage of disease. Therefore, it is possible that the degree of the effect of carotid cross clamping on ipsilateral cerebral perfusion varies in each patient. Although most patients undergoing CEA can tolerate short-term carotid cross clamping, they might suffer from cerebral hypoperfusion to a certain extent, which may not cause irreversible neurological damage<sup>17</sup>. Thus, it is preferable to employ a continuous monitor to detect cerebral hypoperfusion before causing cerebral tissue damage.

In the early era of CEA, SP was commonly used to estimate collateral circulation during carotid clamping<sup>4,12</sup>. However, SP represents the pressure of relatively proximal collateral vessels to the clamped carotid artery and may not give information about blood flow in the more distal regions. Actually, the unreliability of SP<sup>7,10</sup> and the discrepancy between SP and EEG monitoring<sup>3,5</sup> have been documented. Indeed, our data showed little correlation between changes in SEP and SP at the time of carotid cross clamping and declamping. This supports the contention that SP measurement is limited in the prediction of cerebral ischaemia during CEA.

The prolonged central conduction time and reduced amplitude of SEP caused by carotid clamping were recoverable with shunt insertion<sup>14,18</sup>. Furthermore, a close correlation between loss of SEP amplitude during clamping and the appearance of new neurological deficits after the operation was reported<sup>15</sup>. These observations suggest that SEP amplitude well represents cerebral perfusion and that SEP monitoring can predict cerebral ischaemia during CEA.

Although we could not find a correlation between the raw value of  $SjO_2$  and SEP amplitude,  $SjO_2$  fell and rose significantly at the time of carotid clamping and declamping accompanied by the decrease and increase in SEP amplitude. As there was a significant relationship between changes in  $SjO_2$  and SEP amplitude during the clamping and declamping procedure, a fall in the  $SjO_2$  value during carotid clamping could be a warning that the collateral blood flow is not sufficient to maintain cerebral perfusion. Increased oxygen extraction by the hypoxic cerebral tissue causes a fall in venous oxygen saturation. After declamping, an immediate increase in  $SjO_2$  accompanied by the recovery of SEP amplitude shows that the ipsilateral cere-

bral perfusion has increased enough to supply a suitable amount of oxygen to the ischaemic cerebral tissue. Actually, there were patients who showed little change in  $SjO_2$  and SEP amplitude at clamping and declamping. This can be explained by the existence of an appropriate collateral blood flow from the contralateral carotid artery and/or the vertebral arteries.

In early studies of  $SjO_2$  during CEA<sup>8,9</sup>, directly sampled jugular bulb venous blood was analyzed. Under local anaesthesia, it was reported that the lower acceptable limit of  $SjO_2$  in patients undergoing CEA was 50%<sup>9</sup>. Even under general anaesthesia,  $SjO_2$  less than 50% could be critical and we would have to cope with the possibility of cerebral hypoperfusion. However, our results showed the importance of evaluating the changes in  $SjO_2$ . In most cases, SEP amplitude decreased with a reduction in  $SjO_2$ , although the  $SjO_2$  value remained over 50%. As long as the cerebral tissue is properly perfused with oxygenated blood and the cerebral metabolism is not altered, SEP amplitude should be maintained consistently during carotid clamping. Since  $SjO_2$  provides information of global cerebral perfusion, it is possibly maintained within an appropriate level even if ischaemia exists in only a small region. Therefore, we have to consider focal ischaemia to prevent neurological complications. Monitoring of  $SjO_2$  should preferably be combined with SEP to obtain more precise ischaemic information during clamping.

It was reported that near infra-red spectroscopy (NIRS) had a good correlation to  $SjO_2$  analyzed by co-oximetry<sup>19</sup>. However, continuous  $SjO_2$  measurement is not equivalent to NIRS, which could be affected by the extracranial blood flow<sup>6,19</sup>.  $SjO_2$  is oxygen saturation of the drained blood from the intracranial tissue and is less influenced by extracranial factors as long as the tip of the catheter is located properly in the jugular bulb.

We suggest that continuous monitoring of  $SjO_2$  is superior to SP and applicable to CEA. It enables anaesthetists to give a quick warning of cerebral hypoperfusion to the surgeons during carotid clamping. Nevertheless, for a more precise detection of cerebral ischaemia, we propose combining  $SjO_2$  with other monitors such as SEP if electrical interference is avoidable in the operating theatre.

