

# Anaesthesia for Carotid Endarterectomy

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## Introduction

About 120 carotid endarterectomies are performed each year at our hospital. All patients have more than 70% stenosis of one or both internal carotid arteries as shown by Duplex-scan and/or arteriography. Most of the patients suffered previous transient ischemic attacks or reversible ischemic neurologic deficits although some patients are asymptomatic.

It is thought that surgical removal of the stenosis can significantly diminish the risk of stroke.

Two main questions arise when giving anaesthesia for these operations: firstly, can the brain be protected from ischemia by our anaesthetic technique and secondly, how is brain perfusion best monitored during the cross-clamping of the carotid artery?

## Anaesthetic technique

Some surgeons prefer to operate under local or loco-regional (interscalene cervical plexus block) anaesthesia, so that the neurologic state can be monitored directly<sup>(1)</sup>.

We believe that this method imposes too much stress on these patients who often have some degree of coronary disease<sup>(2)</sup>. For this reason all of the carotid operations are performed under general anaesthesia at our hospital. During general anaesthesia one should try to provide sufficient cerebral circulation and oxygenation and to avoid myocardial ischemia. Therefore controlled respiration with sufficient inspiratory oxygen concentration is mandatory. End-tidal pCO<sub>2</sub> is routinely monitored. The patient is kept normocapnic<sup>(3)</sup> as hypocapnia induces cerebral vasoconstriction and diminishes collateral bloodflow. Hypercapnia results in cerebral vasodilatation and can thereby cause deviation of bloodflow from the ischemic to the vasodilated region of the brain.

Throughout the operation blood pressure should be very stable and kept at or slightly above the

patient's normal blood pressure level to assure collateral circulation through the heterolateral carotid artery and vertebral arteries and the circle of Willis<sup>(4)</sup>. Depending on the patient's heart rate we eventually administer small I.V. boluses of ephedrine or phenylephrine to achieve the required blood pressure. To prevent myocardial ischemia, tachycardia or extreme hypertension should be avoided. Glucose is not given. In the case of cerebral ischemia a glycemia of 150 mg% or more seems to be associated with worse neurologic outcome in experimental animal studies as well as in outcome studies in humans after circulator arrest<sup>(5,6,7,8,9)</sup>.

## Pharmacological brain protection

Can we protect the brain during the ischemic episode by the anaesthetic used? One might speculate that lowering the functional activity of the brain cells may diminish the need for oxygen, and thus blood flow, to levels sufficient to assure cell integrity. The blood flow level needed is about 12-18 ml/100 gr of tissue/minute.

An alternative explanation for the possible brain protective action of barbiturates is their role as free radical scavengers.

Ca-channel blockers are supposed to have a protective action by preventing Ca-influx at the cellular level or by preventing the postischemic hypoperfusion state. In a lot of animal and human experiments a protective action of barbiturates, isoflurane and Ca-channel blockers is thought to be proven by the amelioration of some biochemical and physiological parameters and more rarely by outcome improvement<sup>(10,11,12,13,14)</sup>.

However when we look at outcome studies in humans a pharmacological brain protective action is far less evident. The Nussmeyer study<sup>(15)</sup> shows some evidence of some brain protective effect with extremely high doses of barbiturates in patients undergoing cardiac surgery. The high doses of barbiturates are impractical in the setting of carotid artery surgery because of their prolonged action

and the hemodynamic instability they provoke. In a retrospective, non-randomized study, the Mayo Clinic group has shown that in patients anaesthetized with isoflurane, ischemic EEG changes occurred significantly less frequently (18%) as in patients anaesthetized with other volatile anaesthetics (26%)<sup>(16)</sup>. Also, ischemic changes under isoflurane anaesthesia occurred at lower cerebral blood flows and the need for a shunt was less frequent (37% vs. 44%) as with other volatile agents. The same group of investigators showed that the critical flow value (the flow value at which ischemic EEG changes occur in 50% of the patients) is 10-12 ml/100 gr/minute in the isoflurane group versus 16-18 ml/100 gr/minute for the other volatile agents.

### Monitoring cerebral perfusion

Measuring cerebral blood flow by the radioactive Xenon technique might be considered to be the best way to verify that cerebral perfusion is adequate. However the technique is impractical in the operation room and often not available. The excellent correlation between CBF and EEG activity explains why EEG monitoring is more popular<sup>(18)</sup>.

