THE ELECTROENCEPHALOGRAM IN
SUBDURAL HEMATOMA

WITH A REVIEW OF THE LITERATURE AND THE
PRESENTATION OF SEVEN CASES*

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The pathology and treatment of subdural hematoma is well understood but the clinical picture is subject to great variance. The writers are impressed, both in clinical experience and in the review of the literature, by the difficulty with which an early diagnosis is made.4,6 Most publications on subdural hematoma are prefaced by statements that emphasize the inconsistent symptomatology and difficulty in diagnosis. It is the attitude of some surgeons that whenever subdural hematoma is suspected a simple and expedient method of proof is to perform bilateral trephines.3 With such a routine the percentage of negative explorations increases. It is desirable to place the trephine openings in only those patients having a surgical lesion. This feeling gains added strength since trephine openings have an implication in the patient’s industrial, insurance and psychic future.

For these reasons additional diagnostic procedures have been employed. Pneumoencephalography is not without risk and is considered contraindicated by some authorities.8 In the study of subdural hematoma, as in all neurological disease, diagnostic tests have been directed mainly toward disturbances of anatomical relations. With the confirmation of Berger’s observation that the electrical activity of the brain followed a certain rhythmic pattern, and that in pathological states this fundamental pattern was altered, a useful diagnostic aid was placed in the hands of the clinician. The electroencephalograph offers a new and different approach in that alteration in the physiologic (electric) activity of the brain can be studied directly as well as indirectly. With subdural hematoma the abnormalities in electrical activity of the brain are shown to be altered dependent on: (1) the amount of the bleeding, (2) the location, and (3) the time interval between the bleeding and the EEG.

There are reported in the literature several clinical and experimental observations on the EEG in subdural clot that appear conflicting. The EEG has been described as showing (1) increased voltage and slow wave pattern

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on the side of the lesion; (2) decreased voltage and slow wave pattern on the side of the lesion with normal pattern on the contralateral side; (3) decreased voltage and slow wave pattern on the side of the hemorrhage with increased voltage and fast wave activity on the contralateral side; or (4) normal electrical activity.

Walter in 1939,14 who was the first to report focal abnormal electrical activity of the brain with subdural hematoma, concluded that a "silent area" existed over the site of the hematoma. He apparently used the term "silent area" as meaning an area of decreased electrical activity (amplitude).

Cohn,1 in a carefully executed experiment with cats, produced subdural hemorrhages by instilling the animals' own blood into the subdural space. He was able to produce varied EEG findings by taking recordings at intervals up to 21 days. The abnormal electrical activity of the brain in these animals correlated directly with the extent and duration of the hemorrhage, which in turn caused varying degrees of injury to the cortical neurones. With the experimentally produced subdural hematoma there was decreased electrical activity over the site of the hematoma after the first 30 minutes, which persisted for at least 1 week. He found that subdural hematoma of 1 week or more in duration may show: (1) focal decreased activity (amplitude) over the site of the hematoma; (2) generalized slow waves with a focus of abnormality; or (3) the tracing may be normal.

Cohn et al.,2 in reporting 6 clinical cases, stated that the EEG showed one of two primary characteristics; a maximum of high voltage slow waves over the lesion (3 cases) or slow wave activity of reduced amplitude over the site of the disturbance (2 cases). He was unable to explain the absence of electrical changes in 1 case of surgically proved subdural hematoma.

Gurdjian and Webster,5 in their article on intracranial hemorrhage, commented that the EEG may be of important assistance in the diagnosis of subdural hematoma and stated that correct localization had been accomplished by this technique in 40 per cent of their cases. They did not describe the criteria used for localization except to state that their records showed "disorganization of electrical pattern on the affected side." Of the 3 tracings that they showed, each case appeared to us to show decreased amplitude over the hemisphere underlying the hematoma. They failed to comment on this amplitude variation.