(Received April 16, 1998)

(Accepted September 21, 1998)

## REFERENCES

1. **Asiddao, C.B., Donegan, J.H., Whitesell, R.C. and Kalbfleisch, J.H.** 1982. Factors associated with perioperative complications during carotid endarterectomy. *Anesth. Analg.* **61**: 631–637.
2. **European Carotid Surgery Trialists' Collaborative Group.** 1991. MRC European

- Carotid Surgery Trial: interim results for symptomatic patients with severe (70–90%) or with mild (0–29%) carotid stenosis. *Lancet* **337**: 1235–1243.
3. **Harada, R.N., Comerota, A.J., Good, G.M., Hashemi, H.A. and Hulihan, J.F.** 1995. Stump pressure, electroencephalographic changes, and the contralateral carotid artery: another look at selective shunting. *Am. J. Surg.* **170**: 148–153.
  4. **Hays, R.J., Levinson, S.A. and Wylie, E.J.** 1972. Intraoperative measurement of carotid back pressure as a guide to operative management for carotid endarterectomy. *Surgery* **72**: 953–960.
  5. **Kelly, J.J., Callow, A.D., O'Donnell, T.F., McBride, K., Ehrenberg, B., Korwin, S., Welch, H. and Gembarowicz, R.M.** 1979. Failure of carotid stump pressures. Its incidence as a predictor for a temporary shunt during carotid endarterectomy. *Arch. Surg.* **114**: 1361–1366.
  6. **Kirkpatrick, P.J., Smielewski, P., Whitfield, P.C., Czosnyka, M., Menon, D. and Pickard, J.D.** 1995. An observational study of near-infrared spectroscopy during carotid endarterectomy. *J. Neurosurg.* **82**: 756–763.
  7. **Kwaan, J.H.M., Peterson, G.J. and Connolly, J.E.** 1980. Stump pressure. An unreliable guide for shunting during carotid endarterectomy. *Arch. Surg.* **115**: 1083–1085.
  8. **Larson, C.P., Ehrenfeld, W.K., Wade, J.G. and Wylie, E.J.** 1967. Jugular venous oxygen saturation as an index of adequacy of cerebral oxygenation. *Surgery* **62**: 31–39.
  9. **Lyons, C., Clark, L.C., McDowell, H. and McArthur, K.** 1964. Cerebral venous oxygen content during carotid thrombintectomy. *Ann. Surg.* **160**: 561–567.
  10. **McKay, R.D., Sundt, T.M., Michenfelder, J.D., Gronert, G.A., Messick, J.M., Sharbrough, F.W. and Piepgras, D.G.** 1976. Internal carotid artery stump pressure and cerebral blood flow during carotid endarterectomy: modification by halothane, enflurane and Innovar. *Anesthesiology* **45**: 390–399.
  11. **Michenfelder, J.D., Sundt, T.M., Fode, N. and Sharbrough, F.W.** 1987. Isoflurane when compared to enflurane and halothane decreases the frequency of cerebral ischemia during carotid endarterectomy. *Anesthesiology* **67**: 336–340.
  12. **Moore, W.S. and Hall, A.D.** 1969. Carotid artery back pressure. A test of cerebral tolerance to temporary carotid occlusion. *Arch. Surg.* **99**: 702–710.
  13. **North American Symptomatic Carotid Endarterectomy Trial Collaborators.** 1991. Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. *N. Engl. J. Med.* **325**: 445–453.
  14. **Pozzessere, G., Valle, E., Santoro, A., Delfini, R., Rizzo, P.A., Cantore, G.P. and Morocutti, C.** 1987. Prognostic value of early somatosensory evoked potentials during carotid surgery: relationship with electroencephalogram, stump pressure and clinical outcome. *Acta Neurochir.* **89**: 28–33.
  15. **Schweiger, H., Kamp, H.D. and Dinkel, M.** 1991. Somatosensory-evoked potentials during carotid artery surgery: experienced in 400 operations. *Surgery* **109**: 602–609.
  16. **Smith, J.S., Roizen, M.F., Cahalan, M.K., Benefiel, D.J., Beaupre, P.N., Sohn, Y.J., Bryd, B.F., Schiller, N.B., Stoney, R.J., Ehrenfeld, W.K., Ellis, J.E. and Aronson, S.** 1988. Does anesthetic technique make a difference? Augmentation of systolic blood pressure during carotid endarterectomy: effects of phenylephrine versus light anesthesia and of isoflurane versus halothane on the incidence of myocardial ischemia. *Anesthesiology* **69**: 846–853.
  17. **Sundt, T.M.** 1983. The ischemic tolerance of neural tissue and the need for monitoring and selective shunting during carotid endarterectomy. *Stroke* **14**: 93–98.
  18. **Tiberio, G., Floriani, M., Giulini, S.M., Bonardelli, S., Portolani, N., Pulcini, G., Guarneri, B., DeMaria, G., Antonini, L., Tomasoni, G. and Renato, F.** 1991. Monitoring of somatosensory evoked potentials during carotid endarterectomy: relationship with different haemodynamic parameters and clinical outcome. *Eur. J. Vasc. Surg.* **5**: 647–653.
  19. **Williams, I.M., Picton, A., Farrell, A., Mead, G.E., Mortimer, A.J. and McCollum, C.N.** 1994. Light-reflective cerebral oximetry and jugular bulb venous oxygen saturation during carotid endarterectomy. *Br. J. Surg.* **81**: 1291–1295.