Monitoring in Carotid Endarterectomies

Introduction

In discussing the use of electrophysiological monitoring during carotid endarterectomy one needs to consider the philosophical aspects of monitoring. Monitors only provide information about physiological variables. They rarely initiate any actions on their own. Benefits can therefore only be gained from the appropriate use of the monitoring information obtained so as to improve patient management.

If the information is misinterpreted or the wrong clinical decision is made then harm rather than benefit may result. The monitors themselves may also have intrinsic risks (small with electrophysiology) and may distract us from other important concerns.

A discussion on this topic thus necessarily involves a discussion on management issues especially, in this case, the method of preventing cross clamp related neuronal damage.

The Problem - Cerebral Ischaemia/Infarction

The fundamental reason for the operation is to prevent strokes. The exact risks of this depends on a number of factors including preexisting conditions, preoperative neurological state (ie TIAs, asymptomatic etc.), and the angiographic assessment of the vascular supply. The Mayo risk classification has been used to provide objective classification of these patients. The optimal risk varies from <1% in asymptomatic stenosis to ≤20% in high risk patients with no cerebral protective measures. Perhaps paradoxically, those patients at greatest risk for peri-operative deficits are often those most likely to benefit from surgery.

Given the reasons for surgery it is mandatory that we minimise the risk of surgical and anaesthetic related neurological deficits. In order to do this we have to understand what factors produce deficits.

Essentially deficits may arise from either haemodynamic factors, embolic phenomenon, thrombosis of the operated artery, and hyperperfusion syndromes post-operatively (I have assumed that anaesthetic related hypoxaemia should never occur).

The haemodynamic effects may relate to inadequate blood pressure or to the occlusion of the common carotid artery by the cross clamp during the endarterectomy. Inadequate flow may also occur with shunt malfunction.

Embolic phenomenon occur during arterial dissection, shunt insertion (if used), and re-establishment of flow. This relates directly to the skill of the surgeon and is probably the major cause of deficits but is much higher in some centres than others.

Post operative thrombosis occurs more often with non-patch grafted endarterectomy, in patients in whom anti-coagulation is reversed and probably also relates to the surgical skill in ensuring that no intimal flaps etc. are left.

Much of the discussion has centred on the haemodynamic effects of cross clamping and the methods of detecting and dealing with the decreased flow that often occurs.

Operate for stoke prevention - depends on risk group

Asymptomatic bruits ≈ 4%/yr symptoms

TIA's ≈ 5-7% stroke rate/yr

Successful operation should reduce the stroke rate to 1-2%

Operative risks

Comparing differing series is made very difficult because of the major role that patient risk factors play in determining complications. Many series do not adequately define their patient populations. Without comparing similar populations conclusion about operative, monitoring, and anaesthetic techniques are nearly impossible.

Risk factors: Mayo Classification

Grade I - Neurologically stable, no major angiographic or medical risk

Grade II - Neurologically stable, angiographic risks only

Grade III - Neurologically stable, major medical risks ± angiographic risks

Grade IV - Neurologically unstable,± major medical or aniographic risks

Grade V - known symptomatic acute ICA occlusion, who have progressive neurologic deficit or who have sustained a major neurologic deficit within 4hrs of examination.

nb recurrent stenosis is a separate category in this classification

Neurological Stability

Classified as stable, unstable (progressive deficit or one that has resolved within 24/24 of surgery, generalised ischaemia, cerebral infarction <7/7 old, or TIAs uncontroled by heparin), or known symptomatic acute ICA occlusion, who have progressive neurologic deficit or who have sustained a major neurologic deficit within 4hrs of examination.

Medical risk factors (major)

Angina, MI ≤6/12, advanced PVD, uncontrolled Hypertension (BP >180/110. CAL, age >70, and morbid obesity.

Angiographic risk factors

Controlateral ICA occlusion, stenosis of the ICA in the region of the carotid siphon, extensive atherosclerotic involvement of the vessel to be operated on with extensin of the plaque >3cm distally in the ICA of 5cm proximally in the common carotid, high bifurcation of the carotid at the level of C2 in conjuction with a short neck, evidence of a soft thrombus extending from an ulcerative lesion, and carotid slim sign.

Surgical Technique

Meticulous technique

Patch grafts to Ø rate of thrombosis and recurrent stenosis

X-Clamp time if shunt not used

Longitudinal vs Transverse incision

Morbidity/Mortality

At the Mayo clinic the morbity and mortality risks where: <1% GI , 1.8% GII, 4% GIII, 8.5% GIV.

Cardiac - often main cause of mortality

Neurological - stokes (overall should be ≤ 3%)

The overall figure is somewhat misleading as it depends on the risk group the patient is in. The upper limits of morbidity and mortality that should prompt peer group review have been defined by an Ad Hoc Commitee on Carotid Surgery Standards of the Stroke Council, American Heart Association. The 30 day mortality from all causes should not exceed 2%. The strokerate should not exceed 3% for asymptomatic patients, 5% for TIA patients, 7% for ischaemic stroke, and 10% for recurrent carotid disease in the same artery after endarterectomy.

Embolic - most common but seems to be Ø ing

Haemodynamic

hypotension

head positioning

X-clamp

shunt malfunction

Thrombosis of carotid

Haemorrhage - post operative, intracerebral

Detection of decreased flow

It is clear that cross clamping results in many patients in decreased blood flow but this decrease is not always functionally important.

Neuronal function is what we are interested in. Provide there is an adequate supply of nutrients for the neuronal demand then no dysfunction occurs. Assessment of adequacy of flow therefore needs to consider the demand side of the equation as well as the supply side. The only methods available to do this are those that directly assess neuronal cellular oxygenation (near infrared spectroscopy) or function (the awake patient, EEG, or evoked potentials). Methods that assess blood flow (rCBF techniques such as Xe-133 injections are useful but still only assess one side of the equation. The transcranial doppler provides an assessment velocity which only relates to flow if the cross sectional area of the artery insonated remains constant, this is not always true.

