EXTRADURAL HEMORRHAGE

A STUDY OF FORTY-FOUR CASES

DONALD MUNRO, M.D., AND GEORGE L. MALTBY, M.D.
BOSTON, MASS.

EXTRADURAL HEMORRHAGE is a dramatic and theoretically well-recognized traumatic lesion of the cranium. Much has been written about it in both early and recent literature. However, after reviewing the reported cases, it has been our feeling that we need make no excuse for bringing the problem forward again. Most of the teaching in medical schools and in the standard text-books of general surgery is misleading or, at the best, cursory. The so-called classical picture, with its initial unconsciousness, lucid interval and secondary period of unconsciousness plus clear spinal fluid under elevated pressure, is so rare as to be the exception rather than the rule. This classical picture has been the more firmly entrenched because some authorities, both neurosurgical and pathologic, have insisted that the bleeding is always arterial. We feel definitely that, in a certain portion of the cases, the source of the extradural bleeding is venous in origin. These facts have also been recognized by other authors.

Our series of 44 cases is somewhat larger than any group heretofore reported from one clinic. It seems to us that certain very definite conclusions may be drawn from an analysis of the results of the fairly standardized method of treatment that we have used over a number of years.

Hippocrates recommended that the cranium be perforated when injuries might be followed by serious consequences such as the extravasation of blood. Celsus advised waiting until untoward symptoms appeared before operating. Since then, many articles have been written on this subject. The more important authors are listed in the references.[1–8] The classical work on extradural hemorrhage, however, is that of W. H. A. Jacobson,[9] written in 1885. Little has been added to the picture since this appeared. He thoroughly analyzed 70 cases; 27 from Guy's Hospital and 43 from the English literature. Any report on this subject must refer frequently to his excellent clinical descriptions. Since that time, several papers reporting the clinical findings and treatment of small groups of cases have appeared annually. In 1938, Pringle[10] published what appeared to be another large series. This was a review of 71 cases of traumatic meningeal hemorrhage. However, in analyzing these cases only 23 can be considered as true extradural hemorrhages, the others being subdural hematomata.

In reviewing the literature, several things stand out: First, many of the early authors were aware of the rarity of the so-called classical picture. Second, not a few of the men writing on this subject realized the probability of venous bleeding as a source of the clot in some of their cases. Finally, there

Submitted for publication July 13, 1940.

has been no large series of cases reported previously by a single clinic in which a well-standardized method of treatment was used.

Incidence.—Extradural hemorrhages are by no means a common complication of a blow on the head. This is illustrated by the fact that in over 1,200 head injuries treated on the Neurosurgical Service of the Boston City Hospital from 1932–1939, only 44 or about 3 per cent showed extradural hematomata. LeCount and Apfelbach[11] report a study of 504 cases that had fractures of the skull discovered at autopsy. In their series, 19 per cent showed extradural hemorrhage which produced appreciable compression of the brain. Moody[12] studied 908 cases of proven fracture of the skull and found 100 instances of extradural hemorrhage (9 per cent).

Etiology and Pathogenesis.—The immediate cause of a traumatic extradural hematoma is, of course, a blow on the head, which may or may not leave external evidence of violence.

The common assumption is that extradural hematomata are always caused by a rupture of the middle meningeal artery or one of its larger branches. This belief has been inculcated by certain authoritative statements to the effect that arterial, and only arterial, bleeding was capable of stripping the dura from the bone. We feel sure that in certain instances the source of the bleeding is venous, either from a great venous sinus or one of the smaller veins. Wood Jones,[13, 14] in two excellent papers, has proven, beyond a doubt, that the grooves in the skull usually believed to be due to the pulsating arteries, are for the most part produced by the veins that accompany these arteries. His convincing discussion will not be elaborated upon here. However, in 1911, anatomists had entirely accepted his findings, although most surgeons are still not aware of this anatomic fact. Wood Jones not only made purely anatomic studies but by injection methods, in three cases in which death had occurred from massive extradural hemorrhage, proved that the venous sinuses around the arteries were torn while the arteries themselves were intact. Verbrugghen[15] has recently reviewed and reiterated the findings of Wood Jones and has reproduced one of the latter's graphic illustrations. In the majority of cases where the bleeding point was definitely observed it has been arterial, but in a smaller number the bleeding was seen to come from one of the great venous sinuses or a tributary vein. LeCount and Apfelbach say that in their series the bleeding was attributable to the anterior branch of the middle meningeal artery in 49 cases and to the posterior branch in 44 cases. They also found three instances in which the bleeding was from a laceration of the superior longitudinal sinus, and eight in which the bleeding was considered to have originated from the lateral sinus. Erichson,[7] Jacobson,[9] and Verbrugghen[15] also record instances of venous bleeding as the source of extradural clots. McKenzie[16] stresses the fact that the bleeding is not always arterial in origin; and speaks of three cerebellar clots. One of these was found at operation. They were caused by torn lateral sinuses. Our series, likewise, includes one cerebellar extradural hematoma. This was found at autopsy, the source of the bleeding being from the lateral sinus.

