

Carotid and Vertebral Artery Surgery, EEG Monitoring, and the Operating Room

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The electroencephalogram (EEG) reflects the perfusion of the supratentorial brain (3-5,9, 11,24,26,31). Research relating brain blood flow to EEG changes was pioneered in 1913 by Gibbs et al. (10) and Lennox et al. (14,15). The significance of their observations went unappreciated until the advent of cardiac surgery and cardiopulmonary bypass. Safeguarding the brain during elective cardiac arrest was the sine qua non for continuation of cardiac surgery.

Monitoring by EEG during normothermic cardiopulmonary bypass procedures can give continuous and dynamic information about brain blood flow and cerebral status from minute to minute (20,25,29). Though undisputed in its merit, the application met with limited acceptance (12,17,31), perhaps in part due to the continuous improvement in the technology of cardiopulmonary bypass, as well as the recognition of thrombogenic factors and of air embolism, and in part due to the very real need to simplify complex operating room procedure. Interest was again revived with the introduction of aortic counterpulsation as an adjunct to cardiopulmonary bypass, which provides nonpulsatile blood flow. The EEG was used as a measure of cerebral perfusion during pulsatile and nonpulsatile bypass support (23). Hypothermia

with cardiopulmonary bypass and deep hypothermia with circulatory arrest so depress brain metabolism and function that the EEG becomes flat and can no longer be used to predict the survival of brain function following these procedures.

With the increasing frequency of carotid surgery, intraoperative EEG monitoring became clinically valuable. Here the EEG reflects the adequacy of brain perfusion and of the collateral circulation that occurs with clamping of the carotid artery (4,11,18,24,30). With this information, the safety of carotid clamping during the time required for reconstruction is determined. In order to simplify the operating procedure and avoid continuous EEG monitoring, two approaches have been employed; first, the arterial pressure from the distal carotid, the "stump" pressure, serves as a guide to the selective use of intraluminal shunts, and second, the intraluminal shunt is used in all cases. However, the basic premises of these approaches are false. The use of the stump pressure presumes that a single determination could adequately substitute for continuous EEG monitoring, in other words, that the only hazard to the patient occurs during preliminary carotid clamping. The routine use of the intraluminal shunts pre-

sumes that adequate blood flow is guaranteed, that the shunts remain patent, that the shunts do not produce thrombosis or intimal dissection, and that shunt monitoring is not necessary.

On the other hand, direct vertebral artery surgery is taking on greater prominence, and neither have criteria for brain protection been proposed nor can the use of an intraluminal shunt be considered feasible. So the solution must lie in a better understanding of brain perfusion to provide safeguards. Many dynamic changes in brain blood flow that occur during cerebral vascular surgery are related to physiologic stress and are not related to the initial clamping of the carotid artery. Continuous EEG monitoring provides safe conduct through a host of hazards.

METHODS AND MATERIALS

The EEG and ECG were continually recorded prior to the induction of anesthesia, during the entire operative procedure, and, if the patient was difficult to manage, in the intensive care unit, throughout the night. Recordings were made on a Grass Model 8, 10-channel EEG machine, with one channel of ECG being constantly recorded. The referential montage was employed with gain manipulation where indicated. The interpretation of the EEG was provided by one of the authors (Anderson), or a highly experienced EEG technologist with 20 years of experience.

Observations were made during 225 surgical procedures for carotid or vertebral artery reconstruction from January 1976 to June 1980. Preoperatively, all patients underwent cerebral hemodynamic evaluation (CHE) and were brought to optimum hemodynamic status. All procedures were conducted with the patient's head in neutral position, fixed upon a neurosurgical headrest. The Trendelenburg position was frequently employed to reduce or eliminate EEG abnormalities encountered. The depth of anesthesia was light and was controlled to provide optimal EEG activity. Drugs that suppress EEG activity were avoided.

Seventy-seven instances of significant EEG

change (34%) were observed and classified. These changes could be classified as caused by anesthesia and ventilation, cardiogenic stress, intracranial venous hypertension, neurogenic stress, test clamping, high-risk patients, and surgically aggravated intracranial steals.

ANESTHESIA AND VENTILATION

Careful positioning of the patient's head in neutral position is done to prevent compromise of blood flow through any of the unoperated cervical vessels (Chapter 27). Extremes of byperextension and head rotation (11) with consequent EEG changes were avoided in all patients. Careful control of the depth of anesthesia is necessary to assure a technically accurate EEG (26). Respirator malfunction sufficient to produce ischemic changes in the EEG was undetected by the anesthesiologist in three instances (1.3%) until brought to the attention of the anesthesiologist by the EEG technologist. Surgery on a fourth patient was cancelled because diffuse slow wave activity occurred after the induction of anesthesia and could not be rectified. Later, valve malfunction of the anesthesia machine was found and corrected.

Faulty ventilation produced a gradual slowing of the EEG and a loss of voltage at a time when there was no surgical compromise of blood flow or other factor to account for it. In a patient with precarious brain perfusion, the malfunction of an anesthetic coupling or respirator could be disastrous. More important, the review of the anesthesia record does not permit an accurate retrospective analysis of intraoperative events, an analysis that is simply performed with continuous physiologic recording.

CARDIOGENIC STRESS

Historically, the ECG trace was added to the EEG to improve artifact detection, but the use of this lead in surgery has extended the scope of the EEG to serve as a mini-Holter, providing the data for cardiac rhythm analysis. Generally, the lead does not produce the graphic representation characteristic of the standard ECG, but

evaluation of heart rate, atrial and ventricular contractions, and conduction patterns are possible with EEG/ECG. Bradycardia or dysrhythmia are not unusual during the induction of anesthesia or intubation. At times, much concern is aroused by these incidents.

CASE 1

Sinus arrest and nodal rhythm with intubation

A 54-year-old man had a normal EEG during induction of anesthesia. With intubation, a nodal rhythm appeared after 4.6 sec of sinus arrest. The nodal rhythm continued for 30 sec. The episode was terminated with atropine.

Comment: The onset of ECG changes during the course of carotid or vertebral artery surgery are most often first detected by the EEG technician. Bradycardia was encountered during manipulation of the carotid sinus, the superior cervical ganglion, the stellate ganglion, and the internal jugular vein. Tachycardia with ectopic

beats was produced by jugular vein compression on one occasion. Cardiac dysrhythmia may induce profound changes in cortical activity (14) and the EEG.

CASE 2, H. T.

Right ventricular pacing, shock, and slowing of the EEG

A 68-year-old man presented with syncope and extreme sinus bradycardia (22 bpm), with a resting heart rate of 52 to 55 bpm. His heart was markedly enlarged. A right ventricular (RV) electrode was positioned epicardially and thresholds were determined. During the time when all personnel were concerned with pacemaker and electrode evaluation, marked EEG changes of extreme slowing and attenuation (Fig. 1) were noted by the EEG technician before the systemic hypotension was observed or recorded by the anesthesiologist. Each time right ventricular pacing was attempted, these severe EEG changes would immediately reappear.

Comment: During threshold determination, the focus of attention is on the ECG monitor.