Normal cerebral blood flow (CBF) is 50mls/100gm/min and if demand is "normal" then neuronal function does not begin to be impaired until this falls to ≈25mls/100gms/min (ischaemia). In awake humans or primates neurones become completely electrically dysfunctional at ≈15mls/100gm/min. At ≈10mls/100gms/min cell membranes become dysfunctional, if the decreased flow continues cell death will occur (infarction).

This gap between electrical failure and membrane failure is termed the ischaemic penumbra and is critical in the explanation of how TIAs, RINDs, and the occurrence of transient major EEG changes may fail to produce long term neurological deficits. Essentially if the decrement in blood supply is above the limits of membrane function but below that for electrical activity then the neurones will not function (neurological deficit, EEG/EP abnormalities) but if the blood supply is restored function returns.

The evoked potentials are slightly more resistant to reduced flow than the EEG and will usually be recordable down to flows of ≈15mls/100gm/min. The EEG becomes isoelectric at ≈20mls/100gm/min. It must be noted that this critical flow for the EEG depends on whether or not the patient is anaesthetised and the Mayo Clinic has shown that the rCBF for 50% incidence of EEG changes decreases from ≈20 to 15 to 10 mls/100gm/min whether Halothane/Enflurane/or Isoflurane is used.

Studies with local anaesthesia indicate an overall incidence of neurological changes with cross clamp of ≈10% (but clearly depends on patient population). Studies with GA and EEG show variable rates depending on the criteria for determining important changes. Some report on any changes others require major changes before they use the information. Those that use "major changes" report an incidence of 15-20% and those reporting minor changes have rates <40%. Studies that use evoked potentials and use a 50% decrease in amplitudes as the criteria report a rate of 10% abnormalities.

Overall then there is at least a 10% rate of cross clamp ischaemia. The question is do all these patients if left untreated progress to infarction?

Carotid Stump Pressure

Usually the mean pressure is quoted but CSP has been shown to be extremely unreliable

Kelly 1979 38% of pts with EEG changes had CSP >50mmHg
Kwan 1980 33% of pts who had LOC had CSP > 50mmHg
Mckay 1976 8% of pts with CSP > 50mmHg had rCBF < 18mls/100gm/m
Mcfarland 1988 CSP < 50mmHg 50% had no EEG changes
Brewster 1980 CSP < 50mmHg 53% had no EEG changes
Blackshear 1986 CSP < 25mmHg 77% had EEG changes but sensitivity only 40%
Harada 1995 CSP < 50mmHg 36% had EEG changes
neither CSP/MAP or contralateral artery status changed this

<u>Transcranial Doppler</u>

<u>TCD vs EEG</u>

Jansen 1993

12% had MCAV ↓ > 70%, 69% EEG changes, 31% none (13% with 0 flow) No EEG change, no deficit
42% emboli (only 1pt symptomatic)

Chiesa 1992

TCD couldn't differentiate mild or no EEG changes.
Severe EEG changes all had 0 flow
8% with no EEG changes had ↓↓ flow (no deficits)

McDowel 1995

5 patients with "normal" EEG and ØØ flow, 2 deficits

Gavrilescu 1995

Microemboli: Shunt opening, carotid reopening, wound closure

Jansen et al Stroke 1993

TCD and EEG (?lifescan, details not given)
Selective Shunting on EEG
16/130 had MCAV Ø > 70%
9/16 had marked EEG changes, shunted, no deficits
2/7 moderate EEG changes, no shunt, 1 deficit
5/7 No EEG change, no shunt no deficits
2 of these 5 had MCAV of 0!

(?cortical perfusion via Leptomeningeal Anastomoses)
55/130 (42%) had emboli
54 microemboli, no deficits
1 massive emboli post clamp release, no EEG changes
post op transient deficit

Gavrilescu et al American Journal of Surgery 1995

Microemboli rates during operation (37pts)

Occured:
Shunting opening - 63% (7/11)
Carotid reopening - 62% (23/37)
Wound closure - 59%
Didn't occur during test clamp

McDowell et al Annals of Surgery 1995

238 pts
TCD in all, EEG in most
10 lead EEG
Shunting uncontrolled
5 pts with MCAV 0-15% of baseline and "normal" EEG
2 deficits
5% overall deficit rate

Chiesa et al European J. Vascular Surgery 1992

90pts
TCD and EEG (14 channel)
EEG based shunting (14%)
X-Clamp time 28/60
No and Mild EEG changes - MCAV the same
Severe EEG changes - MCAV 0% in all
5/64 pts with no EEG changes had MCAV 10-30% of baseline
No deficits occured
Also useful for emboli and intimal flap

Regional CBF

Intra-carotid Xenon133 in saline
Single/multiple detectors over parietal boss (MCA territory)\

Near Infrared Spectroscopy

Electroencephalography

raw vs. processed
Number of electrodes
Electrode placement
each electrode mainly detects area relatively near it

Evoked Potentials

Awake Patient

Incidence of Ischaemia

Local Anaesthesia ≈10%

General Anaesthesia

EEG - any change ≈30-40%, major changes ≈15-20%

Evoked Potentials - 50% Ø amplitude N_{20} ≈10%

rCBF - <10mls/100gm/m (Isoflurane) ≈20%

Neuronal Ischaemic Tolerance

The reduction in blood flow below that for membrane failure is only one part of the problem. The duration of flow reduction as well as the magnitude of the reduction is important. 0 flow in normal neurones in tolerable for only 3-4 minutes. Between 0-5mls/100gms/min does not seem to make much difference and may even be worse than a complete cessation of flow. Between 5-10mls/100gm/min neurones will survive longer but for how long is not clear.

As I have said before even if flow is reduced we need to look at demand. Barbiturates have been shown to increase the neural tolerance of ischaemia.