Ulett12 produced subdural hematomas in dogs experimentally and concluded that decreased voltage occurred over the site of the hematoma with no changes being observed over the contralateral side. This abnormality persisted until the dogs were killed 2 days later.

Heersema and Freeman6 reported that of 25 cases of subdural hematoma the EEG was helpful in one-half and disclosed unilateral delta waves. They stated that the delta waves were so extensive in some instances that the EEG was not an aid to localizing the lesion. In 12 per cent of their cases the EEG findings were normal but these were considered erroneous. From their article it is impossible to draw any definite conclusions. They failed to give
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the electrode locations, the type of tracings, the duration of the hematoma prior to the EEG and the extent of the hematoma over the hemisphere as shown at surgery or autopsy. They made no mention of amplitude variations.

Sjaardema and Glaser, in a report of 3 verified cases of subdural hematoma, concluded that the EEG pattern consisted of high voltage beta activity combined with delta waves. They felt this pattern was not pathognomonic of subdural hematoma since it had been observed in other pathological states. Careful analysis of their cases and of tracings in their Case 3 (the patient expired) showed the brain to have sustained multiple severe injuries. We conclude that their EEG was not representative of a “pure” subdural pattern. In their Case 2 the tracing presented in Fig. 2 appears to the writers to show decreased amplitude over the site of the hematoma. The representative tracing in their Case 1 was filled with artifacts and no conclusion seemed apparent.

Rogers, in a single case report of chronic subdural hematoma, showed the EGG to be characterized by a “very low potential discharge at varying frequencies in the right fronto-temporal region, most definite in the neighborhood of the Sylvian point.” A subtemporal craniotomy revealed a chronic subdural hematoma in that area.

Jasper et al. found that with a subdural collection of fluid the EEG showed decreased amplitude over the site of the hematoma when a bipolar tracing was taken with the recording electrodes placed over the hematoma and not adjacent to it. Their observations were based on clinical cases. In a later article they concluded that an asymmetry in amplitude (with the decreased amplitude being over the hemisphere of the hematoma) of simultaneous records from bilaterally homologous regions was the most reliable localizing criterion.

The following 7 proved cases of unilateral subdural hematoma show EEG patterns that correlate with the operative findings. Following the surgical removal of the hematomas the abnormal EEGs progressively improved and in the 6 surviving patients returned to normal over a period of 1 to 5 months.

CASE REPORTS

Case 1. W.B., a white male aged 36, was admitted to the University Hospital in a moderate stupor on Nov. 16, 1947. On Nov. 1, 1947 he had been found unconscious on the street after allegedly having been struck by an auto. He regained consciousness 48 hours after injury and was discharged from a county hospital ambulatory on the 10th day. He was admitted to this hospital 16 days after injury because of headache, nausea, vomiting and stupor.

Examination. He had pulse 64, respirations 30, bilateral early papilledema, dilated and fixed right pupil, right hemiparesis, and unsustained ankle clonus on the right. Lumbar puncture: initial pressure 140 mm. water; xanthochromic CSF.; traces of globulin.

Operation. A chronic encapsulated liquid subdural hematoma on the right was evacuated through a right temporoparietal trephine. Exploratory trephine on the left revealed no intracranial pathological process.
Course. He was discharged asymptomatic 10 days following operation.

EEG. The pre-operative EEG (Fig. 1), bipolar potentials, was characterized by a 75 per cent reduction in amplitude over the entire right hemisphere with low random, low voltage 1-2/sec. waves. An underlying 22/sec. spiky low-voltage pattern was seen generalized. Repeated tracings (Fig. 2) showed a gradual return to what is thought to be the normal pattern for this person.

Figure 1. Case 1. Pre-operative record. Note the great lowering of amplitude over the entire right hemisphere, particularly RF-RP.

Figure 2. Case 1. Tracing 5 months postoperative—thought to be normal for this individual. The amplitude over the right hemisphere now equals that over the left.