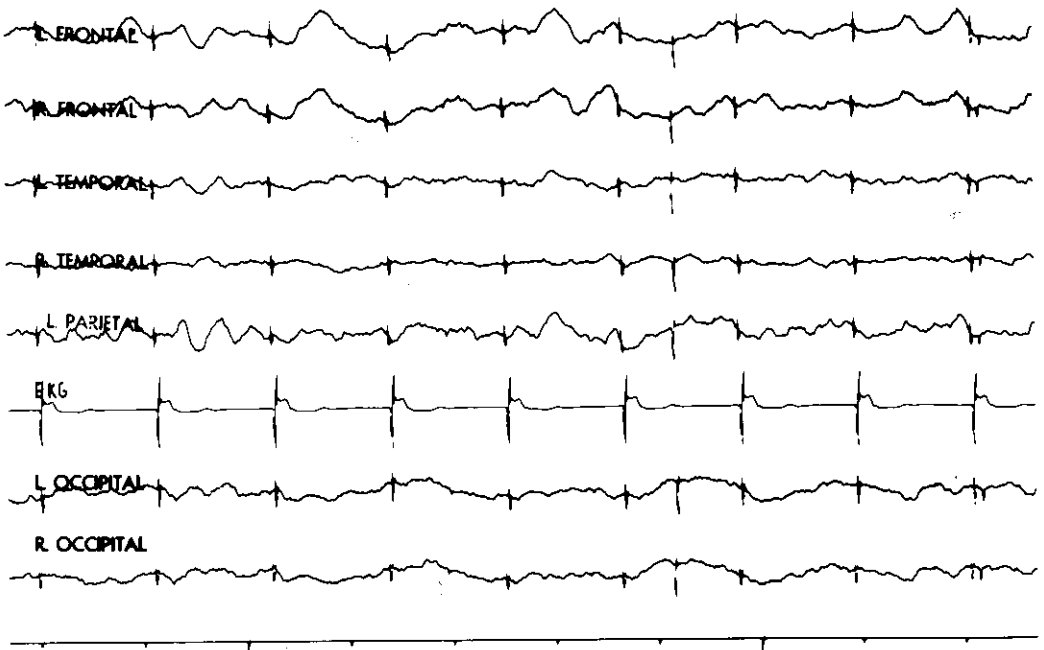


FIG. 1. Pacemaker implantation, shock, and EEG slowing. Marked slowing and attenuation of EEG developed during electrode threshold determination. All eyes watched the oscilloscope. The EEG technician alerted the anesthesiologist that the patient was dying of these catastrophic changes. The finding was reproducible. Case 2, H.T.

No one may be monitoring the patient's physiologic response to pacing. In this patient, RV pacing would have been fatal if it had gone unrecognized for the few minutes of testing. The patient was subsequently paced physiologically with an atria! synchronous pacemaker (1, Chapter 1) (Fig. 2) which was tolerated.

INTRACRANIAL VENOUS HYPERTENSION

During carotid or vertebral artery surgery, it is sometimes necessary to retract, clamp, or ligate the jugular vein. Such manipulation of the jugular vein may produce changes in the EEG by two mechanisms: intracranial hypertension and reflex bradycardia with hypoperfusion.

CASE 3, C.W. EEG changes with jugular vein ligation

Reconstruction of the proximal left vertebral artery (LVA) in a 60-year-old man necessitated division of the jugular vein. This jugular vein was the dominant

venous drainage of the brain. Thirty seconds after ligation of the vein, moderate global slowing of frequencies and 30% attenuation appeared (Fig. 3). The heart rate and blood pressure were unchanged.

Comment: These changes indicated that cortical (slowing of frequencies) and brainstem (attenuation) perfusion pressure was decreased. These changes returned to normal when the vein was repaired (Fig. 4).

CASE 4, B.R. Bradycardia due to retraction of the jugular vein

A 65-year-old woman, while undergoing proximal left vertebral artery reconstruction, developed bradycardia and slowing of EEG frequencies 10 sec after retraction of the jugular vein. There was no change in brachial blood pressure. The abnormality disappeared promptly with release of the pressure and would reappear when pressure was reapplied. Atropine was administered intravenously.

Comment: Bradycardia may be induced by retraction or clamping the jugular vein. Whether this is a direct result of intracranial



FIG. 2. Physiologic pacing works. The EEG returned to normal when paced with a physiological pacemaker, the atrial synchronous, which retains the atria] contribution. Case 2, H.T.

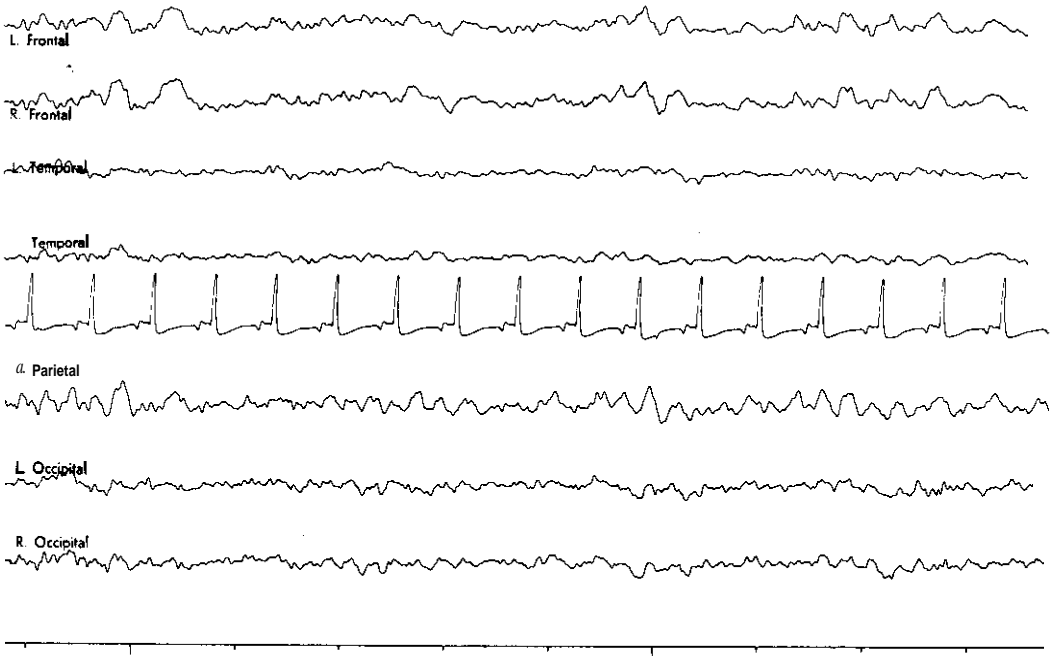


FIG. 3. Intracranial hypertension, jugular ligation, and EEG. Global slowing and 30% attenuation following ligation of the left jugular vein. Heart rate and BP unchanged. Case 3, C. W.

