ELECTRICAL ACTIVITY OF THE CEREBRAL CORTEX DURING INDUCED HYPOTENSION IN MAN
A Comparison of Sodium Nitroprusside and Trimetaphan

W. A. THOMAS, P. V. COLE, N. J. ETHERINGTON, P. F. PRIOR AND S. B. STEFANSSON

Since the advent of controlled hypotension during anaesthesia and surgery in the 1950s, doubts have been expressed as to the adequacy of the cerebral circulation during the hypotensive period. Critical values of cerebral perfusion pressure (CPP), their correlations with the EEG and evoked potentials, and the pattern of brain damage produced by inadequate CPP were defined in the Rhesus monkey by Brierley and colleagues (1969) and Meldrum and Brierley (1969). There is evidence that cerebral perfusion and oxygen metabolism are better maintained with sodium nitroprusside (SNP) than with trimetaphan (TMP) in the dog (Stoyka and Schutz, 1975; Michenfelder and Theye, 1977) and the cat (Maekawa, McDowall and Okuda, 1979). In 1981, Ishikawa and McDowall confirmed that cerebral blood flow in the cat was greater at low arterial pressures with SNP than with TMP. In addition, they demonstrated a close relationship between the EEG (both assessed visually and processed by the Cerebral Function Monitor (CFM)) and the cortical cerebral blood flow. If these animal models are relevant to man, SNP would seem to be the agent of choice for induced hypotension.

To obtain an objective comparison of SNP and TMP during induced hypotension in man, we designed a controlled, single-blind study, with monitoring of the EEG (primarily by the CFM) to give presumptive evidence of the adequacy of cerebral perfusion. Since no anaesthetic technique used deviated from the standard practice of this hospital, no ethical approval was considered necessary.

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SUMMARY
During the routine use of controlled hypotension the electroencephalogram (EEG) and mean arterial pressure (MAP) were monitored in 20 normotensive patients (younger than 70 years-of-age) receiving either trimetaphan (TMP) or sodium nitroprusside (SNP). The reduction in MAP was quicker and greater with SNP. Significant differences in EEG voltage between the two agents were seen in the range 55–40 mm Hg, electrical activity being better maintained with SNP. However, all patients showed some decline in EEG voltage with hypotension and half of these showed significant correlations with MAP. These pressure-dependent cerebral effects were not predictable in terms of age, preoperative arterial pressure or hypotensive agent. Our work supports previous experimental evidence that, during more profound hypotension, cerebral electrical activity is better maintained with SNP than with TMP. A simple measure of total EEG power, or filtered EEG voltage envelope (CFM) was shown to be a more useful monitor of cerebral electrical activity during controlled hypotension than measurements of power distribution in different frequency bands.

PATIENTS AND METHODS
Twenty patients undergoing major orthopaedic, gynaecological and plastic surgical procedures for which induced hypotension was routine practice, were allocated randomly to two groups (table I). One group (mean age 53 ± 10.2 yr) received SNP, and the other (mean age 47 ± 15.3 yr) received TMP. Patients were excluded if they were over 70 years-of-age; their diastolic arterial pressure exceeded...
TABLE I. Details of patients undergoing surgery and controlled hypotension with sodium nitroprusside (SNP) or trimetaphan (TMP). Data include age, sex, MAP, arterial Po$_2$ and carbon dioxide tensions and duration of hypotension, as well as the arterial pressure at which average CFM voltage became pressure dependent. Significance of differences between mean values for SNP and TMP compared using Student’s $t$ test.

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100 mm Hg before operation; they had historical or clinical evidence of cardiovascular, cerebrovascular, renal or neurological disease.

All the patients were screened by one investigator before operation and premedicated with papaveretum 0.2 mg kg$^{-1}$ and hyoscine 4 g kg$^{-1}$. After induction of anaesthesia and tracheal intubation, artificial ventilation (60% nitrous oxide and 0.5% halothane in oxygen) was instituted, neuromuscular blockade being provided by tubocurarine 0.5 mg kg$^{-1}$. Before surgery, all patients received fentanyl 2-4 g kg$^{-1}$. TMP was prepared as a 0.2% solution in 5% dextrose and infused at a rate not exceeding 10 mg kg$^{-1}$ h$^{-1}$ with an automatic infusion controller. SNP 0.01% in 5% dextrose was infused at a rate so as not to exceed a total dose of 1.5 mg kg$^{-1}$ (Cole, 1978). Propranolol in 1-mg increments was administered if the mean arterial pressure (MAP) failed to reach the target value of 40 mm Hg with the maximum dose rate of hypotensive agent, or if the patient developed a tachycardia, defined as a heart rate greater than 80 beat min$^{-1}$.