Case 2. L.M., a white female aged 59, was admitted Sept. 25, 1948. She had had frequent headaches since childhood and forgetfulness for the last 2 months. Three weeks before admission she fell down a flight of stairs. Ten days later she complained of severe headache, went into coma and was transferred to this hospital.

Examination. Temperature, pulse and respirations were normal. The left pupil was slightly larger than the right. Bilateral Babinski response to plantar stimulation was elicited. There was right hemiparesis, with hyperactive deep tendon reflexes on the right.

Operation. An encapsulated liquid subdural hematoma on the right was evacuated through a right temporoparietal trephine. Exploratory trephine on the left revealed no evidence of an intracranial pathological process.
Course. Convalescence was uneventful and she was discharged asymptomatic 16 days after operation.

EEG. The pre-operative EEG (Fig. 3) showed a 50 per cent reduction in amplitude over the right fronto-temporo-parietal region during the bipolar potential tracing. Irregular 4–6/sec. sharp waves were seen mainly over the right hemisphere. A repeat tracing 2 weeks later (Fig. 4) showed a generalized 20/sec. low voltage spiky pattern with no obvious asymmetry in amplitude.

![Fig. 3. Case 2. Pre-operative tracing. Note the great reduction in amplitude over RF-RP areas (channel 2). Generalized multiple sharp and spike waves are present.](image)

![Fig. 4. Case 2. Tracing 14 days postoperative. Minimal asymmetry between RF-RP and LF-LP persists. The record generally shows great improvement.](image)

Case 3. S.J., a colored female aged 40, was admitted Oct. 25, 1948. Three weeks previously she had been struck on the right side of the head. Two weeks after injury slurred speech, drowsiness, weakness of right side of body and blurring of vision developed.

Examination. Temperature, pulse and respirations were normal. She had a right hemiparesis, hyperactive deep tendon reflexes on right, Babinski response to plantar stimulation on right, and dilated right pupil.

Operation. There was an encapsulated liquid subdural hematoma over the left hemisphere. Exploratory trephine on the right disclosed no evidence of an intracranial pathological process.
Course. She was discharged asymptomatic 17 days after the operation.

EEG. The pre-operative EEG (Fig. 5) was characterized by a 50 per cent decreased amplitude over the left hemisphere, particularly over the left frontoparietal area. Generalized sharp waves and spike forms were seen over the right hemisphere. Only occasional random 1/sec. waves were seen over the left hemisphere. A repeat tracing (Fig. 6) 1 month postoperative showed no appreciable amplitude variation.

Fig. 5. Case 3. Pre-operative tracing. Note lowered amplitude over the left hemisphere, particularly the left frontoparietal region.

Fig. 6. Case 3. Tracing 1 month postoperative. Note that the amplitude variation between hemispheres is no longer present. Random slow waves persist over the left hemisphere.

Case 4. J.H., a colored male aged 37, was admitted on Nov. 9, 1948. He had been struck on the left side of the head with a bottle 8 days previously. He had complained of headache and lethargy, and 8 days after injury weakness of the right upper and lower extremities developed and he then became stuporous.

Examination revealed pulse 60, stupor, right hemiparesis, and right central facial weakness.

Operation. An encapsulated liquid subdural hematoma on the right was evacuated through a right temporoparietal trephine opening. Exploratory trephine on the left revealed no evidence of an intracranial pathological process.

Course. He was discharged asymptomatic 7 days after operation.

EEG. The pre-operative EEG (Fig. 7), with bipolar potentials, was charac-
Fig. 7. Case 4. Pre-operative tracing. Note marked lowering of amplitude over entire right hemisphere. Random low voltage slow waves are seen over the right hemisphere.

Fig. 8. Case 4. Tracing 4 days postoperative. The asymmetry of amplitude between hemispheres is minimal. Generalized multiple high voltage slow waves are now present.

