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TABLE 5 Annual Incidence Rate per 100,000 Population in Published Series

<table>
<thead>
<tr>
<th>Authors and period of survey</th>
<th>Age (yr)</th>
<th>Number of cases</th>
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</thead>
<tbody>
<tr>
<td>Matsumoto et al. 1955-1969</td>
<td>0-34</td>
<td>4</td>
</tr>
<tr>
<td>Present study 1958-1968</td>
<td>0-35</td>
<td>7.5</td>
</tr>
<tr>
<td>Matsumoto et al. 1955-1969</td>
<td>0-36</td>
<td>7.9</td>
</tr>
<tr>
<td>Aho 1972-1973</td>
<td>0-37</td>
<td>7.7</td>
</tr>
<tr>
<td>Brewis et al. 1955-1961</td>
<td>0-38</td>
<td>8.9</td>
</tr>
<tr>
<td>Melamed et al. 1960-1967</td>
<td>0-39</td>
<td>2.5</td>
</tr>
</tbody>
</table>

References
2. Statistical Bureau of Iceland (Hagstofa Islands) 1975
8. Aho K: Personal communication, 1975

TABLE 6 Etiology of Stroke in Young People in Published Series

<table>
<thead>
<tr>
<th>Authors</th>
<th>Age groups (yr)</th>
<th>Unknown Etiology %</th>
<th>ICH%</th>
<th>Thromboembolic</th>
<th>SAH%</th>
<th>CH+ SAH %</th>
<th>CH %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Matsumoto et al. Rochester U.S.A.</td>
<td>0-34</td>
<td>5.9</td>
<td>5.9</td>
<td>47.0</td>
<td>41.2</td>
<td>47.1</td>
<td></td>
</tr>
<tr>
<td>Present study Iceland</td>
<td>0-35</td>
<td>15.2</td>
<td>21.9</td>
<td>3.8</td>
<td>41.9</td>
<td>63.8</td>
<td>17.1</td>
</tr>
<tr>
<td>Aho, Finland</td>
<td>0-35</td>
<td>0.0</td>
<td>15.4</td>
<td>30.8</td>
<td>53.8</td>
<td>69.2</td>
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<tr>
<td>Melamed et al. Jerusalem</td>
<td>0-34</td>
<td>13.8</td>
<td></td>
<td></td>
<td></td>
<td>48.3</td>
<td></td>
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Monitoring of Cerebral Perfusion During Anesthesia by Time-Compressed Fourier Analysis of the Electroencephalogram

ROBERT R. MYERS, PH.D., JAMES J. STOCKARD, M.D., PH.D., AND LAWRENCE J. SAIDMAN, M.D.

SUMMARY Time-compressed Fourier analysis of the electroencephalogram has proven to be a useful analytical procedure during anesthesia and surgery which simplifies data interpretation by presenting the EEG in a time-compressed frequency domain rather than the conventional time domain. This method of data analysis graphically accentuates the electroencephalographic correlates of ischemia-induced cerebral dysfunction and other cerebral oxygen consumption abnormalities. The ability to accentuate trends in frequency and power is derived from sequential plotting of spectra to produce a graph with three dimensional axes of frequency, time, and power.

In carotid endarterectomies the system has proven more useful than the conventional EEG in assessing the need for a vascular shunt to maintain internal carotid flow during endarterectomy. In open-heart surgery time-compressed EEG spectral analysis has allowed early recognition of cerebral ischemia resulting from arterial hypotension and venous hypertension. Five cases are presented which demonstrate the ability of our system to reflect developing cerebral ischemia.