MAP and end-tidal carbon dioxide concentration were displayed continually and recorded, on a chart recorder and a Racal "Store 4" tape recorder. Sterile intradermal EEG needle electrodes were placed in the left and right parietal regions, overlying the arterial boundary zones, with a third "guard" electrode in the mid-frontal region. The electrode sites were standardized by the use of a plastic template. The EEG signal was processed and displayed by a CFM on a polygraphic chart and both raw and processed signals stored on the tape recorder. Intermittent blood samples were taken to confirm normal alveolar ventilation, and to ensure adequate oxygenation and absence of metabolic acidosis.

The chart and tape recordings were marked with a numerical code for timing of events such as drug administration. They were analysed blind by two investigators not involved in the acquisition of the data.
Measurements were made manually of MAP and simultaneous average and minimum CFM voltage from the start of the administration of the hypotensive agent until arterial pressure had returned to a steady value once the drug had been discontinued.

The measurements were made at 10-s intervals during the initial decrease in arterial pressure towards the target value, and then at 50-s intervals during the remaining period of hypotension and the final increase in pressure. All CFM measurements were made in mm above the zero µV baseline. This represents the logarithmic transformation of the voltage information and gives a Gaussian distribution of data for statistical processing. They were subsequently converted to equivalent µV values. Each measurement was normalized by expression as a percentage of the mean of the three consecutive 50-s interval values immediately preceding the induction of hypotension. Measurements of minimum CFM value proved to have no advantage over average values and are not referred to further.

A replay of tape recordings of raw EEG signals permitted subsequent visual and automated assessment of alterations in frequency and voltage. Assessment of frequency information was based on visual inspection of the raw EEG traces and of analogue displays of the outputs of high-order Chebyshev filters (Etherington, 1981), the modified CFM, the CFAM (Maynard and Jenkinson, 1984) and fast Fourier transform data (Matousek, Arvidsson and Friberg, 1978; Matousek and Petersén, 1983; Matoušek et al., 1985).

RESULTS

Mean arterial pressure decreased more slowly and reached the target value less often with TMP, whereas with SNP the decrease was rapid and sometimes overshot the target. The durations of hypotension and the mean decreases and the rates of decrease in MAP for patients receiving SNP and TMP, are presented in tables I and II. Expressed as percentages, the mean decreases in MAP were 37% and 51% for TMP and SNP, respectively.

Throughout the hypotensive period all patients appeared adequately perfused, with warm, pink extremities. Because of tachycardia or difficulty in achieving the target value of hypotension, 11 patients required β-blockade with propranolol: six of those receiving TMP and five of those receiving SNP.

In all 20 patients the average CFM voltage decreased during the initial reduction in arterial pressure. The decreases in mean voltage and the rates of decrease of the average CFM voltage for the two agents are given in table II. These represent mean decreases of 26% and 25% for patients receiving TMP and SNP, respectively. Thus, the decrease in CFM voltage was similar for the two agents in spite of a much greater decrease in MAP with SNP. No neurological or other sequelae were observed in any patient during the period immediately after operation.

There was a highly significant correlation between MAP and average CFM voltage during the initial reduction in arterial pressure in 10 out of the 20 patients regardless of agent, previous arterial pressure or age (tables I and II). When rates of decrease in arterial pressure were lower, there was a tendency to lower correlation coefficients between MAP and average CFM values.
to age or to arterial pressure before operation. When the mean values for patients receiving TMP (54 ± 7.5 mm Hg) and for those receiving SNP (61 ± 9.94 mm Hg) were compared, a trend emerged, but did not reach statistical significance.

As the amplitude of the CFM trace decreased (by a mean of 26%), the raw EEG, the Chebyshev filter outputs and the Fourier frequency analyses showed fairly consistent decreases in amplitude in all frequency bands. Comparing activity immediately before hypotension with that when the initial decrease in arterial pressure was complete, significant changes \( P < 0.05 \) on Student’s \( t \) tests were seen in several variables in all patients. The significant alterations from the Fourier analysis all concerned amplitudes (square roots of power). Expressed as mean percentages in order of severity, these were: amplitude in the upper alpha (10–12.5 Hz) band, 25% decrease; amplitude in delta (1.5–3.5 Hz) band, 23% decrease; amplitudes in lower alpha (8–9.5 Hz) and lower beta (13–17 Hz) bands, both 18% decrease; sum of all amplitudes (1.5–25 Hz), 17% decrease. In all patients there were significant decreases in the sum of all the Fourier amplitudes in the 1.5–25 Hz range and the five CFAM amplitude measures (maximum, 90th centile, mean, 10th centile and minimum amplitudes). The significant changes in the other Fourier variables listed above occurred in the majority of patients, but were not universal. There were no significant alterations in frequency distribution, expressed as percentage of power in each band, in any patient. Frequency ratios, that is, of slow:fast EEG activity \( [(\theta + \delta)/(\alpha + \beta)] \) showed significant alterations (either an increase or a decrease) in only a minority of patients.