We have evidence from studies where patients have had CEs done without shunts when the EEG is severely effected that the stroke rate is ≈15-20%. One factor that needs to be considered in this would be the cross clamp time. These studies indicate that cross clamp times of under 15 minutes may be tolerated. Certainly the time it takes to insert a shunt (2-5 minutes usually) seems to be almost always tolerated without any special precautions. Most of these studies that report cases where no shunt is used even with EEG changes have cross clamp times of 20-30 minutes.

Clearly the EEG grossly overestimates the number of patients that absolutely need a shunt. EPs have been suggested as preferable because they change at a lower rCBF. Other authors have combined abnormal EEG and very low stump pressures as their criteria.

Cerebral Blood Flow (Supply vs Demand)

Normothermic person with a normal brain
Autoregulation = rCBF proportional to rCMR
Normal CBF ≈ 54mls/100gms/m (average) grey > white
EEG starts to change ≈ 20-25
Loss of function starts at ≈ 20 (awake patient)
Evoked Potentials change ≈ 15-20
EEG flat ≈ 15-20
Membrane failure ≈ 10

K leakage and decreased ATP stores

time dependent cell death
Ischaemic Penumbra
Non-functional electrically but membrane still functional
? Time dependent cell death
Cerebral Protection
≠ supply (≠rCBF)
≠ CPP
Shunt
≠ systemic BP

Robin Hood effect - ? barbiturates/? hypocapnia

Ø Demand - Hypothermia/Barbiturates

Membrane Stabilisation eg Lignocaine coma (experimental)

Problem Detection - ischaemia not stroke detection

Carotid Occlusion ischaemia pattern dependent on:

Feeding Vessel condition

Circle of Willis

MCA distribution, ipsilateral - most common

ACA distribution, ipsilateral

Bilateral

Ischaemia - variable but usually MCA cf. induced ØBP

Anaesthesia

Local Anaesthesia

Awake patient the best detection of ischaemia

General Anaesthesia

Better control of:

Patient movement

Airway

Oxygenation

PaCO2

Surgical/Patient preference

GA lower CBF for neurological compromise

Wells et al 1963

Showed that carotid occlusion that produced delta waves and blacking out in 4/19 patients when awake but same patients under Cyclopropane GA had no EEG changes with X-clamping and awoke with no neurological deficits.

Michenfelder - Iso vs. Halo vs. Enfl

Ischaemia Detection

CSP

Transcranial Doppler

rCBF

Near Infrared Spectroscopy

EEG - Gold Standard

Evoked Potentials

EEG usage

If one uses the EEG then you need to consider the number of electrodes one uses as well as where they are placed. The ischaemia is most commonly in the area of the middle cerebral artery territory. EEG systems that only use 2 channels are quite reliable (>95%) in detecting major EEG changes provided they use electrodes that cover this territory. Nearly all of the articles that criticise these types of system are invalid because they do not cover the appropriate areas of the brain. The other major reason is that the processed 2 channel systems may miss minor changes occurring away from the leads. Of course it is a reasonable argument that, given the EEGs sensitivity, these aren't important.

raw EEG vs. Processed

Raw

Number of leads

≠ numbers = ≠ sensitivity

if only 2 channel position leads over maximally ischaemic area

EEG changes with ischaemia (most within 60s)

Decreased high frequencies

Increased low frequencies

Decreased amplitude

Isoelectric EEG

Processed

Amplitude processing

Frequency processing - pre-filtering of low frequencies

Aperiodic Analysis
Spectral Analysis (Fourier transformation)
Technique
Presentation
CSA
DSA

Ischaemic vs. Anaesthetic - changes similar

Ischaemia Protection

Having decided that one is going to detect the ischaemia the question then is how to deal with it. Personally I feel that the literature compels one to do something about it. The choices are shunts (everyone or selective) or pharmacological protection.

If shunts had no risks then shunting everyone would be logical, however there are real risks to shunts from embolus and intimal damage. As well they limit the exposure and make a complete endarterectomy more difficult. The overall risk in the best hands (Mayo Clinic) gives a risk rate of ≈0.5%. Shunting everyone is therefore not really appropriate if one can be more selective.

Studies that compare stroke rates with and without shunts are very difficult to compare due to radically different patient populations and shunt complication rate.

Barbiturates are used by some either on everyone or selectively instead of shunts. Whilst they have been shown to prevent neuronal damage due to focal incomplete ischaemia they are not beneficial incomplete ischaemia and have problems of their own.

≠ Systemic BP

Temporary shunt

Benefits - improves blood flow

Risks ≈ 0.5% (Mayo Clinic)

Embolism (from or through the shunt)

Intimal damage

More difficulty dissection

Barbiturates

Ø PaCO2

Literature

Problem with Randomised Studies

Inadequate patient numbers to use randomised comparisons

true stroke rate is 5%

treatment reduces this by 50%

Type I 5%

Type II 20%

Æ 874 patients per treatment group!

Approaches

No shunts

Increased BP during X - clamping

Selective shunting

All shunted

Cerebral Barbiturate Protection for all

Selective Cerebral Barbiturate Protection

Things to keep in mind when looking at studies

Differing patient populations

Differing Anaesthetic techniques

GA vs. LA

If GA what anaesthetic agents used

Blood Pressure control

EEG type/electrode placement

Definition of EEG/EP/TCD change

Duration of Monitoring

Differing Surgical Techniques

shunts/no shunts/selective shunting/barbiturates

X-Clamp time

Patch grafts

Heparin reversal

EEG studies that look at subgroups where the monitoring was not used or not fully used

Fletcher et al

ANZ J. Surg 1988

142pts (randomised)

- **No X-clamp, Anaesthetic details**

G1 (72) EEG/CSP

- **Shunt if CSP <50 and EEG changes (12.7%)**

G2 (70) CSP

- **Shunt if CSP <50 (25.7%)**

Results:

- **If shunted no complications**
- **CSP ≥50 and EEG changes 40% (2/5) had a stroke**