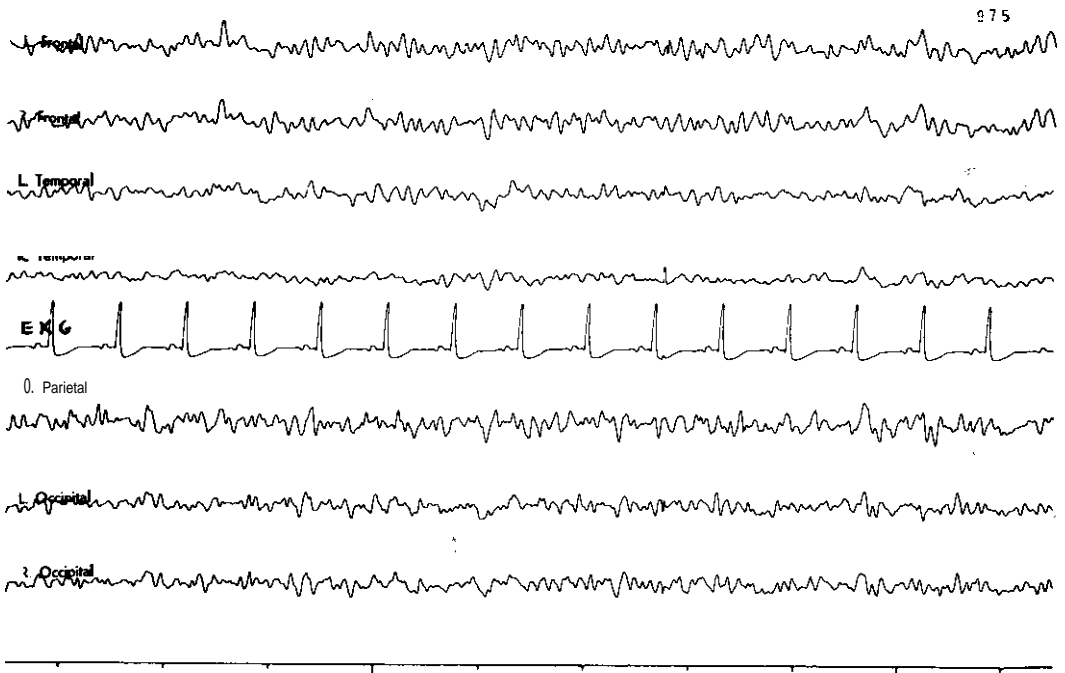


FIG. 4. Cerebral perfusion pressure and the EEG. Normal intraoperative EEG after repair of jugular vein, reduction of intracranial hypertension, and improvement of cerebral perfusion pressure. Case 3, C.W.

hypertension or due to traction on the cervical „sympathetics cannot be proven. But it was observed that the venous pressure on the side of the retractor near the head was markedly elevated. Intracranial venous hypertension due to retraction of a dominant or solitary jugular vein will reduce cerebral perfusion pressure (2,32).

NEUROGENIC STRESS

It has long been appreciated that the stimulation of the carotid sinus can result in bradycardia (14); however, other nerves may be subjected to physical stress by the arterial pulsation, with varying impact on the cardiac rate and rhythm. It has been shown (22) that stimulation of the left stellate ganglion produces a prominent shift in the intrinsic pacemaker of the heart from high on the atrium to the low atrium or to the A-V

node, and may lead to ventricular fibrillation. Repeated bouts of tachycardia (16) and syncope due to the prolonged Q-T syndrome (19) have responded to excision of the left stellate ganglion.

CASE 5, C.S.

Stellate ganglion and vertebral artery mobilization

A 45-year-old athletic woman presented with positional vertigo induced by head rotation. Angiography revealed entrapment and obstruction of the left proximal vertebral artery by sympathetic fibers. The patient's intraoperative EEG was normal until mobilization of the vertebral artery was undertaken. This required traction on the nerve fibers and division and retraction of the stellate ganglion, so that the vertebral artery could be mobilized to the vertebral foramen. During this manipulation, a change in the intrinsic pacemaker occurred (Fig. 5) and a nodal

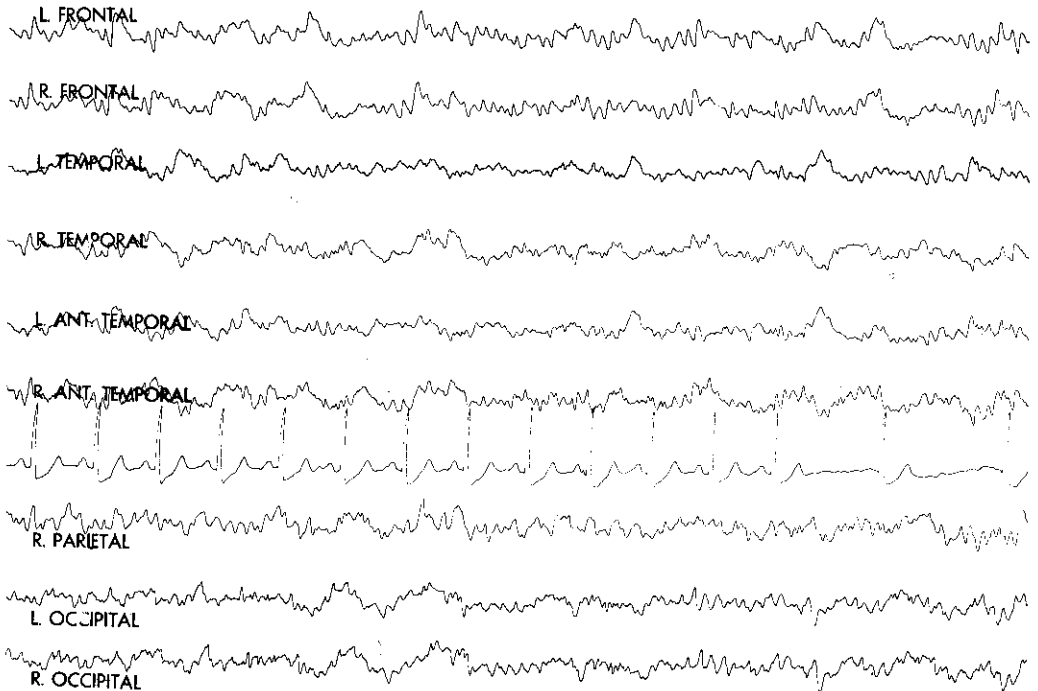


FIG. 5. Vertebral artery constriction, stellate ganglion, and EEG. Manipulation of the left stellate ganglion to decompress the proximal VA on the left side produced a nodal rhythm and gradual attenuation of the EEG. Case 5, C.S.

rhythm developed. This rhythm was accompanied by gradually increasing attenuation of the EEG over a period of 55 sec (Fig. 6), when it was terminated by blockade of the stellate ganglion (1% lidocaine).

Comment: The relationship of the stellate ganglion to vertebral insufficiency and cardiac dysrhythmia is ill defined. Nonetheless, the relationship does exist and may merit more serious investigation. With continuous EEG monitoring, the diagnosis of the cardiac conduction disturbance and determination of the hemodynamic significance of the bradycardia to the brain was achieved and proper treatment instituted without delay. The response of the EEG and the continual recording of the ECG were critical to the detection, documentation, and treatment of the dysrhythmia in this patient.

CLAMPING OF THE CAROTID AND/OR VERTEBRAL ARTERIES

The primary use of intraoperative EEG monitoring during cerebrovascular surgery has been to detect threatening ischemic changes when clamping the carotid artery (4,5,8,11,24,26). A 3-min trial of clamping of the carotid and/or vertebral artery was employed in all patients. Two types of changes are seen: slowing of the frequencies and reduction of voltage or attenuation, either diffuse or focal, alone or in combination. Slowing of the frequencies is the most common abnormality encountered. Reduction of voltage of the beta frequencies usually accompanies slowing but is less significant than attenuation of the anesthesia-induced, alpha-like frequencies.