Fig. 9. Case 4. Tracing 6 months postoperative. The asymmetry of amplitude between hemispheres is no longer present, and the generalized multiple high voltage slow waves have disappeared. The pattern shows random and multiple sharp waves of increased voltage and random spike forms.
characterized by a 60 per cent reduction of amplitude over the right fronto-temporoparietal area. Random sharp waves were present generally with occasional 2/sec. slow waves over the right hemisphere. A tracing 4 days postoperative (Fig. 8) showed only minimal lowering of amplitude over the right hemisphere with multiple high voltage slow waves. Six months postoperative (Fig. 9) the multiple high voltage slow waves disappeared and random sharp waves of increased voltage and spike forms were present.

Case 5. E.B., a colored male aged 54, was admitted on Dec. 21, 1948, complaining of numbness and weakness in the right upper, and to a lesser extent, right lower extremities. Two weeks previously he had been struck on the head in a fight. Three days prior to admission he had complained of headache and became confused, and on the last day he had a convulsive seizure (details not known).

Examination. Pulse was 52, and blood pressure 200/120. He was confused and lethargic. He had dilated right pupil, right central facial weakness, right hemi-

Fig. 10. Case 5. Pre-operative record. Note the marked lowering of amplitude over the left hemisphere, most particularly the LF-LO leads. Minimal random slow waves are seen.

pareisis, and loss of position sense and stereognostic sense in the right upper extremity.

Operation. A chronic encapsulated liquid subdural hematoma on the left was evacuated through a left temporoparietal trephine. Exploratory trephine on the right revealed no intracranial pathological process.

Course. He was discharged asymptomatic 14 days after operation.

EEG. The pre-operative tracing (Fig. 10) was characterized by a 50 per cent decrease in amplitude over the left hemisphere. Random 1–2/sec. waves of increased voltage are superimposed on a fundamental frequency of 10/sec. waves.

A tracing taken immediately after operation (Fig. 11) showed no appreciable amplitude difference. The 1–2/sec. waves were replaced by random 3–4/sec. slow waves. A repeat tracing 3 weeks postoperatively (Fig. 12) showed the pattern to be approaching normal.

Case 6. M.R., a white female aged 45, was admitted Feb. 21, 1949. One hour before admission she had fallen on the sidewalk, and lapsed into profound coma.
Examination. Pulse was 60. She had absent corneal reflexes, flaccid paralysis of all extremities, most marked on left, left central facial weakness and a dilated right pupil. Within ½ hour both pupils became dilated. Bloody CSF was oozing from the left ear. Roentgenograms of skull showed a linear fracture of the left frontotemporal region.

Operation was performed immediately and a massive subdural hematoma, 4 cm. thick, over the entire right hemisphere, consisting of clotted blood, was evacuated. Exploratory trephine on the left showed no evidence of extradural or subdural hematoma.

Course. Patient expired 8 hours after operation. Postmortem examination of the brain showed marked cerebral edema, uncinate hernia on the right, and laceration of the cerebral peduncle on the left by the tentorium cerebelli.

EEG. There was approximately 60 per cent decrease in amplitude over the right hemisphere with occasional low voltage 2/sec. slow waves (Fig. 13). The left hemi-
sphere showed multiple sharp 5/sec. waves and multiple 1–2/sec. high voltage waves. The tracing showed an extremely sick brain generally and was characteristic of those included in the acute phase as described by Cohn.

Case 7. C.F., a white male aged 31, was admitted on Jan. 31, 1949. He had struck his head in the right occiput 2 weeks before. He was not rendered unconscious but sought medical aid because of headache, lethargy and weakness of right side of body.

Examination. Pulse was 56. He had bilateral early papilledema, right Babinski, and minimal right hemiparesis.

Operation. A left parietal trephine revealed a minimal subdural hematoma. An exploratory right parietal trephine showed no evidence of hematoma.