CSP <50 and no EEG changes 0% (0/14) had a stroke

Ferguson et al

Arch Neurol 1986

211 pts

All EEG monitoring but no shunts

113 had CSP

X-clamp time ≈30/60

8% (9/113) had CSP < 25mmHg all had major EEG changes

22% of these (2/9) had a stroke

15% (4/26) with EEG changes had a stroke

2 thought to be embolic (on angio/CT)

2 thought to be haemodynamic

0% (0/148) with no EEG changes had a stroke

0% (0/37) with moderate EEG changes had a stroke

Redekop and Ferguson

Neurosurgery 1992

293 pts 77 with contralateral stenosis(>70%)/occlusion

All EEG monitoring but no shunts

X-clamp time ≈30/60

Major EEG changes with X-clamp

14.3% (11/77) with contralateral disease

5.1% (11/216) without contralateral disease

Immediate deficits:

2.6% (2/77) with contralateral disease

3.2% (7/216) without contralateral disease

1.4% (3/221) without EEG changes

4% (2/50) with moderate EEG changes

18.2% (4/22) with major EEG changes

Collice et al

J. Neurosurgery 1986

141 pts

Shunting if EEG changes and X-clamp expected > 30/60

“mild” hypertension used prior to X-clamp

Major EEG changes with X-clamp

- **14% (2/141) overall**
- **10% (12/119) without contralateral occlusion**
- **36% (8/22) with contralateral occlusion**

Deficits

- **0% (0/121) No EEG changes, X-clamp 37/60**
- **0% (0/8) EEG changes, shunt within 3-15/60**
- **25% (3/12) EEG changes and X-clamp > 15/60**
- **67% (2/3) EEG changes, shunt after 16-30/60**
- **11% (1/9) EEG changes, no shunt, X-clamped for 22/60**

Zampella et al

Neurosurgery 1991

431 pts

No shunting

EEG and rCBF measurements

X-clamp “20-30/60”

14% X-clamp EEG changes

rCBF

- **24% <13 mls/100gms/m (32% EEG changes)**
- **76% ≥13 (8.2% EEG changes)**

Deficits

- **15% (9/59) with EEG changes**
- **2.9% (11/272) without EEG changes**
- **3.2% (2/63) rCBF <9**
- **5.1% (2/39) rCBF 9-13**

4.8% (16/329) rCBF >13

There was a significant relationship between defecits and higher flows in those patients with EEG changes.

Morawetz et al

Surgery 1984

129 pts

No shunting but increased BP with X-clamp

EEG and rCBF

X-clamp time "20-30/60"

19% (25/129) EEG changes

6.9% (9/129) rCBF< 9mls/100gm/m (87% had EEG changes)

Deficits

4% (1/25) with EEG changes

3.8% (4/104) without EEG changes

0% (0/8) with rCBF < 9

7% (1/14) with rCBF 9-13

3.7% (4/107) with rCBF >13

80% new deficits not predicted by EEG

EEG studies demonstrating some problems

Kresowik et al

J. Vascular Surg 1991

458 pts

EEG guided shunting (16 channel)

EEG not always continued till end of case

15% (70/458) shunted

10% (1/10) had X-clamp changes which reversed with ≠ BP

50% of strokes (5/10) had normal EEGs at X-clamp and no shunts

100% (5/5) of transient deficits had the same

nb all strokes mild with marked resolution over 1-4/52

Rosenthal et al

Arch Surg 1981

125 pts with stroke or RIND

Shunting in three stages

36 shunt on surgical preference

- **8% permanent deficits (all not shunted)**

36 selective shunting on EEG (18% shunted)

- **9% deficits (all not shunted)**

41 all shunted, also had EEGs

- **2% deficits**

All post operative deficits the same as at the initial stroke or RIND

EEG may be unreliable in this group of patient

False Negative Rates

McFarland et al 1988 377pts no false -ves

Sundt et al 1992 1900pts no false -ves

Cho et al 1986 172pts no false -ves

Chiapa 1979 367pts no false -ves

Collice 1986 141pts no false -ves

Transcranial Doppler Studies

Chiesa et al

Eur J Vas Surg 1992

90 pts

TCD vs EEG

EEG used for shunting

X-clamp time 28/60

14% (13/90) shunted

Compared %MCAV with none, moderate, and major EEG changes

- **4 patients with major changes had 0 velocities**
- **no and moderate EEG change grps same %MCAV Ø**

Also useful for emboli and intimal flap detection

Jansen et al

Stroke 1993

130 pts

TCD vs EEG (?Lifescan, details not given)

EEG used for shunting (6.9%) nb very low figure

69% (11/16) with ≥70% Ø MCAV had EEG changes

40% (2/5) with no EEG changes had %MCAV of 0! (0 deficits)

?cortical perfusion via leptomengeal anastomoses

1 pt with massive embolus post X-clamp had no EEG

changes but had transient arm weakness post op

McDowel et al

Ann Surg 1992

238 pts

TCD in all, EEG in "most"

Uncontrolled shunt usage

9.2% (22/238) of cases ≥85% Ø in MCAV

Deficits

0% (0/13) shunted

44% (4/9) not shunted

22% (5/22) no EEG changes and not shunted

40% (2/5) had deficits

Bergeron et al

Cardiovascular Surg 1991

114 pts

16 pts had TCD and LA

Shunts on neurological changes

15.9% (19/114) shunted

0% Complications

MCAV Ø

20-29cm/s: 20% (1/5) needed shunting

<19: 100% (1/1) needed shunting

Somatosensory Evoked Potentials Studies

SSEPs vs EEG

Fava 1992

21% abnormal EEG but only half abnormal SSEPs

Abnormal EEG, "Normal" SSEP, no shunt, no deficits

Lam 91

9% Abnormal EEG, 67% of these had abnormal SSEPs

No shunts, 9.3% EEG changes had deficits, 17% with SSEPs

1 transient deficit had a "normal" EEG and reversible SSEP Ø

Kearse 1992

44% EEG changes but only 1 of these had 50% Ø amplitude

D'Addato et al

Eur J Vasc Surg 1993

72pts

SSEPs vs CSP

Shunted on SSEPs (23%) (>49% Ø in $N_{20}P_{25}$amplitude)