ALTHOUGH THE POTENTIAL of electroencephalography in assessing the functional state of the cerebral is well known, EEG monitoring during anesthesia has not gained wide acceptance. The complexity and nonspecific nature of EEG information, the difficulty in assessing trends in EEG activity as it is displayed conventionally, and the high cost and space requirement of recording equipment are the chief reasons for the lack of acceptance. The difficulty in interpreting and quantifying changes in activity probably represents the most serious shortcoming of conventional EEG monitoring for anesthesiologists. A major consideration is that the anesthesiologist has limited time to study the primary EEG record as it is being recorded. Even when there is time to peruse the conventional voltage vs. time tracing,
the information being presented oscillographically or on standard EEG chart paper cannot easily be compared with preceding activity; important trends such as developing cerebral hypoxia/ischemia or changing anesthetic state may not be appreciated. Visual inspection of the conventional EEG record has other drawbacks. Secondary EEG frequencies can be obscured by activity at the primary frequency, and conversely, changes in the fundamental frequency may not be observable because of superimposed activity at other frequencies. High-frequency muscle artifact further complicates interpretation of the conventional EEG.

Fourier spectral processing of the electroencephalogram alleviates many of these problems by presenting data in the frequency domain rather than the conventional time domain. This mathematical transformation extracts the most clinically important information from the EEG, namely, frequency and amplitude, and displays them in an easily interpretable form. Dietzch was the first to apply Fourier analysis to the EEG,1 while Gibbs et al. in 1937 recommended EEG recording during anesthesia.a However, it was not until Bickford et al. applied time-compression techniques to the EEG spectra presentation that the clinical importance of computerized EEG monitoring was fully realized.

We have utilized these spectral compression techniques in the design of an intraoperative EEG recording system termed VITAC (Vital Indices Transmission and Analysis by Computer). In this report we wish to present the results of computerized intraoperative EEG monitoring in five patients at increased risk of intraoperative cerebral ischemia; these five cases were selected from over 100 similar operations monitored by VITAC to demonstrate the ability of the system to reflect developing cerebral ischemia. Data were collected both from a prototype system as well as from a second-generation system used exclusively for anesthesia patient monitoring. Technical aspects of the prototype system, which telephonically linked a laboratory computer to the operating room, have been described elsewhere.a

Central to the success of intraoperative spectral plotting of the EEG is the final form of the spectra. We chose the time-compressed spectra technique6 of presentation because of its ability to accentuate trends in frequency and power. This method of presentation adds a third dimension of time to the two-dimensional spectral axes of power and frequency. To obtain this third dimension, successive spectra (derived from 4-second epochs of the primary EEG) are plotted slightly above the preceding spectrum. A computer subroutine suppresses the printing of any spectral component which would otherwise trace over previously plotted spectral peaks. Figure 1 summarizes the computing process. A compilation of spectra plotted with this technique appears three-dimensional with a horizontal axis of frequency, a vertical axis of time (each spectrum representing 4 seconds), and an imaginary third dimensional axis of power manifested in the height of the spectral power peaks.

Results

The delineation of focal cerebral pathology demands detailed multichannel EEG analysis and is best performed by trained electroencephalographers, whereas the functional state of the brain during anesthesia and surgery may be assessed generally by the anesthesiologist through analysis of one or two electroencephalograph traces. The information most important to the anesthesiologist is not the initial state of the EEG, which may be influenced by existing pathology, but its change during anesthesia. These EEG changes most commonly are due to variations in the anesthetic state or uncompensated changes in cerebral perfusion pressure.

Figure 2 directly compares the primary EEG in the time domain to the corresponding spectra of the frequency domain during a drop in arterial pressure below the

Methods

Two or more channels of primary EEG data were collected from Sn-SnCl electrodes applied with collodion and gauze to the occipital, frontal, and central areas of the scalp as specified by the International 10-20 System.6 These signals were amplified and bandpass-limited to frequencies between 0.5 and 32 Hz. A Digital Equipment Corporation PDP-12 or PDP-11 computer was used for Fourier analysis of these signals. The second-generation system, which is used exclusively now, employs the more powerful and versatile PDP-11 computing system. Primary data were converted from analog to digital form by sampling 64 times per second (i.e., at the Nyquist frequency) and digitally encoding the amplitude at one of the possible 1024 digital amplitude levels. Fast-Fourier transformations were performed on successive 4-second epochs of digitized data. Analysis and spectral plotting of the previously digitized epoch of data was interleaved with digitization of the subsequent epoch so that all data could be analyzed on-line. The spectral output was plotted in the operating room and therefore was immediately available to the anesthesiologist.