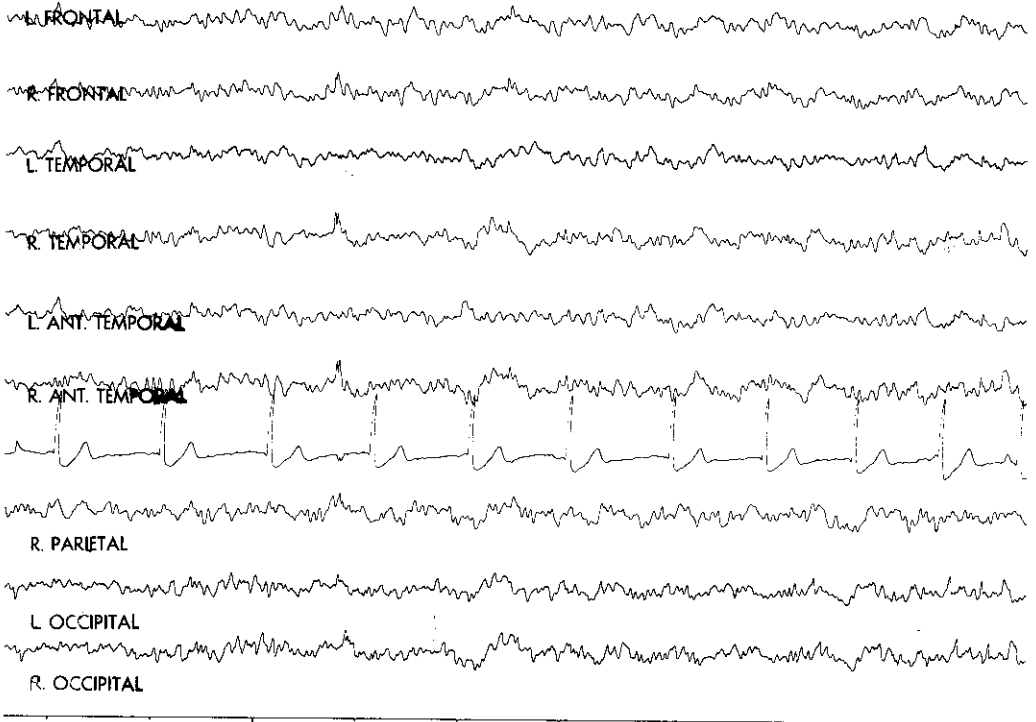


FIG. 6. Stellate block, cardiac rhythm, and the EEG. The nodal rhythm terminated 55 sec after stellate block with 1% lidocaine. The EEG normalized 90 sec later. Case 5, C.S.

Slowing with occlusion of the carotid artery may be of short duration and appear maximally in the frontal-parietal areas. This slowing commonly corrects itself over a period of 60 to 100 sec. Slowing of this type may also be corrected with a mild Trendelenburg position. On the other hand, attenuation in the EEG below 50% following carotid clamping is most serious and demands immediate correction (8). It is generally associated with slowing of the frequencies and suggests that collateral flow is inadequate for brain survival. Attenuation may be global or may occur only in the ipsilateral hemisphere. Unless cerebral perfusion is quickly improved, as indicated by improvement in the attenuation, neurologic deficit may result.

In our series, slowing and attenuation following cross clamping of the carotid artery necessitated the use of a shunt in only two patients. Although significant EEG improvement followed shunt placement, complete correction of these abnormalities did not occur until flow was restored through the reconstructed vessel. It was postulated that the internal diameter of the shunt was not sufficiently large to provide pre-clamping flow volume in patients with a high flow in the stenotic artery.

THE HIGH-RISK PATIENT

Matsumoto and associates (18) have shown that the inherent risk of severe brain ischemia during reconstruction of the carotid arteries is greater in patients with multiple neurovascular arterial lesions. Ordinarily, carotid clamping is tolerated because blood flow is stolen from the opposite carotid or from the vertebral system. The creation of interhemispheric or a hindbrain-forebrain steal is inherent whenever a carotid or vertebral artery is occluded. The EEG can not only be used to evaluate tolerance to clamping but to evaluate the direction and the severity of the shunting that is occurring.

CASE 6, J.M.

*Normal EEG, bilateral carotid stenosis
Interhemispheric steal and overextended
vascular bed*

A 62-year-old white man presented with a 90% stenosis of the left internal carotid artery (ICA) and

a 70% stenosis of the right ICA. The preoperative EEG and the EEG noted with anesthesia were normal (Fig. 7). With left carotid clamping, this patient developed a 30% attenuation in the left frontal and anterior temporal areas within 10 sec (4); 40 sec later, slowing of the frequencies of the right frontal-parietal region occurred (Fig. 8). A shunt was not used. These abnormalities cleared upon completion of the reconstruction and resumption of perfusion. There was no neurologic deficit.

Comment: The steal from the contralateral hemisphere was of such magnitude that the carotid with lesser stenosis could not supply either vascular bed adequately; the right carotid could not supply the overextended vascular bed to sustain normal cortical function (Chapter 1). Yet the abnormalities were not so profound as to warrant shunting.

CASE 7, W.D.

Abnormal EEG, bilateral carotid stenosis

An 86-year-old white woman had an abnormal presurgical EEG with mild theta slowing in the frontal areas. Angiography revealed an 80% stenosis of the left ICA and 70% stenosis of the right ICA. Clamping of the left carotid resulted in an immediate attenuation of 40% on the ipsilateral side and increased slowing in the contralateral frontal-parietal areas. These abnormalities cleared with the reestablishment of flow after the left carotid endarterectomy. This patient exhibited no localizing neurologic deficit in the postoperative period.

Comment: The blood-flow capacity of the carotid arteries was not adequate to sustain the EEG under conditions of rest. With left carotid clamping, the steal from the right hemisphere was created and the changes in both hemispheres were more profound than those of the previous patient.

CASE 8, R.S.

*Hindbrain forebrain steal with carotid and
vertebral clamping*

A 66-year-old white man underwent distal reconstruction of the solitary left vertebral artery. Both ICAs appeared normal on angiogram but the right external carotid artery (ECA) was occluded. Left carotid compression was not tolerated during the preoperative CHE.

At surgery, clamping of the LVA produced a modified burst-suppression pattern with 60% attenuation