Course. He made an uneventful recovery and was discharged asymptomatic 17 days after operation.

EEG. The findings (Fig. 14) in this case were thought at first to be questionable or borderline for a subdural hematoma over the left hemisphere, with minimal
depression of the amplitude and multiple 2-3/sec. slow waves over the left frontoparieto-temporal area. The fact that the layer of subdural fluid was quite thin probably accounted for the minimal EEG abnormality.

COMMENT

In 3 of the reported cases the patient showed ipsilateral hemiparesis and in 4 a contralateral dilated pupil was present. In spite of these misleading signs it was possible to predict the location of the hematoma on EEG findings. This study represents cases presented to the electroencephalographic department over a 14-month period. Of the other craniocerebral injuries that were studied with the EEG during this period, none showed electrical evidence of subdural hematoma. The accuracy of the procedure was confirmed in each case by the subsequent course of the patient.

The characteristic EEG findings in these cases were marked lowering of the amplitude and random low voltage slow waves over the affected hemisphere. This follows the pattern found in Jasper's clinical cases, and is compatible with the experimental pattern found by Cohn. There was no evidence of concurrent brain laceration or other intracranial bleeding except in Cases 6 and 7, so it was concluded that Cases 1 to 5 were uncomplicated subdural hematomas and accordingly the pre-operative tracings in these are representative of “pure” subdural hematomas. In Case 6 there was severe contusion and laceration of the brain, but the subdural hematoma was so massive that the characteristic EEG pattern was present and dominated the tracing. Case 7 had only a minimal subdural hematoma with cerebral edema and slight subarachnoid hemorrhage. Because of the paucity of subdural blood and the associated craniocerebral injuries the pattern was not as obvious or classical but suggested the diagnosis.

After a careful review of the literature on this subject it is postulated that the many conflicting EEGs taken on patients with subdural hematoma are not representative of “pure” subdural hematoma, but may well be the result of concomitant brain damage. Other observers⁶,¹⁰ emphasize the frequency of the combination of vascular lesions and parenchymatous damage, or the combination of various types of hemorrhage such as subdural, extradural and intracerebral. Gurdjian and Webster,⁶ in their series of 30 cases of extradural hemorrhage, stated that in 11 of these there was concomitant subdural hemorrhage. They stated “a number of patients in this series had associated severe diffuse cerebral injuries” and that they frequently found subarachnoid hemorrhage to coexist with subdural hemorrhage. It is obvious that the EEGs taken on such damaged brains will give variable findings and that the asymmetry in amplitude as described by others, and seen by us, is typical only in the “pure” subdural hematoma, and only when the bipolar leads are used over the clot.

The EEG is a diagnostic aid and is to be used only as an adjunct to the clinical findings. The EEG tracing should be correlated with the history and findings, and is in no way meant to replace careful neurological examination and observation.
CONCLUSIONS

1. The clinical course of subdural hematoma is variable and the diagnosis frequently difficult to establish.

2. A review of the literature concerning EEG in subdural hematoma revealed many conflicting reports.

3. Seven cases of surgically proven unilateral chronic subdural hematoma are presented, along with their EEG pattern.

4. With the exception of 2, these are clinically and surgically cases of "pure" chronic subdural hematomas showing no concurrent brain contusion or laceration.

5. The pre-operative EEG was characterized by a marked generalized asymmetry between hemispheres, with the hemisphere underlying the hematoma showing decreased amplitude and random low voltage slow waves. This is representative of "pure" chronic subdural hematoma tracings.

6. A period of 5 months may be required for the EEG to return to normal.

7. It is our opinion that the variations in the EEGs of subdural hematomas can be explained by the presence of concomitant intracranial hemorrhage and/or brain damage.

8. Failure to take a bipolar tracing with the recording electrodes over the site of the hematoma will result in an "atypical" record.

9. The EEG is of aid in establishing the diagnosis of subdural hematoma.

REFERENCES