Measuring the pressure in the distal carotid stump after clamping gives an idea about the collateral circulation. However there is little agreement about which stump pressure is adequate to prevent neurologic deficits. Values from 25 to 70 mmHg have been recommended<sup>(19,20)</sup>.

Bilateral evoked potential monitoring is a reliable method to monitor cerebral perfusion because of the good correlation with EP-latency and amplitude changes<sup>(21)</sup>. This method is less popular than EEG monitoring but certainly deserves more investigation, because it does not only monitor the cerebral cortex but also deeper brain structures.

The traditional method for detecting cerebral ischemia is the electroencephalogram. When unilateral cerebral ischemia is present a lowering of frequency or voltage attenuation will occur within one minute. EEG changes are evident when CBF drops below 18 to 16 ml/100 gr/min. Irreversible cell damage occurs when CBF falls below 10-12 ml/100 gr/min.<sup>(18)</sup>. This means that there is a safety margin between EEG changes and cell damage.

The standard 16 channel-EEG has the advantage of detecting regional ischemic events, but is rather impractical in the operation theatre. Also special personnel is required to interpret the formation it generates.

There are a number of simple two or four channel EEG monitors available which are provided with several automatic processing techniques which

help the anaesthetist with the interpretation of the data. Regional information is sacrificed, as the monitor only registers the electroencephalographic signals in the region where the electrodes are placed.

Blume found that only major EEG changes (extreme slowing or electroencephalographic silence) were indicative for possible postoperative neurologic deficit<sup>(22)</sup>.

A two channel monitor is perfectly capable of detecting these major changes and thus to identify hemicerebral ischemia. In the same study Blume found that only 9% of the patients with major EEG changes had postoperative neurologic deficit; no patients without EEG changes had postoperative deficits. EEG monitoring can identify those patients in which the cerebral perfusion is at risk during clamping of the carotid artery and those who may benefit from a shunt. A malfunctioning shunt is also readily detected by this monitoring technique. Neurologic deficit can be caused by embolisation phenomena which can occur during the whole perioperative period, and when these involve a sufficiently small region, they can be missed by EEG monitoring. This explains why one sometimes finds patients with post-operative neurologic deficits without evidence of EEG changes during operation.

### Experience from the University Hospital Leuven

At our hospital we use a balanced anaesthetic technique based on an etomidate infusion combined with isoflurane inhalation and a low dose of fentanyl as an analgesic.

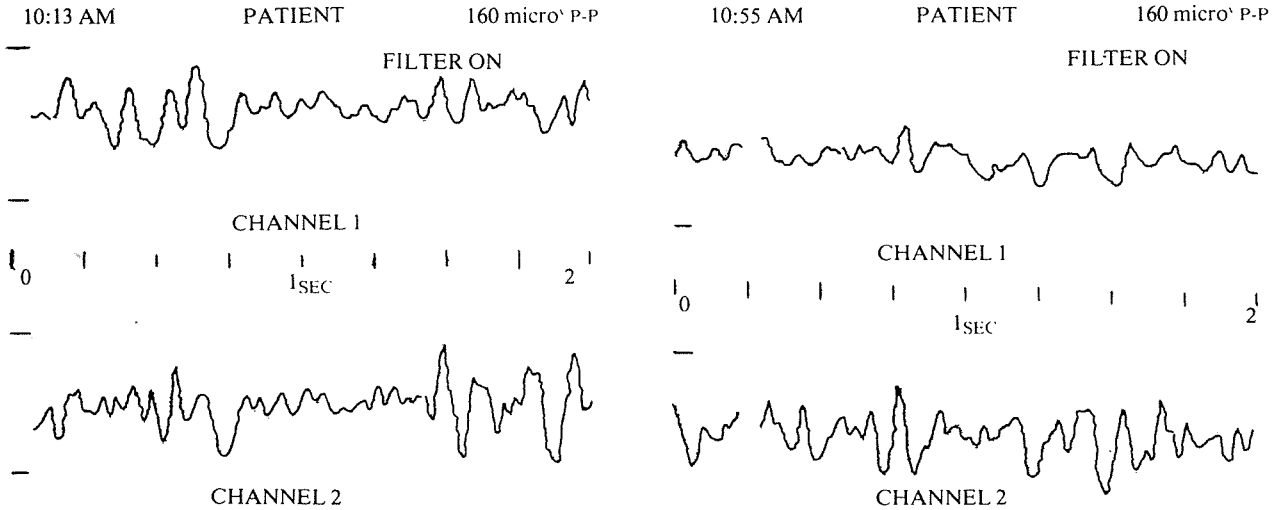
An induction dose of etomidate (0,3 mg/kg) is followed by an infusion at 0,03 mg/kg/min for 27 minutes and is thereafter diminished to 0,01 mg/kg/min. The infusion rate can be altered according to the depth of anaesthesia as measured by the encephalogram. Etomidate lowers the cerebral metabolic rate of oxygen in a similar fashion to the barbiturates. It also provides strikingly stable hemodynamic conditions at induction. A 24 hours cortisol substitution therapy is started immediately after the operation (100 mg of hydrocortisone every 8 hours) because of the cortisol synthesis blocking effect of etomidate.