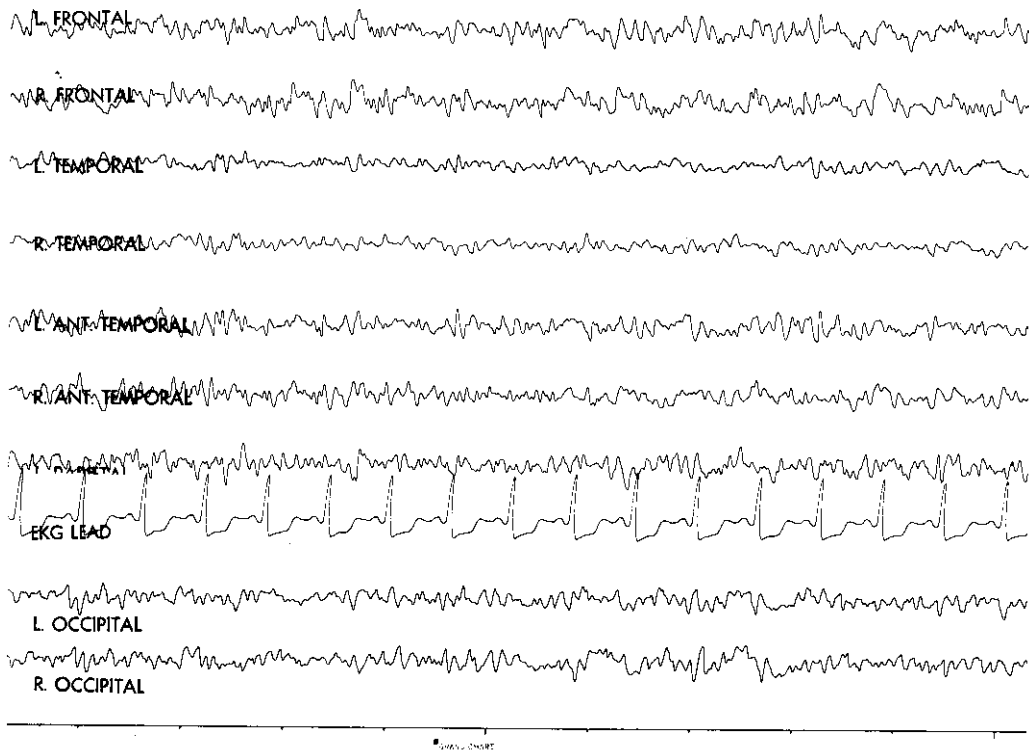


FIG. 7. Bilateral carotid stenosis and normal EEG. Patient with 90% stenosis of left ICA and 70% stenosis of right ICA. Normal intmoperative EEG. Case 6, I.M.

and intermittent bursts of theta frequencies (Figs. 9 and 10), which slowly corrected itself over a 6-min period. This is a hindbrain response. A 10% reduction of voltage persisted in the left cerebral hemisphere, and mild slowing appeared in the right frontal-temporal areas. Clamping of the left common carotid artery (CCA) was accompanied by marked slowing and attenuation of the entire record, most evident in the frontal and parietal areas. On the basis of this EEG response, the vein bypass to the distal vertebral artery (VA) was anastomosed to the subclavian rather than to the CCA. Slight left hemisphere attenuation was evident throughout the period of occlusion of the VA but corrected itself within 20 sec after the establishment of blood flow through the VA. (See also Chapter 23.)

Comment: Initial VA compression provoked severe attenuation and bursts of theta activity, which is a hindbrain response. Left carotid compression provoked a forebrain steal from the hindbrain with ischemia in both forebrain and hindbrain. Vertebral artery occlusion provoked a hindbrain steal from the forebrain, but of lesser magnitude than that observed with left

carotid compression. Since carotid compression was not tolerated, the vertebral bypass arose from the subclavian artery. Should the VA reconstruction fail because of vessel friability, the sensitivity to carotid clamping will worsen. For this reason, if both carotid and vertebral arteries are obstructed, reconstruction of the carotid first is desirable. This patient has been free of syncopal episodes since surgery, but he continues to be intolerant of left carotid compression. (Confer Chapter 23, case R.S.)

CASE 9, L.G.

Side of greater stenosis intolerant to carotid clamping

A 75-year-old woman had a 90% stenosis of the left ICA and a 70% stenosis of the right. During CHE there was marked intolerance to left carotid compression, the side of greater stenosis. In the operating room under light anesthesia, left carotid compression low in the neck was repeated and produced a mild bradycardia with hypotension. Within

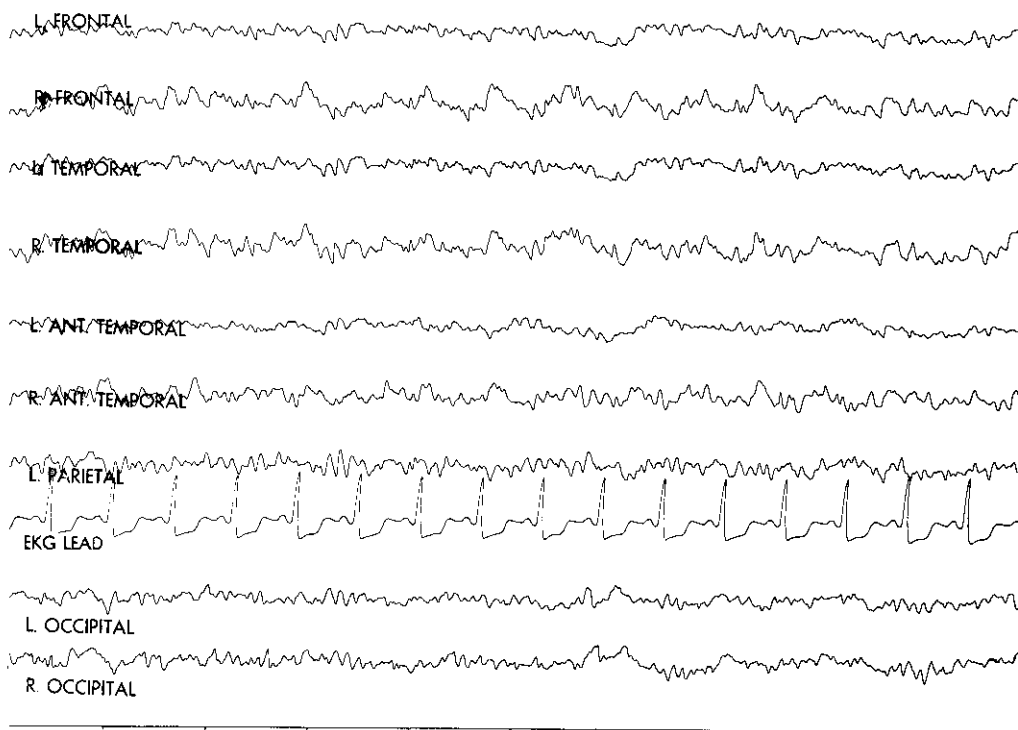


FIG. 8. Interhemispheric steal, contralateral ischemia (mild). After clamping of left ICA, 30% attenuation of the EEG in the left hemisphere was followed in 40 sec by slowing in the right carotid distribution. Clamping of the left carotid artery extends the vascular bed of the obstructed right carotid beyond its ability to maintain adequate cerebral perfusion. Case 6, J.M.

10 sec, a burst-suppression pattern was apparent in the EEG (Fig. 11). Gradual improvement and normalization occurred during the next 10 min. Right carotid compression was uneventful.

Comment: Ordinarily, the side of greater stenosis of the ICA tolerates clamping best. In this patient, this was not the case. Reconstruction of the less stenotic and less sensitive right carotid artery was completed without incident.

THE AGGRAVATED STEAL (SURGICAL)

The most common cause of EEG change in our series was blood loss from the suture line or during backflow to eliminate air or flush the anastomotic site. Although carotid or vertebral clamping always produce an intracranial steal, backbleeding from the artery greatly aggravates

this steal. With proximal VA surgery, backbleeding from the VA steals into the subclavian artery and is compounded by the reflex hyperemia of the arm. Change related to surgical events was seen more frequently during vertebral than carotid surgery.

Carotid (Forebrain) Steal

Minor loss of blood from the ICA during backflow flush is reflected in the EEG as slowing, which is apparent within 4 to 6 sec in the frontal and parietal areas. Slowing with backflow from the ECA was also seen (Fig. 12). When this situation occurs the changes are more profound and rapid than those seen with internal carotid backflow. Prevention of further backflow by replacement of the vascular clamp or completion of the suture line is followed by immediate correction of slowing.