Figure 1. Time-compressed Fourier analysis of the EEG. Primary data are Fourier-processed in 4-second epochs to produce power spectra of 0.25 Hz resolution from 0.25 to 16 Hz (or 0.5 Hz resolution from 0.5 to 32 Hz). Time-compression is accomplished by vertically stacking subsequent spectra. The resultant accumulation of spectra appear three-dimensional with a horizontal axis of frequency, a vertical axis of time (each spectrum representing 4 seconds), and an imaginary third dimensional axis of power manifested in the height of the spectral power peaks. Normal EEG alpha activity recorded from the occipital region of the scalp is displayed.

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autoregulatory range of cerebral perfusion during cardiac surgery. This hypotensive episode resulted from abrupt compromise of venous return to the pump-oxygenator by manipulation of the heart and kinking of right atrial cannula (for venous return). Thirty-six seconds of EEG data were analyzed in successive 4-second epochs beginning at the time when arterial pressure fell below 50 mm Hg, continuing through a minimum pressure of 25 mm Hg, and terminating when the pressure was 35 mm Hg and rising slowly. The electroencephalographic manifestations of this insult are characteristic of those associated with developing cerebral ischemia: The dominant cerebral electrical rhythm (usually in the alpha or theta band of frequency*1) decreases in frequency and is replaced by large amplitude slow waves (less than 4 Hz); this change is followed by reduction in EEG amplitude culminating in EEG isoelectricity. These monotonic frequency and biphasic amplitude changes are appreciated most readily if the EEG is viewed in the frequency domain over a compressed time interval since the amplitude changes may occur too slowly to be appreciated at the recording speeds used for conventional EEG. The characteristic ischemic EEG trend shown in figure 2 can be completely reversed if adequate perfusion is re-established while the cerebral microvasculature is still functional (i.e., prior to establishment of a no-reflow condition).*2

In carotid endarterectomies the VITAC system has proven more useful than conventional EEG in assessing the need for a vascular shunt to maintain internal carotid flow during endarterectomy since trends in amplitude and frequency are accentuated and easily compared with preceding activity. In three carotid endarterectomies test clamping of the internal carotid artery produced ipsilateral slowing and diminution of spectral components, and a Javid shunt was then placed which reversed the ischemic EEG changes. An on-line, two-channel intraoperative recording of EEG spectra is reproduced in figure 3 for one of these cases. The dominant cerebral rhythm preceding carotid clamping was 8 Hz and symmetrical between hemispheres. When the clamp was applied to the left common carotid artery after two and one-half minutes of baseline recording, the EEG remained symmetrical for an additional minute before a power reduction in the left channel was observed. Symmetry was then reduced quickly as the EEG ipsilateral to the operative site progressively lost alpha, theta, and delta activity. Based on these data, a Javid shunt was placed surgically between the left common and internal carotid arteries to maintain blood flow around the endarterectomy site at the carotid bifurcation. This procedure improved blood flow to the left hemisphere which subsequently reversed EEG changes in the ipsilateral hemisphere (recovery is not seen in fig. 3).