Low dose Fentanyl (3-4 ug/kg) and Isoflurane inhalation are added to blunt over hypertensive responses.

This anaesthetic technique allows prompt postoperative awakening and neurologic evaluation.

Collateral cerebral circulation is evaluated in two ways. First, EEG is continuously followed by a two

**FIGURE 1**



Asymmetry of the raw EEG after clamping the left carotid artery in a patient with a 75% stenosis on the right side and a filiform stenosis of the left carotid artery. Stump pressure was 30 mmHg.

**FIGURE 2**

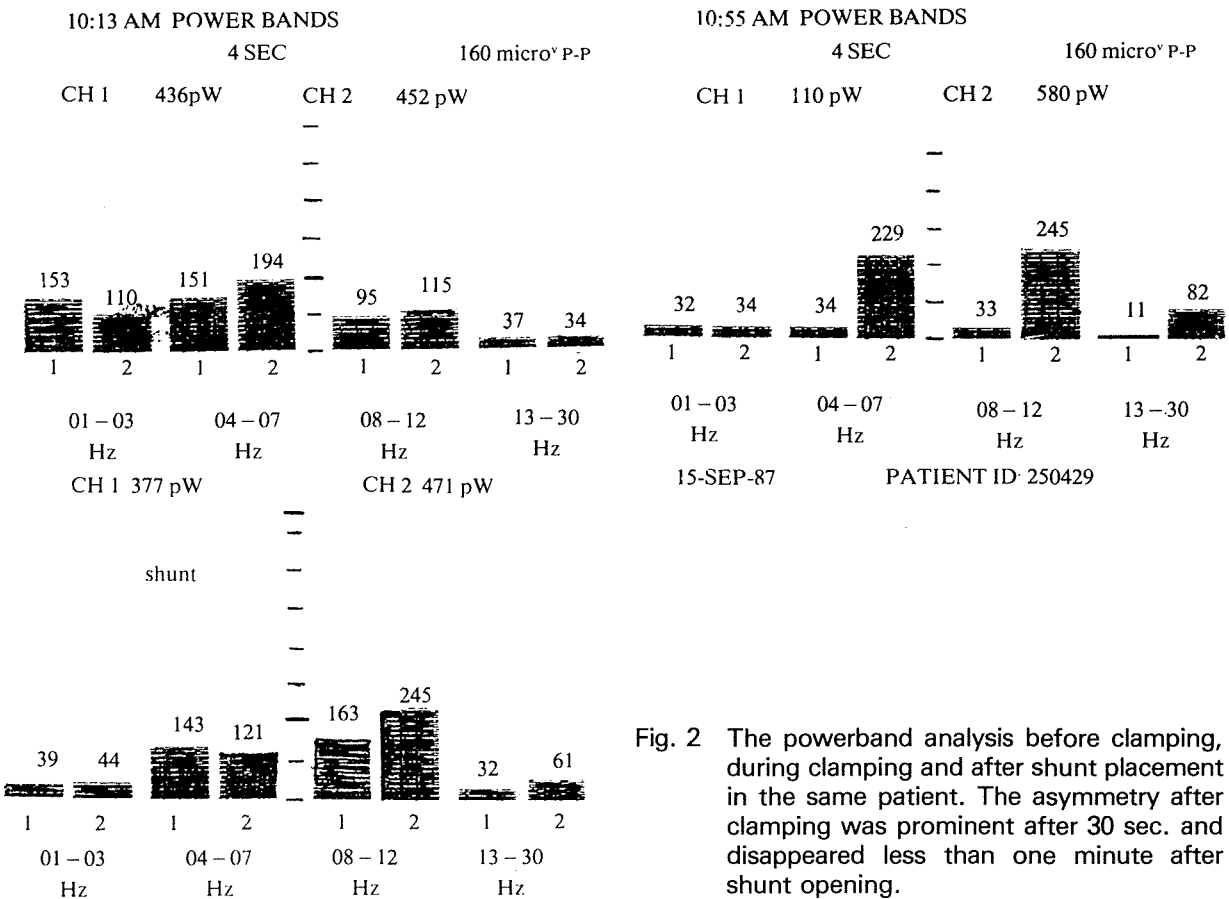
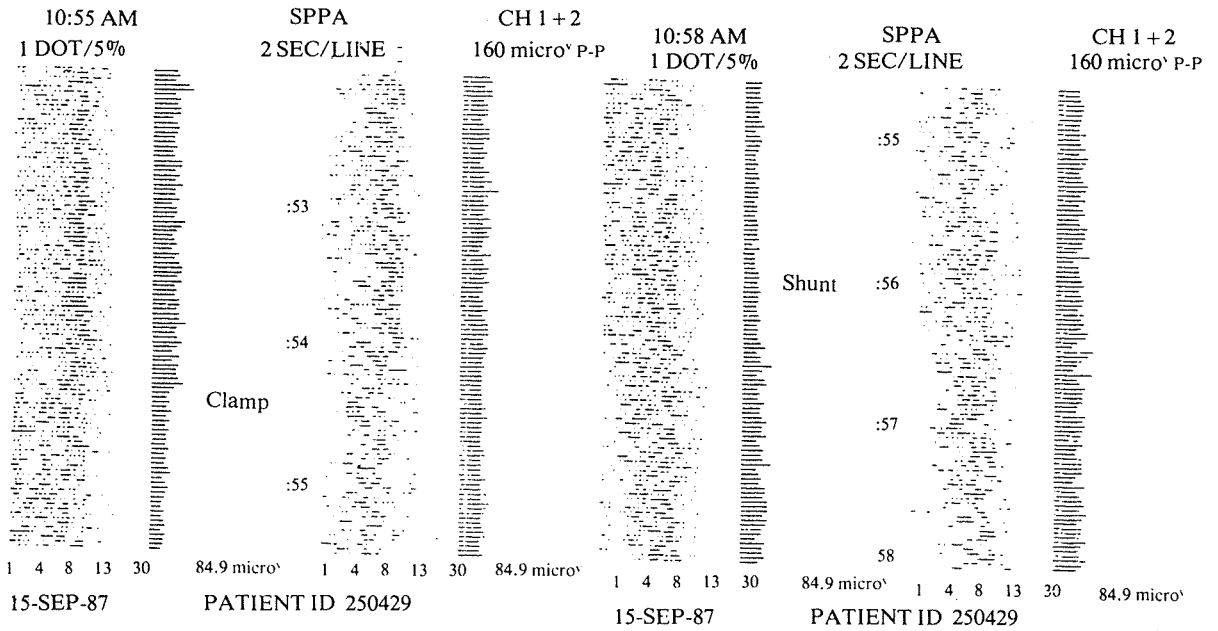