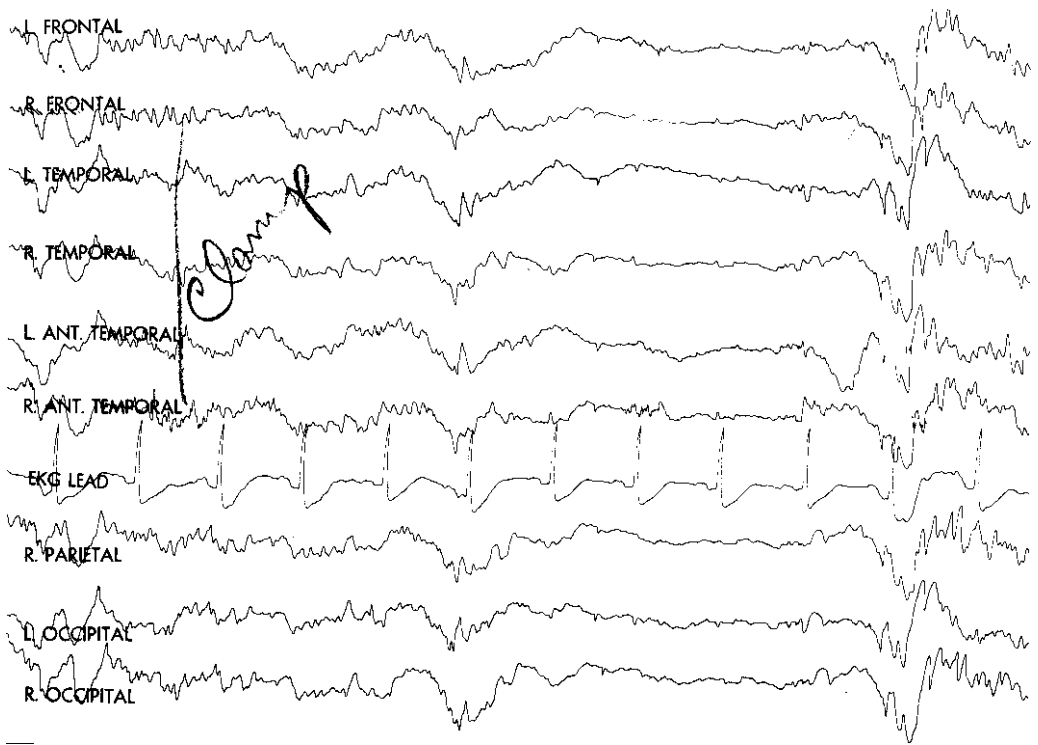


FIG. 9. Hindbrain ischemia, VA clamped. Clamping of LVA produced this modified burst-suppression pattern. A hindbrain response. Case 8, R.S. (See Chapter 23.)

Vertebral (Hindbrain) Steal and Reflex Hyperemia

During surgery of the proximal VA, a subclavian steal is precipitated when the VA and the distal subclavian artery clamps are released but the proximal subclavian artery clamp remains in place. This is done in order to flush any microthrombi on the suture line into the arm and is accompanied by immediate ischemic changes in the EEG. An EEG (Fig. 13) before removal of clamps shows normal interoperative frequencies during reconstruction of the VA. Ten seconds after the vertebral clamp is removed slowing is evident and maximal in the carotid distribution (Fig. 14). Recovery from hindbrain steal is slow, with changes persisting for 90 to 100 sec after the vertebral clamp is reapplied or the proximal subclavian clamp is removed.

Comment: With clamping of the carotid artery, 3 of 134 patients with bilateral carotid lesions developed EEG evidence of ischemia deep in the ipsilateral carotid bed (attenuation) and also in the contralateral cortical carotid bed (slowing) (13). Six patients who tolerated carotid clamping without EEG change developed EEG findings of attenuation over the ipsilateral hemisphere and slowing over the contralateral carotid bed during backbleeding, indicating the frequency of aggravation.

Every patient undergoing distal VA bypass showed EEG evidence of hindbrain steal when backbleeding. Only 3 patients undergoing distal VA reconstruction failed to show this steal phenomenon: 2 had aplastic basilar arteries and 1 had an occlusion of the basilar artery. These findings are significant, for they suggest that the EEG reflects changes during aggravated VA steal only when there is free communication

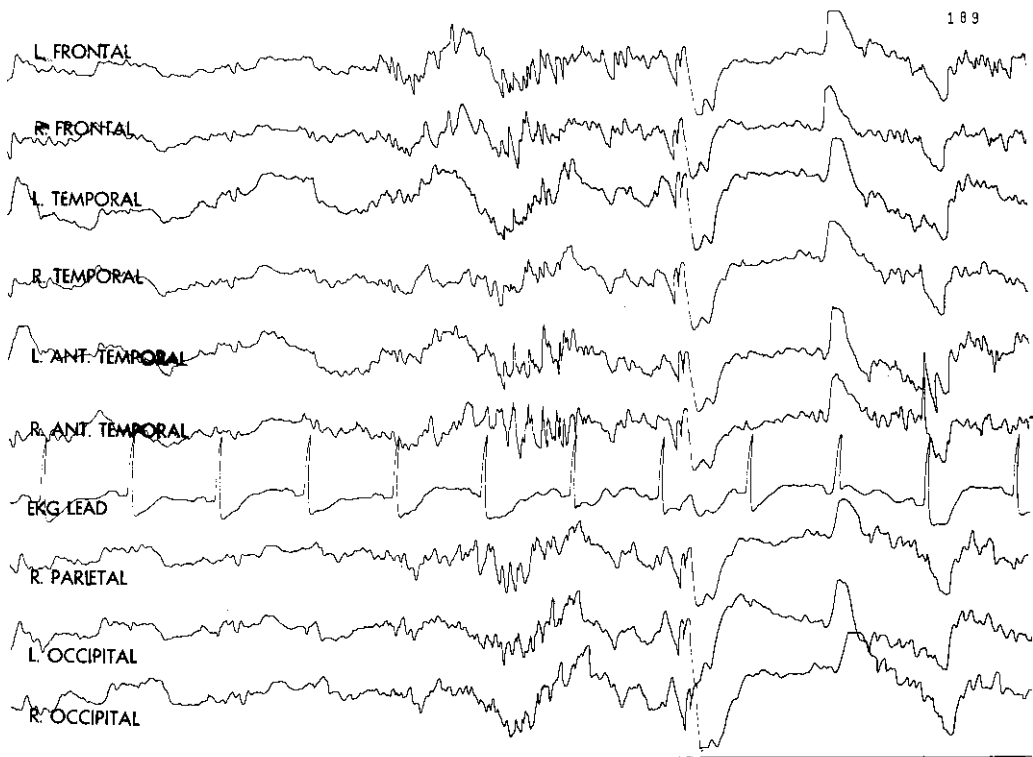


FIG. 10. Hindbrain steal, interhemispheric steal, and VA surgery. The burst suppression slowly corrected itself in 6 min but was followed by attenuation in the left hemisphere and moderate slowing over the right carotid bed: a hindbrain and interhemispheric steal. The frontal lobes were dependent on VA blood flow. Case 8, R.S.