In another case with similar EEG changes after left carotid clamping, the decision was made not to use a vascular shunt because of the relatively high stump-pressure (55 mm Hg) transmitted through the circle of Willis by the noninvolved vertebral and right carotid arteries. The patient was a right-handed, 50-year-old male with a history of transient ischemic symptomatology in the left carotid system but no persistent neurological deficit. Figure 4 displays the left frontocentral EEG spectra recorded during carotid clamping. The infusion of 500 mg calcium chloride immediately prior to carotid clamping resulted in an increase in mean arterial pressure from 90 to 135 mm Hg. A 7-Hz peak dominated the EEG spectra during the initial period of the record but then rapidly broadened and diminished in amplitude as the left carotid was clamped. The EEG deteriorated progressively as activity in the alpha band of

*EEG frequency bands: delta 0.5-4 Hz, theta 4-8, alpha 8-13, beta > 13.
†An initial increase in EEG frequency is usually seen with hypoxia, which is mediated by stimulation of the aortic and carotid chemoreceptors and secondary reticular activation. However, cerebral ischemia during cardiopulmonary bypass is not associated with any lowering of PaO2 at these more proximal chemoreceptors, and thus the initial fast EEG phase is seldom seen.
Intraoperative EEG recording during test clamping of the left carotid artery prior to endarterectomy. The 8-Hz activity in the left fronto-occipital channel (F3-O1) continued to be symmetrical with the contralateral channel for one minute after clamping (at the arrow), at which time EEG activity ipsilateral to the operative site progressively deteriorated. This indication of developing unilateral cerebral ischemia resulted in placement of a vascular shunt around the endarterectomy site to maintain internal carotid flow during the operation.

Left hemisphere (F3-O1) EEG spectra and mean arterial pressure (MAP) recorded on-line during left carotid endarterectomy. The development of large amplitude, slow-waves from a baseline of low-voltage alpha activity was apparent spectrally within one minute after carotid clamping. Internal carotid artery back-pressure (stump-pressure) was 55 mm Hg. Despite the infusion of phenylephrine to increase arterial pressure, EEG improvement was not seen; only low-voltage theta and delta activity persisted.

Right hemisphere centro-occipital (C4-O2) EEG spectra during an arterial hypotensive episode in cardiac surgery for mitral valve replacement. The mean arterial pressure fell from over 100 to 35 mm Hg following transfer to complete cardiopulmonary bypass. The EEG concomitantly deteriorated as rhythmic 6-Hz baseline theta activity diminished in amplitude and was replaced by low-voltage theta with occasional bursts of delta activity.
occipital EEG, the constant and well-defined spectral peak at 6 Hz disappeared one minute into bypass as the mean arterial pressure dropped to 55 mm Hg. Other theta activity was similarly diminished as the pressure continued to fall. Throughout the remainder of the surgery, the EEG consisted of random low-voltage theta activity with occasional superposition of large amplitude delta activity. Postoperatively, the patient developed right-sided periodic lateralized epileptiform discharges (PLED’s) in the EEG and left-sided hemiplegia, both events compatible with ischemic damage of the right hemisphere.

In another case cerebral perfusion was compromised during partial cardiopulmonary bypass by venous hypertension secondary to partial occlusion of the caval catheters (fig. 6). Prior to occlusion the EEG consisted of low-voltage theta activity which then increased in amplitude as jugular pressure rose from 17 mm Hg to a peak of 32 mm Hg. At that time large amplitude, monorhythmic delta activity dominated the EEG. Delta activity persisted until venous pressure fell back below 20 mm Hg, at which time alpha activity appeared. The appearance of alpha rather than theta activity was due to a concomitant rise in arterial pressure which further increased cerebral perfusion pressure.

**Discussion**

The data presented above illustrate the correlation between cerebral function (as reflected in the EEG) and cerebral perfusion pressure (as reflected by the difference of systemic arterial and jugular venous pressure). These data were collected during the development of the VITAC system, and consequently some analyses were performed offline and correlated retrospectively. We have emphasized here (1) the narrow margin which exists between adequate cerebral perfusion and cerebral ischemia during anesthesia in patients with frank or subclinical cerebrovascular disease and (2) the benefit of time-compressed Fourier analysis of the EEG in qualitatively assessing cerebral function and thus assuring adequate cerebral perfusion.