Significant correlations were shown between MAP and EEG measurements taken at 10-s intervals during the acute decrease in arterial pressure. The CFM showed the highest correlation with MAP (table

### Table II. Profile of controlled hypotension in each patient, together with observed changes in CFM for both sodium nitroprusside (SNP) and trimetaphan (TMP) groups. Correlation coefficients between MAP and average CFM voltage are based on 10-s measurements during the decrease in MAP from values immediately before hypotension to the end of the initial rapid decrease induced by the hypotensive agent, and from 50-s measurements for the full period of administration of hypotensive agents. Significance of differences between mean values for SNP and TMP compared with Student’s \( t \) test: * \( P < 0.01 \), ** \( P < 0.001 \)

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\( P < 0.01 < 0.05 < 0.01 \) ns ≤ 0.05 ns ≤ 0.05 ns ≤ 0.05
but also significant ($P < 0.05$) were the correlations between MAP and delta, upper alpha and lower beta amplitudes (correlation coefficients each 0.36) and lower alpha and theta amplitudes (correlation coefficients 0.22 and 0.18, respectively). Bearing in mind the loss of information as a result of occurrence of slow-wave artefact in recordings from the operating theatre, the amplitudes in the range 10–17 Hz appear to give the most reliable correlations with decreasing arterial pressure.

DISCUSSION

We have shown that, in man, a decrease in MAP within the usual clinical range of controlled hypotension is reflected consistently in a decrease in cortical electrical activity (EEG). Furthermore, at mean arterial pressures between 55 and 40 mm Hg, the degree of depression is significantly greater with TMP than with SNP. These findings are in keeping with experimental evidence, particularly that of Ishikawa and McDowall (1981). These authors showed that, at low MAP, cerebral blood flow was better maintained with SNP than with TMP. They also demonstrated the close correlation between the EEG ("power" being measured by the CFM) and cerebral blood flow during induced hypotension in the cat. This validated the use of EEG as a non-invasive indicator of the adequacy of blood flow or oxygenation in these circumstances.

The EEG changes began at a mean arterial pressure threshold of 57 mm Hg, close to that at which autoregulation of cerebral blood flow normally fails (Lassen, 1959; Olleson, 1973). The high correlation between decrease in average CFM voltage and MAP, which occurred in half the patients, does not appear to be predictable in terms of expected risk factors. The efficiency of the mechanisms governing autoregulation depends on the method used to induce hypotension and on its rate of administration (Fitch et al., 1976), on the previous arterial pressure (Strandgaard et al., 1973; Strandgaard, 1976; Fitch et al., 1978), the age of the subject (Hoffman, Albrecht and Miletich, 1982) and the rate of decrease in arterial pressure (Patel, 1981). In our relatively small series of patients, we were unable to show any effects of either age or preoperative MAP. Both younger and older individuals appeared equally likely to show a strong correlation between decreasing arterial pressure and declining cortical electrical activity, whether the rate of decrease was in the higher range produced by SNP or the lower range produced by TMP. It should be borne in mind that we had excluded patients who were older than 70 yr or had previous hypertension or evidence of cerebrovascular disease. However, the implication must be that all patients, regardless of age, previous arterial pressure etc., are at risk from increasing cerebral oligaemia when their MAP is reduced below the value at which a decrease in EEG voltage begins.

Cerebral oligaemia, whatever its cause, leads to brain damage of a characteristic arterial boundary zone distribution (Graham, 1977; Brierley et al., 1980). This pattern of cerebral infarction occurs in the Rhesus monkey when oligaemia during profound hypotension has resulted in at least 15 min EEG silence (Brierley et al., 1969). Shorter periods of electrical silence were shown to be completely reversible without cerebral damage when adequate circulation was restored. This is the basis of the so-called dual threshold for cerebral ischaemia (Astrup et al., 1977; Morawetz et al., 1979), whereby the ini-
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ischaemic brain damage of a similar arterial bound-
leagues (1969). Indeed, evidence from studies of
experimental animals studied by Brierley and col-
likely to take less time than in the young healthy
oligaemia before ischaemic damage would occur. It
is not yet known how long it takes to produce cereb-
ral infarction in man when induced hypotension has
resulted in a reduction in cerebral blood flow suffi-
cient to produce electrical silence. However, it is
likely to take less time than in the young healthy
experimental animals studied by Brierley and col-
leagues (1969). Indeed, evidence from studies of
ischaemic brain damage of a similar arterial boundary
zone distribution, following cardiac surgery,
suggests that a period of about 7 min major depres-
ion of EEG must occur before infarction ensues