42% of those with a CSP < 50mmHg had normal SSEPs

No patient with normal SSEPs had a deficit

Fava et al

Electroencephalography and Clinical Neurophysiology 1992

151 pts

EEG and SSEPs

Shunted only if EEG and SSEPs markedly abnormal

21% (31/151) markedly abnormal EEG

11% (16/151) plus markedly abnormal SSEPs, shunted

10% (15/151) SSEPs less effected, not shunted (≠ BP)

0.8% (1/120) normal EEG(no shunt) had a deficit

12.5% (2/16) shunted had a deficit

0% (0/15) marked EEG changes and no shunt

1 patient with a "normalish" EEG had sudden loss of SSEPs 24/60 after X-clamp awoke with a deficit

Horsch et al

J Cardiovasc. Surg 1990

675 pts

SSEP guided shunting(>50% reduction in amplitude)

12% (81/675) shunted

0.6% (4/586) had a deficit despite "normal" SSEPs

2.2% (2/89) of those shunted had a deficit with loss of SSEPs during shunting

100% (6/6) with irreversible loss of SSEPs had deficits

Lam et al

Anesthesiology 1991

67 pts

EEG vs SSEPs

None shunted

9% (6/64) had abnormal X-clamp SSEPs (>50% Ø amplitude)

9% (6/64) had markedly abnormal X-clamp EEGs

67% (4/6) with abnormal EEGs had abnormal SSEPs

9.3% with EEG changes had deficits

17% with SSEP changes had deficits

1 pt with a transient deficit had a normal EEG and reversible SSEP changes

Kearse et al

Stroke 1992

53 pts

EEG vs SSEP

Shunt on any EEG changes

44% (23/53) EEG changes

4.3% (1/23) had > 50% Ø in amplitude of SSEPs

Mayo Clinic

1935pts

Shunt on EEG and rCBF

EEG any changes

rCBF < critical level (15mls/100gm/m)

patients with abnormal EEGs due to ischaemia preop were all shunted if there was a "significant fall in rCBF with X-clamp

18% EEG changes seen when Isoflurane used

37% shunted when Isoflurane used

"In our experience we have not had a patient awaken with a neurological deficit that had not been detected by the EEG."

Raw vs Processed EEGs

Chiapa et al 1987

EEG vs CSA (4 channels) MCA leads

CSA better

Hanowell et al 1992

EEG vs Power analysis and Lifescan (aperiodic analysis)

Power detected 100% (9/9) changes

Lifescan 22% (2/9) changes

Kearse et al 1992

EEG vs 4 channel DSA (MCA leads)

DSA detected EEG changes

61% mild

70% moderate

95% marked

Spackman et al 1987

Lifescan (frontomastoid leads) vs EEG

78% changes detected with Lifescan

2 false negatives

Silbert et al 1989

Lifescan (frontomastoid leads) vs LA

1/6 pts with X-clamp changes had no EEG changes

5/70 pts had EEG changes without neurological changes

Barbiturate Protection

Frawley et al

Journal of Vascular Surgery 1996

CE with contralateral carotid disease - 259 operations

205 > 70% stenosis

54 occluded

All Thiopentone >60s burst supression, no shunts

X-clamp time 35 minutes

19% asymetric bursts on X-clamp (cf 15-50% in other studies)

1.9hrs to wake up

Only 1 "immediate" deficit (intimal flap), recovered

Expected immediate deficit rate 6 (259 * .15 * .15)

Frawley et al

Journal of Vascular Surgery 1994

CE - 621 operations

All Thiopentone 30-60s burst supression, no shunts

X-clamp time 35 minutes

1.9hrs to wake up

2 "immediate" deficits

1 haemorrhage

1 normal intraop EEG, ? embolus in ICU

Expected immedicate deficit rate 9 (621 * .10 * .15)

Besser et al 1997

CE 261 operations

2 Channel EEG

Selective Methohexitone 5-10 s burst supression, 1 shunt

X-clamp time 43 minutes

10% X-clamp EEG changes

1.9% strokes but only 1 case intra-op (with shunt)

AMI 0.3% (non-fatal)

Konstadinos et al

Journal of Vascular Surgery 1997

Selective Shunting (EEG) vs No Shunts

902 consecutive cases, same surgeons

591 - no shunts, no EEGs (1980-88)

311 - selective shunting on EEG changes (1988-94)

Later grp sl. older, more HTN, more re-dos

12.8% shunted, X-clamp time not stated

Stroke Rates

No Shunt grp - 2.19%

Selective Shunts - 0.32% (occured pre X-clamp) ($p < 0.05$)

Costs

In the USA in 1983 it was estimated that the cost of supporting a person with paraplegia for the rest of their life was $200,000. The cost of hemiplegia may well be less than this but inflation would provide a figure at least as high as this.

If we assume that the incidence (overall) of major cross clamp ischaemia is ≈10% (and this will be higher in sub-groups) and that of these 15% will have a neurological deficit if they are not shunted, then the incidence of preventable deficits (from this cause) is 1.5% of all cases. Therefore the cost to the community per case is $3,000 from neurological deficits. If shunting prevented this, and all patients were shunted, but it had its own complication rate of 0.5% then the cost would be reduced to $1,000 per case. If selective shunting, on the basis of monitoring, was used then the rate would be reduced to 0.05% of all cases (90% of patients would not need a shunt, 10% are shunted and 0.05% have a neurological complication of shunting). This reduces the cost per case to $100. So if monitoring can be provide for less than $900 per case it is cost effective. In other words selective shunting, in all carotid endarterectomies, should prevent 1% of patients having a neurological deficit.