Alternative EEG processing methods such as voltage-integration have been applied intraoperatively to monitor cerebral function. Figure 7 directly compares EEG spectral analysis with voltage-integration during a transitory hypotensive episode associated with transfer to extracorporeal flow in cardiac surgery. A constant 8-Hz alpha rhythm dominates the spectra and produces a steady integrated amplitude of 45 μv prior to the hypotensive episode when the EEG degenerates to very low-voltage, random theta activity with occasional bursts of delta activity. Although the transition in amplitude is clearly discernible in both graphs, no additional information is provided by separate voltage-integration. Important frequency transitions and the nature of delta burst activity are lost if integration is used exclusively. Integrated activity is, in fact, present in the spectra since, according to Rayleigh's theorem, "The integral of the squared modulus of a function is equal to the integral of the squared modulus of its spectrum." That is, the area contained under the EEG power spectral peaks is equal to the integral of the square of the absolute value of the original EEG. Therefore, more detailed integration information is directly available from the time-compressed spectra since a separate integration is effectively performed every four seconds.

Other mathematical transformations such as auto- and
cross-correlation, zero-cross, peak detection, and threshold linear coherence have heuristic value but do not provide the anesthesiologist with enough additional information to justify their routine intraoperative application. Since ischemia-induced alterations of the evoked response are not apparent until nearly all spontaneous cortical activity has been lost, the evoked response does not provide useful data on the early development of cerebral ischemia, which concerns the anesthesiologist. However, the evoked response is more valuable than the EEG in assessing cerebral viability and establishing prognosis when the EEG is flat or amorphously irregular.

Two major problems have plagued our early clinical experience with VITAC, both of which are inherent to the time-compressed spectral display. The first is the loss of information about the EEG waveform, in particular, spike activity. Although this does not represent a significant drawback for the anesthesiologist, who is primarily concerned with frequency changes, epileptiform EEG activity during anesthesia can be of clinical importance since under rare circumstances it may represent a manifestation of cerebral anoxia. Many anesthetics have now been shown to be epileptogenic if given in sufficient dose. Enflurane produces EEG spiking in some subjects at 1 minimum alveolar concentration (MAC). The incorporation of spike information into the VITAC system is being accomplished by addition of a computerized spike recognition program. Spikes are recognized when a given EEG voltage change simultaneously meets predetermined slope and amplitude criteria. Such programs have proven highly accurate when tested on data of known spike content.

The second problem has been the elimination of movement artifact and electronic noise generated within the operating room by surgical accessories such as the electrocoagulating device. These disturbances produce large amplitude signals superimposed on the EEG in the delta and theta range of frequencies which, when presented in the frequency domain, appear as high-power, slow-wave, spectral peaks. The amplitude and breadth of these peaks may be so great that subsequent spectra are obscured from view. The problem is minimized significantly by careful electrode application and recording technique. These artifacts can be minimized further by setting a predetermined limit to the height of a spectral peak in the computer or by eliminating (by visual or automatic pattern recognition) these short sections of data from spectral processing.

Unique spectral responses have been associated recently with many of the commonly employed anesthetics. The precise quantification of frequency afforded by the Fourier transform makes the time-compressed Fourier processing of the EEG a more sensitive index of the level of anesthetic depth than conventional EEG. Although the EEG by itself is not sufficient to predict the level of anesthesia under clinical conditions where adjuvant medications are employed, variations in EEG frequency may be directly related to changes in the depth of anesthesia. In a curarized patient receiving morphine and nitrous oxide anesthesia, we have seen an EEG arousal response clearly associated with median sternotomy. This observation suggests that in the absence of clinical manifestations, EEG spectral responses shifting from steady patterns to high-frequency, low-voltage beta activity may be an important indication of sub-MAC levels of anesthesia. For this reason we have included in the computer programming a histogram which plots the average, maximum, and minimum heart rate for every spectrum since heart rate variations are also a valuable indicator of arousal.