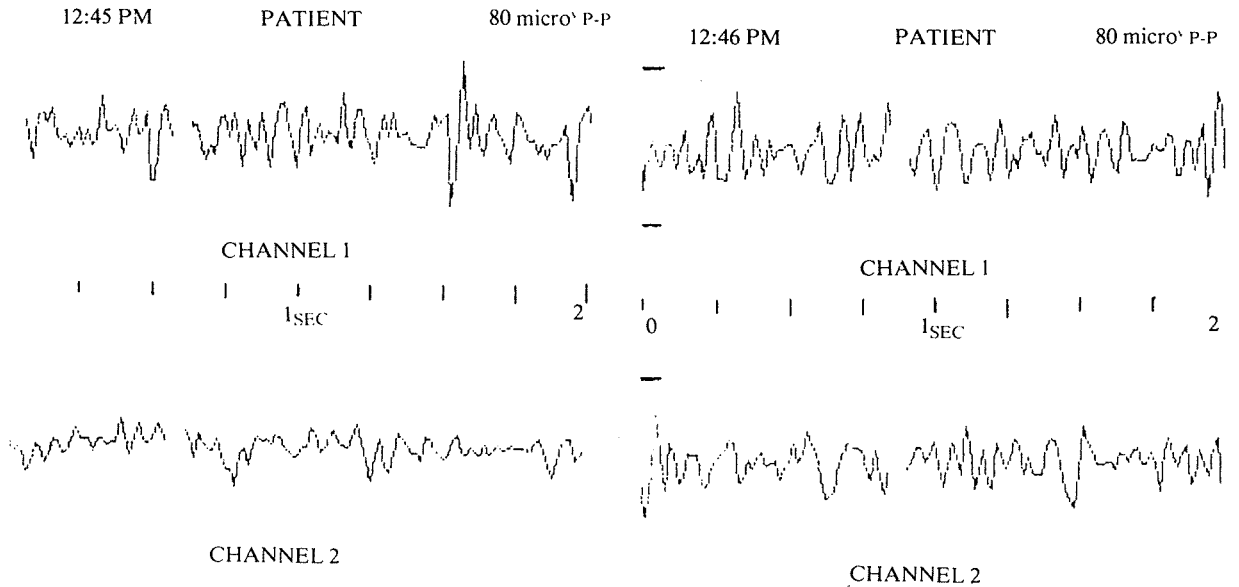
Fig. 2 The powerband analysis before clamping, during clamping and after shunt placement in the same patient. The asymmetry after clamping was prominent after 30 sec. and disappeared less than one minute after shunt opening.

**FIGURE 3**



**Fig. 3** The same phenomena shown by spectral power percentile array (Sppa). The dominating frequency (8-10 Hz) is lost while the total amplitude diminishes on the left side during clamping.

**FIGURE 4**



**Fig. 4** The influence of altering the blood pressure from 105 to 160 mmHg mean during right carotid artery clamping in another patient. The difference in amplitude disappears.

channel EEG recording device: Neurotrac<sup>R</sup>/(Interspec.). Recording electrodes are preoperatively placed in positions F<sub>p1</sub>C<sub>3</sub>, F<sub>p1</sub>C<sub>3</sub>, F<sub>p2</sub>C<sub>4</sub> and F<sub>p2</sub>C<sub>4</sub>. The device allows for a raw signal display and provides different methods of computer aided analysis of this signal: compressed spectral array, spectral power percentile array, power band analysis and spectral histogram. Examples are shown in figures 1 to 4. Second, the pressure in the carotid artery distal to the clamp (stump pressure) is measured. Before clamping all patients receive 7500 units of heparin. A shunt is used whenever stump pressure is lower than 50 mmHg (transducer placed at head level), except when shunt placement is judged to be too risky for causing embolisation or too difficult technically. In these cases our team relies on EEG analysis alone.

Whenever the EEG shows cerebral ischemia a shunt is placed even when the stump pressure is high.

From March '87 till March '88, 129 carotid endarterectomies were done in this way. In 78 cases stump pressure was more than 50 mmHg and in none of these cases did the EEG change significantly. Of these 78 patients 2 had an early postoperative thrombosis and stroke although immediately postoperatively the neurologic examination was normal. The EEG was

asymmetrical at the start of the urgent reoperation. 51 patients had a stump pressure of less than 50 mmHg. In 40 of these cases a shunt was used. In 20 cases EEG did not change during shunt placement and removal and one of these 20 patients had a mild paresis of an arm postoperatively. This paresis was probably due to emboli in a region not detectable by the EEG monitoring used. In the 20 other patients the EEG changed temporarily during shunt placement with rapid normalisation after opening the shunt. One of these patients had a postoperative facialis paresis and one a hemiparesis; both recovered in one week.

In 11 patients with a stump pressure of less than 50 mmHg no shunt was placed, the EEG did not change and none of these patients had postoperative neurologic deficit. All 129 patients survived.

## Conclusion

Our anaesthetic technique for carotid endarterectomy proves to be safe as shown by the morbidity and mortality figures. The combination of EEG monitoring and stump pressure measurement is in our opinion a helpful method for identifying those patients in whom collateral circulation during clamping is insufficient and who would benefit from a shunt.

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