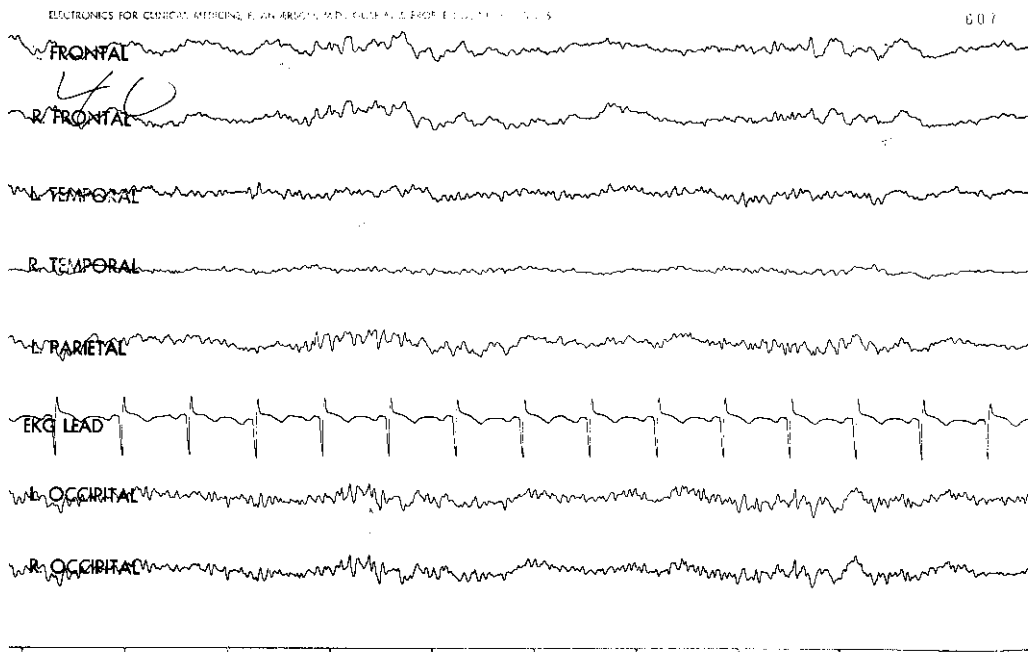


FIG. 11. Greater carotid stenosis tolerated compression poorly. Preliminary compression of the carotid of high-grade stenosis (left ICA, 90%) produced an immediate burst-suppression pattern. The side of lesser stenosis exhibited greater tolerance to carotid occlusion and was reconstructed. As a rule, the side of greater stenosis tolerates carotid compression better, but not always. Case 9, L.G.

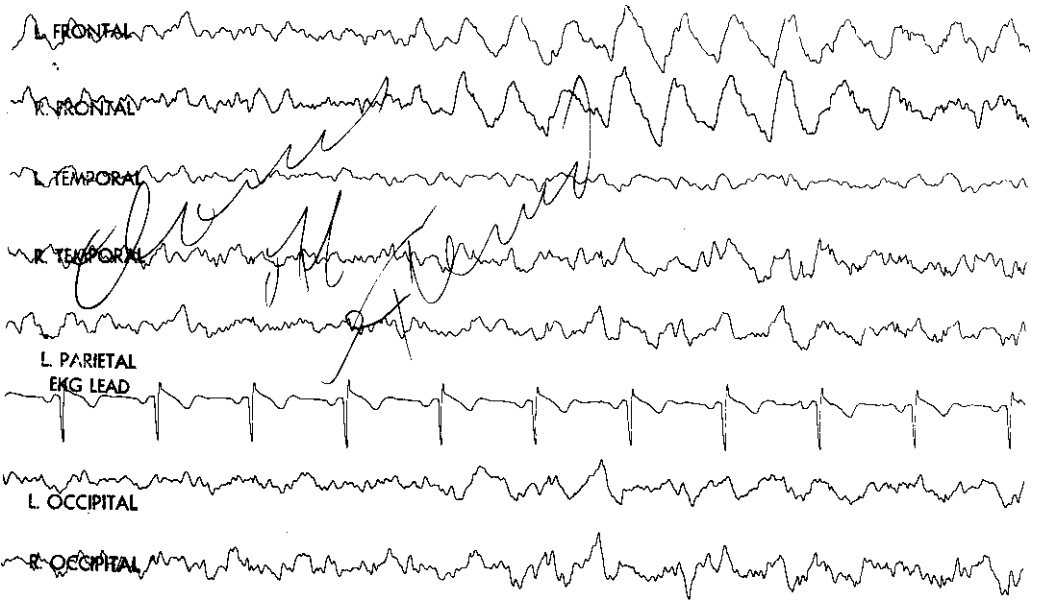


FIG. 12. External carotid artery-the surgical steal. Clamping of the carotid or vertebral artery creates an intracranial steal. Backbleeding aggravates the intracranial steal. When the ECA constitutes a major collateral, backbleeding can produce slowing of the frontal-parietal frequencies. A carotid (forebrain) steal.

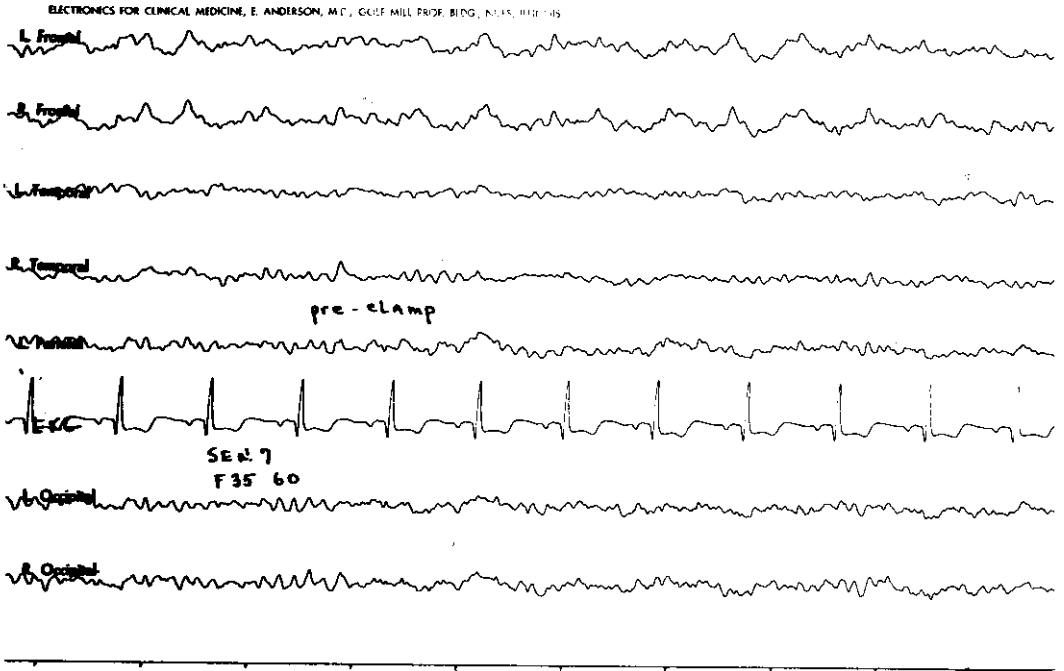


FIG. 13. Proximal vertebral surgery-tolerance to clamping. Normal intraoperative EEG during VA reconstruction. (From ref. 6.)