Direct comparisons between EEG, regional cerebral blood flow (rCBF), and internal carotid artery pressure have been made during carotid endarterectomy. Trojaborg and Boysen report EEG flattening with test occlusion of the carotid artery when regional cerebral blood flow fell to 11 to 19 ml/100 gm/min and when internal carotid artery pressure (stump-pressure) fell to 15 to 46 mm Hg. EEG slowing was seen when rCBF fell to 16 to 22 ml/100 gm/min and internal carotid artery pressure fell to 29 to 50 mm Hg. The Mayo group routinely record EEG in the primary form during carotid endarterectomy since establishing a high correlation between rCBF and EEG. Boysen correlated postoperative aggravation of neurological deficits in patients where rCBF was below 30 ml/100 gm/min during carotid clamping and suggested this level of perfusion as a critical lower limit. She found no significant correlation between distal internal carotid artery pressure and rCBF during clamping (N = 8). In a study of 297 operations for carotid endarterectomy, Hays et al. correlated postoperative neurological deficit with intraoperative ischemia in 50% of the patients in whom a Javid shunt was not used when stump-pressure was less than 50 mm Hg. Although we use a stump-pressure of 50 mm Hg as a guide for placement of a vascular shunt, we also rely on the indication of cerebral function provided by the VITAC system to guarantee adequate perfusion in these borderline cases.

In summary, we have developed a reliable system which provides on-line spectral analysis of the electroencephalogram in the operating room. The major advantages of this system are that it is easily interpreted by those not trained in electroencephalography and that it accentuates trends in EEG frequency and power which may be difficult to extract visually from the primary record. The recognition of these trends can alert the anesthesiologist to developing cerebral dysfunction when it first occurs and is usually still reversible.

Acknowledgment

We are grateful to Reginald G. Bickford, M.B., whose computing facilities were used to analyze some of the EEG data and to Richard R. Uhl, M.D., for his cooperation in this study.

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An Ultrastructural Assessment of an Embolic Method of Producing Cerebral Ischemia

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AND VINOD D. DESHMUKH, M.D., M.S., PH.D.

SUMMARY This study presents ultrastructural confirmation of the embolic method of producing an ischemic lesion. The embolic method was shown to produce more advanced parenchymal changes than those reported for the same postocclusion period following

TWO OF THE MOST reproducible animal models used to study cerebrovascular-related changes involve the surgical occlusion of cerebral vessels with surgical clips or introduction of an embolic material into the cerebrovascular system.

Ultrastructural characterizations of parenchymal response following ischemic insults induced by the clip model have been reported, as well as those alterations that occur within the arterial wall and accompanying nerve bundles. However, to our knowledge no ultrastructural evaluation of parenchymal response following embolization with plastic materials has been carried out, nor has determination been made of arterial response in the embolic area. In this com-

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vascular clipping.

With this method there was less alteration in the morphological integrity of occluded extraparenchymatous areas of the arterial bed, especially in the associated nerve bundles.

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communication ultrastructural observations following introduction of embolic insult will be discussed and compared with the data from the clip model.

**Methods**

Six baboons (Papio anubis) weighing 5 to 8 kg were lightly anesthetized with an injection of sodium pentobarbital (25 mg/kg body weight). Following tracheostomy the animals were immobilized with pancuronium bromide (0.1 mg/kg body weight), and anesthesia was maintained with N2O inhalation by means of a Harvard variable speed respirator. Pancuronium bromide was further supplemented as required to maintain immobilization. End-tidal CO2 was recorded continuously during the study by means of a Beckman infra-
red gas analyzer. Catheters were inserted through the femoral artery into the descending aorta in order to monitor systemic blood pressure and also into the femoral veins to permit intravenous infusion and to allow return of blood from an extracorporeal circulation system which included a Guyton analyzer for assessment of cerebral arteriovenous (A-V) oxygen differences and oxygen and hydrogen elec-
trodes mounted in flow-through cuvettes.
Monitoring of cerebral perfusion during anesthesia by time-compressed Fourier analysis of the electroencephalogram.
R R Myers, J J Stockard and L J Saidman

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