In our 44 cases, the source of the bleeding was definitely mentioned in

37 instances (84 per cent). In 26 (70 per cent) the bleeding was described as from a portion of the middle meningeal artery. However, it must be mentioned that if the contentions of Wood Jones and others are correct, it is probable that even in certain cases in which the middle meningeal artery was assumed to have been torn, the bleeding, especially if it had not been furious, might have been from the surrounding venous channels. In the 26 cases in which the middle meningeal was said to have been torn, there was a marked variation in the description of the difficulty of controlling the bleeding, and also in the length of the lucid interval, as compared to those with a known venous source for the hemorrhage.

In ten cases in our series there was definite mention of a venous, and only a venous, origin for the bleeding. In seven, the source was demonstrated to have been one of the lateral sinuses. In three, the bleeding was found to have originated from a rent in the sagittal sinus or from one of its large tributary veins.

Thus, a review of the literature and a study of our own experience indicates that the source of extradural bleeding is usually but not always a rupture of the middle meningeal artery or one of its branches. Venous bleeding as a source for the clot has been neglected in discussions of the etiology and pathogenesis of extradural hematoma. To thoroughly understand the clinical and therapeutic problems related to extradural hemorrhages one must be aware of this latter possibility.

TABLE I

SUMMARY OF SYMPTOMS IN 44 CASES OF EXTRADURAL HEMORRHAGE

Finding	Times Mentioned	Contra-lateral	Ipsilateral	Bilateral	Remarks
Hemiplegia	25	22	3	—	
Facial weakness	13	11	2	—	2 ipsilateral, peripheral type
Babinski	34	10	1	23	
Spasticity or rigidity of extremities	10	4	3	3	3 spastic quadriplegias
Abdominal reflexes	9	Absent 5	Absent 1	Absent 3	
Deep tendon reflexes	18	Increased 12	Increased 1		Absent in 5
Papilledema*	4			4	Interval between injury and observation: 4, 5, 7, and 11 days
Sensory changes	2	2			
Nuchal rigidity	5				
Aphasia	4				All cases showed left-sided clots
Dysarthria	2				
Convulsions	3				1 generalized 2 jacksonian
Clonus (ankle)	2	1		1	
Absent corneal reflexes	1	1			
Astereognosis	1	1			
Diminished hearing	1	1			
Ptosis of eyelid	1	1			
Kernig and Brudzin ki	1				
Divergent strabismus	1				

* McKenzie mentioned questionable disk changes in five cases, one of which he believed appeared within 24 hours.

Diagnosis.—(Table I.) Our case histories were frequently inadequate because many of the patients were unconscious when first found and no reliable story could be obtained. The cause of injury was, therefore, indefinite in the great majority of cases. However, there were only three or four in which the injury could be considered as slight. Most of the known causes were automobile accidents, assaults and falls. It has been impossible to decide from our histories which cases had sustained a general and unlocalized head injury and which had received a local blow to the temporal region.

In 23 of the 44 cases, some mention of the ingestion of alcohol was made. We are well aware of the inaccuracy of such statements as "odor of alcohol," and this finding is merely mentioned as one of the probable factors in confusing the diagnosis.

Those who write of the classical picture of extradural hematoma stress the importance of a "lucid interval." There are many reasons why this may be missing or camouflaged. Alcohol, concomitant brain injury and the amount and type of bleeding all may play a part. In our series, 21 or 40.7 per cent, of the cases showed a fairly definite lucid interval. The most prolonged was an interval of 16 days while the shortest was one and one-half hours. The average was slightly longer than three days, the mean being 48 hours. These latter statistics are very much the same as those of McKenzie.[16] Thus, we may say that in more than half the cases, no history of a "lucid interval" was obtainable.

The age of the patient was definitely mentioned in 38 of our 44 cases. The average was 36 years; the youngest patient being eight and the oldest 74. Table II shows the distribution. It is clear that this condition is rare under ten and uncommon beyond age 50. All but one of our cases were males.

TABLE II

AGE DISTRIBUTION

Age	Number of Cases
1–10	1
10–20	7
20–30	6
30–40	9
40–50	7
50–60	5
60–70	2
70–80	1

The site of the clot was mentioned in 43 cases. Twenty-six were right-sided and 15 left. One was bilateral and one occurred in the left posterior fossa.