Hemiplegia ≈$200,000

X-clamp related deficits = 0.10 * 0.15 = 1.5% of all patients

Cost per case of neurological injury = $3,000

If we shunt all patients then 0.5% of all cases will have a deficit = $1,000/case

If we selectively shunt only 10% then 0.05% will have a deficit $100/case

Selective shunting cost effective if it can be done for <$900/case

How I do it

I monitor all CEs with a 2-channel CSA and raw EEG system monitoring the MCA territory. Ideally I would prefer to shunt on changes but the surgeon I work with usually claims that it is technically not possible so we then use barbiturate protection with methohexitone.

Machine

Electrode Placement

Decision tree

Conclusions

All patients having CEs should have neuronal monitoring (awake or electrophysiological) and have selective cerebral protection (either shunts or barbiturates)

EEG useful to indicate who should be shunted/barbituratised and for prognosis

Normal EEG Æ >98% will be normal

Abnormal EEG Æ > 99% will be abnormal

Ø operative risk

detection and treatment of reversible ischaemic events

related to Ø shunt usage (or ≠ if use none)

slower more careful dissection (Ø embolus/thrombosis rate)

detection of shunt problems

detection of intraoperative carotid thrombosis

Minimisation of induced hypertension - Ø cardiac risk

CSP not worthwhile

Control of Barbiturate dosing

? Treat if rCBF/TCD very low but EEG unchanged

? reliable in recent stroke/RIND

Case reports

Mr Waters

Mr Ritano - amaurosis fugax

Unknown

References (Carotid Endarterectomy and monitoring, plus some general articles)

American Electroencephalographic Society: Guidelines in EEG: Standards of Practice in Clinical Electroencephalography and Minimal Technical Requirements for Performing Clinical EEG. American EEG Society, Tucker, Georgia, 1980

Artru AA, Merriman HG: Hypocapnia added to hypertension to reverse EEG changes during carotid endarterectomy. Anes, Vol 70:1016-1018, No 6, June 1989

Baker AB, Roxburgh AJ : Computerised EEG monitoring for carotid endarterectomy. Anaesthesia and Intensive Care, Vol 14:32-36, No1, Feb 1986

Bergeron P, Benichou H, Rudondy P, Jausseran JM, Ferdani M; Courbier: Stroke prevention during carotid surgery in high risk patients (value of transcranial doppler and local anesthesia). Cardiovasc Surg 32;713-719;1991

Blackshear WM, DiCarlo V, Seifert KB, Connar RG : Advantages of continuous electroencephalographic monitoring during carotid artery surgery. J Cardiovasc Surg, 27, 1986

Blume WT, Ferguson GG, McNeill K: Significance of EEG Changes at Carotid Endarterectomy. Stroke 1,7 891-897, 1986

Bowdle TA, Rooke A, Kazmers A: Intraoperative stroke during carotid endarterectomy without a change in the spectral edge frequency of the compressed spectral array. J Cardiothorac Anesth 2(2): 204-206, l988.

Brewster DC, O'Hara PJ, Darling CR, Hallett JW: Relationship of intraoperative EEG monitoring and stump pressure measurements during carotid endarterectomy. CIRCULATION Vol 62, Suppl I, August 1980

Bridgers SL, Spencer SS, Spencer DD, Sasaki CT: A cerebral effect of carotid sinus stimulation: observation during intraoperative electroencephalographic monitoring. Arch Neurol 42:574-577, 1985

Chemtob G, Kearse LA Jr: The use of electroencephalography in carotid endarterectomy. Int Anesth Clin 28:143-146, 1990

Cherry KJ, Roland CF, Hallett JW, Gloviczki P, Bower TC,. Toomey BJ, Pairolero PC: Stump pressure, the contralateral carotid artery, and electoencephalographic changes. Amer.Journal Surgery 162:185-189:1991

Chiappa KH, Burke SR, Young RR : Results of electroencephalographic monitoring during 367 carotid endarterectomies. STROKE Vol 10, No 4, July-August, 1979

Cho I, Smullens SN, Streletz LJ, Fariello RG : The value of intraoperative EEG monitoring during carotid endarterectomy. Annals of Neurology: Vol 20, No 4, October 1986

Clark DL, Hosick EC, Rosner BS: Neurophysiological effects of different anesthetics in unconscious man. J Appl Physiol 31:884-891, 1971

Collice M, Arena O, Fontana RA, Mola M, Galbiati N: Role of EEG monitoring and cross-clamping duration in carotid endarterectomy. J Neurosurg 65:815-819, 1986

Davenport J : The value of intraoperative monitoring during carotid endarterectomy: A comment. Annals of Neurology: Vol 22, No 2, August 1987

Deriu GP, Ballotta E, Franceschi L, Facco E : EEG Monitoring, selective shunting and patch graft angioplasty in carotid endarterectomy. Early and longterm results. J Cardiovasc Surg, 29, 1988

Elmore JR, Eldrup-Jorgensen J, Leschey WH, Herbert WE, Dillihunt RC, Ray FS: Computerized topographic brain mapping during carotid endarterectomy. Arch Surg 125; 734-739: 1990

Evans WE, Hayes JP, Waltke EA, Vermilion BD: Optimal cerebral monitoring during carotsid endarterectomy:Neurologic response under loca l anesthesia. vasc.Surg 2/6;775-777;1985

Fava E, Bortolani E, Ducati A, Schieppati M:Role of SEP in identifying patients requiring temporary shunt during carotid endarterectomy. Electro & Clin Neurophysiology 84;426-432;1992

Ferguson GG : Carotid Endarterectomy - To shunt or not to shunt ? Arch Neurol: Vol 43, June 1986

Ferguson GG : Intra-Operative monitoring and internal shunts : Are they necessary in carotid enderterectomy? STROKE Vol 13, No 3, May/June, 1982

Fletcher JP, Morris GL, Little JM, Kershaw LZ : EEG Monitoring during carotid endarterectomy. Aust. N.Z. Surg. 1988, 58: 285-288

Fode NC, Sundt TM, Robertson JT, Peerless SJ, Shields CB: Multicenter retrospective review of results and complications of carotid endarterectomy in l981. Stroke 17:370-376, 1986