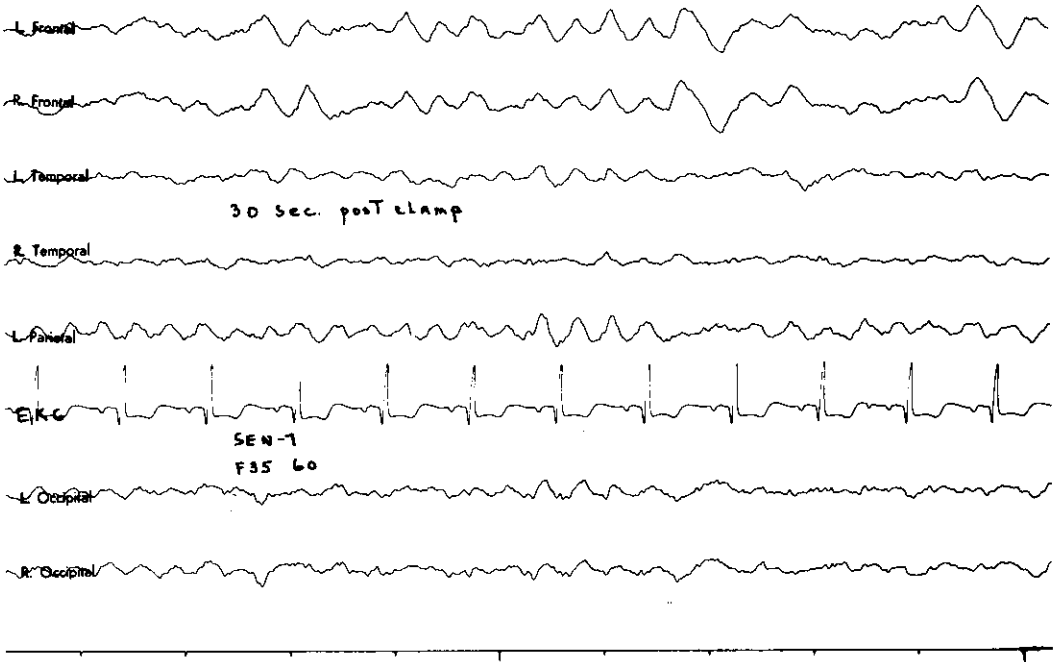


FIG. 14. Proximal vertebral surgery-intolerance to unclamping. Ten seconds after removal of the vertebral and distal subclavian clamp to flush the VA flow into the arm, attenuation of the EEG is evident and slowing is seen maximally in the carotid distribution. The created subclavian steal is aggravated by the reflex hyperemia of the arm (Chapter 20).

with the circle of Willis. Not all proximal VA reconstructions showed EEG changes, because in some a single partial occlusion vascular clamp was applied and its release restored forward flow without the development of the steal. Restoring subclavian artery flow for a period of time before opening the VA clamp permits the reflex hyperemia of the arm to abate, thus minimizing the subclavian steal if backbleeding over the suture line is desired.

DISCUSSION

In selecting patients for whom shunting is advisable during carotid artery reconstruction, the value of intraoperative EEG monitoring to detect cerebral ischemia has been established by numerous studies. We have evaluated the hemodynamic significance of rapidly occurring EEG changes during 225 reconstructions of the carotid and vertebral arteries. Three types of events occur: (a) clamping events; (b) periop-

erative events related to surgical maneuvers or anesthesia; and (c) aggravation of existing steals by surgical events.

Clamping Events

It has been adequately shown by Boysen (5) that clamping of a carotid artery may produce a slowing of EEG frequencies or attenuation and that the latter is associated with severe ischemia that demands immediate attention or placement of a shunt. Symon et al. (27) has shown that there exists a wide range of well-tolerated cerebral blood flow. Only when the perfusion falls to 40% of normal do EEG changes commence, and cell death occurs when flows of less than 20% occur.

Perioperative Events

When the patient is in an optimum cardiovascular status, changes in heart rate due to

anesthesia incidents, carotid sinus reflex (15), manipulation of autonomic nerve fibers (particularly the stellate ganglion) (22), and clamping an artery or retracting the jugular vein may still induce a critical reduction of brain blood flow. Such a reduction, especially in patients with severe regional compromise of blood flow (15,21) and systemic deficits, can be a double hazard and increase the potential of postoperative deficit. Vertebral artery surgery elicits more abnormal responses than does carotid surgery. The onset of changes related to these stresses can readily be seen with the EEG during carotid or vertebral artery surgery, and appropriate action can be taken to correct the deficiency.

The Surgical Steal

The most significant physiologic phenomenon correlated with EEG findings that gives insight into some heretofore unexplained EEG changes is the surgical steal. Backbleeding with minimal blood loss from the internal or external carotid arteries can aggravate a steal from the contralateral and/or ipsilateral carotid bed or from the hindbrain. EEG changes indicative of progressive ischemia occur immediately. Slowing of frequencies occurs in the respective regions. If an inadequate collateral capacity is not present, attenuation will occur in the ipsilateral hemisphere and slowing can be seen in the contralateral carotid distribution.

It is not likely that collateral flow can be adequately evaluated preoperatively with arteriograms in the patient with bilateral carotid or carotid and vertebral artery lesions. However, the EEG may identify those patients with compromised collateral flow at the time of arterial clamping. Here the EEG will reflect the ischemia in the contralateral cerebral hemisphere by slowing in the frontal-temporal-parietal areas 10 to 20 sec after the onset of attenuation in the ipsilateral hemisphere. The attenuation is seen almost immediately after clamping. When the attenuation does not exceed 50% over the first 5 min of clamping, neurologic deficit does not follow the procedure.

The production of a surgical steal by back-

bleeding from the VA gives insight into the capacity of the collateral system through the circle of Willis with the rest of the brain. When the circle is intact and backbleeding is produced, evidence of steal from the carotid system is present in all cases of VA reconstruction. The lack of free communication between the forebrain and hindbrain because of basilar occlusion was made evident in 3 cases by the lack of forebrain ischemia with VA backflow. For this group of patients, the EEG does not reflect hindbrain perfusion, and the supratentorial EEG is useful only in monitoring carotid clamping during the performance of the carotid anastomosis of the carotid distal VA bypass.

One patient (Case 3) with a single LVA and occlusion of the right ECA developed a hindbrain steal aggravated by backbleeding through that VA when the occluding clamp was repositioned. This steal reproduced a burst-suppression pattern for 30 sec and was followed by increased attenuation in the left hemisphere and increased slowing in the right frontal-temporal areas, a combined hindbrain and interhemispheric steal. All these changes cleared with the reestablishment of flow to the VA.

The prediction of collateral flow capacity is not possible with angiographic studies. The use of CT dynamic scans may improve our knowledge, but these studies are performed when the patient is at rest, without stress, and surgery is a situation of stress. Continuous intraoperative EEG monitoring with careful attention to surgical events, techniques, and manipulations can provide valuable information about the cerebral perfusion and systemic hemodynamics that is impossible to acquire by any other technique. The information derived from this type of EEG monitoring has provided a better understanding of the dynamics of intracranial blood flow, providing an invaluable guide to the surgeon and an increased margin of safety for the patient.

CONCLUSION

Continuous EEG monitoring during carotid and vertebral artery surgery has given new insight into the dynamic nature of brain blood flow. Surgically aggravated forebrain-hindbrain

and interhemispheric steals have allowed us to evaluate EEG changes not associated with initial clamping and in turn offer an explanation of some previously unexplained changes seen with clamping. These dynamic changes seen with surgical steals reflect the adequacy and the interdependence of the carotid and vertebral system. Armed with this understanding, the maintenance of cerebral perfusion may be better achieved through correction of nonclamping incidents that compromise systemic hemodynamics. With this on-line knowledge, the surgeon can tailor his approach and technique to the needs of the individual patient and can employ new techniques with confidence. More important, an understanding of the hemodynamic mechanisms should enhance the value of the EEG in the diagnosis of problems encountered outside the operating room. The EEG should be better appreciated for its capability to reflect the hemodynamic status of the brain, just as the ECG is employed to reflect cardiac perfusion.

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