The neurologic status of the extremities has been considered important in the diagnosis of extradural hematoma. However, it is our impression that the neurologic findings on any one examination are not as important as the shifting or changing of neurologic signs and cannot, in any event, replace an adequate history. In our series, a definite mention of unilateral weakness of the extremities was made in 25 instances. In 22, it was contralateral to the

clot while in the remaining three the weakness was ipsilateral. Thus, in 12 per cent of the instances in which a unilateral hemiparesis was present it was of no localizing value.

Little mention is made in the literature of facial weakness. In our series we found it mentioned on 13 occasions. It was contralateral to the lesion in 11 instances and ipsilateral in the other two. Both of the latter were specifically described as having the peripheral type of facial weakness, while the former were frequently referred to as of the central type. Thus, it is probably fair to assume that the two instances of ipsilateral facial weakness were due to local damage to the facial canal in the temporal bone.

Babinski's sign was present in 34 cases. It was bilateral in 23, contralateral in ten, and ipsilateral in one. It seems obvious that this sign is of no great localizing value in these cases.

Much has been written about the pupillary changes in extradural hematomata and certain authors lay a great deal of stress on the localizing value of such changes. The so-called Hutchinson's pupil, or a dilated and fixed pupil on the side of the lesion, has been considered important since Hutchinson's[6] original description in 1867. In our series, definite mention of the state of the pupils was found in 37 instances. In 18 (48 per cent) the ipsilateral and in five (13 per cent) the contralateral pupil was dilated. In 13 cases (35 per cent) there was a definite statement that the pupils were normal and equal. In the single instance of a bilateral extradural hematoma, the pupils were first equal after which the right became greater than the left. We may conclude that the unilateral, dilated, fixed pupil (Hutchinson) is by no means as common an occurrence as has been frequently stated. However, when present, it is probably ipsilateral in respect to the clot.

We are well aware of the difficulty in estimating the state of consciousness of an individual, and have already mentioned some of the extraneous factors that may play a part in the picture. Some mention of this symptom and its severity at the time these patients were admitted to the Neurosurgical Service was made in 42 of our 44 cases. In 21 cases (50 per cent) the terms "unconscious," "deeply unconscious," "coma" or "deep stupor" were used. In 15 cases (35 per cent) such terms as "drowsy," "stuporous" or "semi-stuporous" were used. The remaining six cases were described in the following terms: "Conscious but incoherent"; "amnesia for the accident but conscious"; "alert, oriented but no memory of the accident"; "uncooperative but oriented"; "irrational, resistive and restless"; "semiconscious but responds." In relation to Pringle's[10] statement about prognosis, it is interesting to note that of our 21 cases definitely in coma, 15 (71 per cent) died. In the group of 15 cases described as being drowsy or stuporous on admission, seven cases (46 per cent) died, while in the six cases that were conscious on admission, there was only one fatality.

Pringle lays special stress upon the importance of local hematomata, and bleeding from one ear. He also speaks at some length about percussion of the head over the suspicious area. We have no data on the value of this

procedure. In our series, bleeding from the ear was mentioned on eight occasions. It was ipsilateral in six, contralateral in one, and bilateral in one. Other signs of trauma such as the so-called Battle's sign, scalp hematomata, localized tenderness, ecchymosis of the eyes, and abrasions and lacerations of the scalp, were mentioned in 28 cases. In 23 of these, the signs were ipsilateral, in four contralateral, and in one bilateral. Thus, in over 82 per cent of the cases in which these factors were mentioned they were of definite localizing value.

Cerebrospinal Fluid.—There has been much argument about the value and danger of lumbar puncture in head injuries in general. We believe that the danger is minimal and that the information obtained is not only essential but of a sort that far outweighs any possible risk. Except for the intracranial pressure, however, the cerebrospinal fluid findings are of no value in making the diagnosis of extradural hematoma. They do no more than indicate the amount and type of brain damage. This is important, to be sure, as it is on this complication that the presence and length of the "lucid interval" depends. Even the pressure readings, however, are not entirely reliable as they may be falsified because of toxic dehydration or surgical shock.