Freye E: Cerebral Monitoring in the Operating Room and the Intensive Care Unit. Kluwer Academic Publishers, The Netherlands, 1990

Gewertz BL, McCaffrey M: Intraoperative monitoring during carotid endarterectomy. Curr. Probl. Surg., 480-532, 1987

Graham AM, Gewertz BL, Zarins CK : Predicting cerebral ischemia during carotid endarterectomy. Arch Surg: Vol 121, May 1986

Green MG, Messick WJ, Ricotta JJ, Charlton MH, Satran R, McBride MM, DeWeese JA: Benefits, Shortcomings, and Costs of EEG Monitoring. Ann Surg. June 85; 785-792

Green RM, Messick WJ, Ricotta JJ, Charlton MH, Satran R, McBride MM, DeWeese JA: Benefits, shortcomings and costs of EEG monitoring. Ann Surg 201:785-792, 1985

Gross CE, Adams HP, Sokoll MD, Yamada T : Use of anticoagulants, electroencephalographic monitoring, and barbiturate cerebral protection in carotid endarterectomy. Neurosurgery, 9: 1-5, July 1981

Grundy BL, Sanderson AC, Webster MW, Richey ET : Hemiparesis following carotid endarterectomy: Comparison of monitoring methods. ANESTHESIOLOGY, Vol 55:462-466, No 4, Oct 1981

Hafner CD : Minimizing the risks of carotid endarterectomy. Journal of Vascular Surgery, Volume 1, Number 3, May 1984

Halsey JH, McDowell HA, Gelmon S, et al: Blood velocity in the middle cerebral artery and regional cerebral blood flow during carotid endarterectomy. Stroke 20:53-58, 1989

Hansebout RR, Blomquist G, Gloor P, Thompson C, Trop D : Use of hypertension and electroencephalographic monitoring during carotid endarterectomy. Can.J.Surg, 24:304-307, May 1981

Haupt WF, Horsch S: Evoked potential monitoring in carotid surgery. Neurology 42;835-838;1992

Hicks RG, Kerr DR, Horton DA : Thiopentone cerebral protection under EEG control during carotid endarterectomy. Anesthesia and Intensive Care, Vol. 14, No. 1, February, 1986.

Horsch S, De Vleeschauwer P, Ktenidis K: Intraoperative assessment of cerebral ischemia during carotid surgery. Cardiovas Surg 31;599-602;1990

Jansen C, Vriens EM, Eikelboom BC, Vermeulen FEE, van Gijn J, Ackerstaff RGA: Carotid endarterectomy with transcranial doppler and electroencephalographic monitoring. Stroke 24/5:665-669;1993

Jenkins GM, Cjiappa KH, Young RR : Practical aspects of EEG monitoring during carotid endarterectomies, Am. J. EEG Technol. Vol. 23, 191-203, 1983

Kaieda R, Todd MM, Warner DS: The effects of anesthetics and PaCO2 on the cerebrovascular, metabolic, and electroencephalographic responses to nitrous oxide in the rabbit. Anesth Analg 68:135-143, 1989

Kane CM, Le Cheminant D, Horan BF : Acute cardiovascular responses to internal carotid artery occlusion during carotid endarterectomy and to the restoration of internal carotid flow. Anaesthesia and Intensive Care: Vol 15:289-295, No 3,August 1987

Kearse LA Jr, Brown EN. McPeck: Somatosensory evoked potentials sensitivity relative to electroencephalography for cerebral ischemia during carotid endarterectomy. Stroke 23/4;498-505;1992

Kearse LA, Martin D. McPeck K, Lopez-Bresshahan M. Computer-derived density spectral array in detection of mild analog electroencephalographic ischemic pattern changes during carotid endarterectomy: J Neurosurg 78;884-890:1993

Krul JMJ, van Gijn J, Ackerstaff RGA, Eikelboom BC : Site and pathogenesis of infarcts associated with carotid endarterectomy. Stroke Vol 20, 22-28, No. 3, March 1989

Lam AM, Manninen PH, Ferguson GG, Nantau W: Monitoring electrophysiologic function during carotid endarterectomy: a comparison of somatosensory evoked potentials and conventional electroencephalogram. Anesthesiology 75:15-21, 1991

Lam AM, Manninen PH, Gerfuson GG, Nantau W: Monitoring Electrophysiologic function during carotid endarterectomy: A comparison of somatosensory evoked potentials and conventional electroencephalogram. Anesth 59:A368;15-21;1983

Lanier WL: Cerebral function monitoring during carotid endarterectomy. J Neurosurg Anesth 1:207-210, 1989

Legatt AD: Intraoperative Neurophysiologic Monitoring. In Clinical Anesthesia in Neurosurgery, edited by Elizabeth A.M. Frost. Butterworth Heinemann, Boston 1990, p.63

Matsumoto GH, Baker JD, Watson CW, Gleucklich B, Callow AD : EEG surveillance as a means of extending operability in high risk carotid endarterectomy. STROKE, Vol 7, No 6, Nov-Dec 1976

McDowall DG: Monitoring the brain. Anesthesiology 45:117-34, 1976

McDowell HA, Gross GM, Halsey JH: Carotid endarterectomy monitored with transcranial doppler. Ann.Surg.215,5;514-519;1992

McFarland RH, Pinkerton JA, Frye D : Continuous electroencephalographic monitoring during endarterectomy. J Cardiovasc Surg, 29, 1988

McKay RD, Sundt TM Jr, Michenfelder JD, et al: Internal carotid artery stump pressure and cerebral blood flow during carotid endarterectomy: Modification by halothane, enflurane and Innovar. Anesthesiology 45:390-399, 1976

McMeniman WJ, Kam PCA : Cerebral perfusion and cerebral protection during carotid endarterectomy. Anaesth. Intens. Care Vol 11, No. 3, August, 1983

McMeniman WJ, Purcell GJ: Neurological monitoring during anaesthesia and surgery. Anaesth Intens Care 16:358-367, 1988.