The MAP threshold at which a pressure-dependent effect begins to occur is of interest. This is at a
slightly higher pressure with SNP and probably
merely relates to the moment of onset of maximal cerebral arteriolar vasodilatation, which leads to
inactivation of the autoregulatory capacity. How-
ever, as McDowall (1982) has pointed out, it is incorrect to speak of autoregulation of cerebral blood flow under the particular conditions of hypotension induced by SNP. This cerebral vasodilatation presumably occurs concurrently with dilatation of the systemic vascular bed and the consequent
reduction in arterial pressure. Despite this, cerebral perfusion may actually increase as a result of an increased cardiac output with SNP (Wildsmith et al.,
1973; Pickard et al., 1981). This contrasts sharply
with the case of TMP where the cardiac output is
decreased (Jordan et al., 1971) and, given that autoregulation is in abeyance at these low pressures,
this means reduced cerebral blood flow. There is,
indeed, evidence from our work in man that there is
better maintenance of cerebral electrical activity with SNP than TMP at similar values of hypoten-
sion. However, it was possible to detect the onset of
an alteration in the relationship between average
CFM value and MAP, even though it was rarely as
dramatic as in the patient illustrated in figure 2. The
evidence arising from the frequent need to use β-
blockade to control tachycardia suggests continuing sympathetic activity in patients receiving both
agents. It is surprising that a ganglion blocking drug such as TMP did not obliterate this phenomenon.

Differences in the degree of correlation between
MAP and average CFM voltage were apparent when
the 10-s measurements during the initial decrease in
arterial pressure were compared with the 50-s measure-
ments during the whole period of hypotension in individual patients. In all patients surgical stimu-
lation tended to increase after the initial reduction in
arterial pressure had been achieved. This, given the
generally light level of anaesthesia, tended to lead to
more variable traces and obscure the simple relation-
ship between arterial pressure and EEG. Further-
more, in patients receiving sodium nitroprusside the
initial decrease was achieved over a relatively short
period, limiting the number of measurements obtainable and, thus, the possibility of achieving sig-
ificant correlation; these patients also tended to
have longer periods of hypotension (90 min on aver-
age, compared with 70 min for patients receiving
trimetaphan) with a greater possibility of other fac-
tors affecting the EEG as a consequence. This may
explain our inability to show a significant difference between the two drugs when measurements during
the whole period of hypotension were included.

The automatic frequency analysis supported the
evidence from the CFM traces. There was a consist-
tent decrease in the summed amplitudes from each
of the traditional EEG frequency bands and most significantly in the delta, upper alpha and lower beta
ranges as MAP decreased towards target values.
This contrasted with the infrequent and inconsistent
alterations in the percentage of total activity falling
in each frequency band which rendered indices such
as slow:fast frequency ratios and mean frequency
ineffective. The relatively light level of anaesthesia
in our patients contrasts with that in many of the
earlier experimental studies where decreases in
faster and increases in slower EEG frequencies were
reported as evidence of impending cerebral
ischaemia (Sugar and Gerard, 1938; Gibbs,
Williams and Gibbs, 1940). Thus, frequency
analysis of the EEG provided inconsistent changes
during hypotension and offered no advantage over
the CFM recording.

Patients are monitored during induced hypoten-
sion to reduce unnecessary risks. Clearly, guarding
against unexpected or exceptional decreases in MAP
by intra-arterial pressure monitoring has proved of
major importance in this respect. However, it is
quite unpredictable in which patients the cerebral
circulation is unable to tolerate reductions in MAP
to less than about 60 mm Hg. This implies that
direct cerebral monitoring is mandatory, should it
become necessary to decrease arterial pressure
below this value. The methods need to be simple
but effective. This study has demonstrated that a
suitable measure is total EEG power or the amplitude envelope of filtered EEG as provided by the CFM.

The evidence from the present study also raises the question as to whether decreases in arterial pressure to less than a mean of 60 mm Hg are ever justified during routine surgery. The question is reinforced by reports that a decrease in blood loss is achieved mainly by quite modest decrease of arterial pressure and that greater hypotension does not produce greater benefit (Donald, 1982). Our evidence also supports that of Stoyka and Schutz (1975), Michenfelder and Theye (1977), Maekawa, McDowall and Okuda (1979) and Ishikawa and McDowall (1981) that, if even greater hypotension is required, sodium nitroprusside is the agent of choice to ensure the safety of the brain.

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REFERENCES