Messick JM Jr, Casement B, Sharbrough FW, et al: Correlation of regional cerebral blood flow (rCBF) with EEG changes during isoflurane anesthesia for carotid endarterectomy: Critical rCBF. Anesthesiology 66:344-349, 1987

Meyer FB. Fode NC, Marsh WR, PPiepgras DG: Carotid endarterectomy in patients with contralateral carotid occlusion: Mayo Clin Proc 1993: 337-342: 1993

Modica PA, Tempelhoff R: A comparison of computerized EEG with internal carotid artery stump pressure for detection of ischemia during carotid endarterectomy. J Neurosurg Anesth 1:211-218, 1989

Mola M, Collice M, Levati A: Continuous intraoperative electroencephalographic monitoring in carotid endarterectomy. Eur Neurol 25:53-60, 1986

Morowetz RB, Ziegler HE, McDowell HA, McKay RD, Varner PD, Gelman S, Halsey JH: Correlation of cerebral blood flow and EEG changes during carotid occlusion for endarterectomy (without shunting) and neurologic outcome. Surgery 96:184-189, 1984

Naylor AR, Wildsmith JAW, McClure J, Mcl.Jenkins A, Ruckley CV: Transcranial doppler monitoring during carotid endarterectomy. Br J Surg 78:1264-1268;1991

Peitzman AB, Webster MW, Loubeau JM, Grundy BL, Bahnson HT : Carotid endarterectomy under regional (conductive) anesthesia. Ann.Surg., 196:59-64, July 1982

Phillips MR, Johnson WC, Scott RM, Vollman RW, Levine H, Nabseth DC; Carotid Endarterectomy in th presence of contralateral carotid occlusion. Arch Surg 114:1232-1239;1979

Prior PF : EEG monitoring and evoked potentials in brain ischaemia. Br. J. Anaesth. Vol. 57, 63-81, 1985

Rampil IJ, Holzer JA, Quest DO, Rosenbaum SH, Correll JW : Prognostic value of computerized EEG analysis during carotid endarterectomy. ANESTH ANALG 1983, 62:186-92

Reddy K, West M, Anderson B : Carotid endarterectomy without indwelling shunts and intraoperative electrophysiologic monitoring . Can.J.Neurol.Sci. Vol 14:131-135, No 2, May 1987

Redekop G, Ferguson G: Correlation of contralateral stenosis and intraoperative electroencephalogram change with risk of strtoke during carotid endarterectomy. Neuro.30/2;191-194;1992

Rosenthal D, Stanton PE, Lamis PAA: Carotid Endarterectomy; Arch Surg 116:1569-1575, 1981

Sharbrough FW, Messick Jr JM, Sundt Jr TM: Correlation of continuous electroencephalograms with cerebral blood flow measurements during carotid endarterectomy. Stroke 4:674-683, 1973

Sibert BS, Koumoundouros E, Davies MJ, Cronin KD : Comparison of the processed electroencephalogram and awake neurological assessment during carotid endarterectomy. Anaesthesia and Intensive Care: Vol 17:298-304, No3, August 1989

Silbert BS, Kluger R, Cronin KD, Koumoundouros E : The processed electroencephalogram may not detect neurologic Ischemia during carotid endarterectomy. ANESTHESIOLOGY, Vol 70:356-358, No 2, Feb 1989

Silbert BS, Koumoundouros E, Davies MJ, Cronin KD : The use of Aperiodic Analysis of the EEG during carotid artery surgery. Anaesthesia and Intensive Care, Vol.17:16-23, No1, Feb 1989

Spackman TN, Faust RJ, Cucchiara RF, Sharbrough FW: A comparison of aperiodic analysis of the EEG with standard EEG and cerebral blood flow for detection of ischemia. Anesthesiology 66:229-231, 1987.

Sundt Jr TM, Sharbrough FW, Piepgras DG, Kearns TP, Messick Jr JM, O'Fallon WM: Correlation of cerebral blood flow and electroencephalographic changes during carotid endarterectomy: with results of surgery and hemodynamics of cerebral ischemia. Mayo Clin Proc 56:533-543, 1981

Sundt TM : The Ischemic tolerance of neural tissue and the need for monitoring and selective shunting during carotid endarterectomy. STROKE Vol 14, No 1, Jan/Feb 1983

Sundt TM Jr, Sharbrough FW, Piepgras DG, et al: Correlation of cerebral blood flow and electroencephalographic changes during carotid endarterectomy. Mayo Clin Proc 56:533-543, 1981

Sundt TM, Sharbrough FW, Marsh RW, Ebersold MJ : The risk-benefit of intraoperative shunting during carotid enderterectomy. Vol. 203, No2 , Ann.Surg, February 1986

Sundt TM, Sharbrough, Anderson RE, Michenfelder JD : Cerebral blood flow measurements and electroencephalograms during carotid endarterectomy. J.Neurosurg. Vol 41, Sept 1974

Trojaborg W, Boysen G: Relations between EEG, regional cerebral blood flow and internal carotid artery pressure during carotid endarterectomy. Electroenceph Clin Neurophysiol 34:61-69, 1973

Wells BA, Keats AS, Cooley DA : Increased tolerance to cerebral ischaemia produced by general anesthesia during temporary carotid occlusion. Surgery, July 1963

Wilkins DE : Intraoperative monitoring during carotid endarterectomy. Annals of Neurology: Vol 22, No 2, August 1987

Young WL, Moberg RS, Ornstein E, Matteo RS, Pedley TA, Correll JW, Quest DO, Schwartz AE: Electroencephalographic monitoring for ischemia during carotid endarterectomy: visual versus computer analysis. J Clin Monit 4:78-85, l988

Zampella E, Morawetz RB, McDowell HA, Zeiger HE, Varner PD, McKay RD, Halsey Jr JH: The importance of cerebral ischemia during carotid endarterectomy. Neurosurg 29:727-731, 1991