Our records mentioned preoperative spinal fluid findings in 38 cases. For purposes of the analysis of the cerebrospinal fluid pressure readings, the first puncture that was made on the Neurosurgical Service is the one we have used for our statistical studies. This was chosen because in several instances, earlier punctures made by the General Surgical and Medical Services failed to give accurate pressure readings. We are aware that this arbitrary use of the first "neurosurgical" puncture may alter slightly the composite picture, for in certain instances a primarily elevated pressure may have been lowered by frequent earlier punctures, while in others an initial low pressure, due to surgical shock or dehydration, may have been raised by treatment. The initial pressure was measured in 36 cases. The average reading was 216 Mm. of water (cerebrospinal fluid). The highest pressure recorded was 500 and the lowest 50 Mm. of water. There were 12 cases (33 per cent) with an initial pressure of 150 or below, while 38 per cent were below 200 Mm. of water. Table III shows the variation in pressure:

TABLE III

PRESSURE RANGE OF CEREBROSPINAL FLUID

Pressure in Mm. Water	No. of Cases	Per Cent
50–100	2	5
100–150	10	28
150–200	2	5
200–300	11	30
300–400	5	13
400–500	6	16

It is obvious that in a third of the cases there was an apparently normal spinal fluid pressure but, without associated data as to the patient's pulse pressure and fluid metabolism, such statistics are valueless.

The character and appearance of the fluid was noted in 37 instances. The following descriptive terms were used: Clear; xanthochromic; pink; slightly bloody; and bloody. Only rarely were any accurate cell counts made. In 19 cases (51 per cent) the fluid was described as either grossly bloody or bloody. Ten cases (27 per cent) showed a fluid described as pink. In five cases (13 per cent), the fluid was xanthochromic, while in three cases (8 per cent) the fluid was clear. It is thus apparent that in 92 per cent of our patients who suffered from an extradural hematoma the associated brain injury was at least as severe as a contusion.

In attempting to correlate the spinal fluid findings with the mortality, it is possible to draw only general conclusions. This is because of the influence of such factors as surgical shock, dehydration, the degree of brain injury, the length of the preoperative period, the technical skill of the operator, the hospital operating room equipment, *etc.* In general, it can be said that patients with complicating severe brain injuries, early, excessively high cerebrospinal fluid pressures, severe toxic dehydration, and any associated shock or hemorrhage do very badly and have a poor chance of survival. Thirty-six per cent of the fatal cases had a preoperative pressure of over 300 Mm. of water, while in the group of recoveries only 21 per cent had pressures above this level. In considering the appearance of the spinal fluid, it appears that 90 per cent of the fatalities showed either bloody or pink fluid, while only 62 per cent of the recoveries had fluids of this character. This discrepancy is even more marked if we use only those cases in which the fluid was described as grossly bloody. Sixty-six per cent of the fatalities showed gross blood, while this finding was present in only 37 per cent of the recoveries. Thus, the extent of the brain damage as measured by the appearance of the cerebrospinal fluid plays a more important rôle in the prognosis than does the increase in cerebrospinal fluid pressure.

Roentgenographic Findings.—It has been our experience that the suspicion of the presence of a cerebral extradural hematoma is one of the two absolute indications for roentgenologic examination. It should be realized, however, that the so-called standard roentgenograms are always unnecessary and often inadequate. This particular examination is made only for the purpose of demonstrating a fracture line in relation to either the middle meningeal artery or to one of the large venous sinuses. No more than one film will be necessary but this film must show not only the fracture but also the groove of the artery in the bone and the shadows of the suspected sinuses. To enable the roentgenologist to make such a film, the surgeon must indicate the side of the head that is to be taken, accept the responsibility for the necessary transportation and handling of the patient, and be prepared to provide enough assistance to ensure absolute immobility of the patient's head during the single exposure. Under these conditions, he can accept a negative film as being as truly negative as is possible. In such circumstances, he will revise his estimate of the diagnostic significance of his other findings, whereas with a positive film—that is, one in which a fracture can be shown to cross any part of the artery

or the sinuses—the balance will be weighted in favor of a positive diagnosis of clot and therefore of immediate operative interference. Although the demonstration of a fracture has no prognostic significance in these cases, its diagnostic importance cannot be overrated. We have yet to see an extradural hematoma in the absence of a fracture of the bone in the immediate vicinity of the bleeding point—providing an adequate search for the latter has been made.

In the series under consideration, preoperative skull roentgenograms were taken in 18 cases (40 per cent). In three instances, the reports were negative for fracture and in each of these a fracture was found either at operation or at autopsy. Operation or autopsy also confirmed the roentgenographic findings in the remaining 15 cases. Of the 26 cases in which no roentgenograms were taken, 16 showed a definite fracture either at operation or autopsy. In ten, no fracture was demonstrated. Six of these were autopsied and four operated upon. It is our opinion that extradural hematomata probably do not occur in the absence of fracture.

Differential Diagnosis.—Detailed analysis of the cases in this study bears out the statement that one of us (D. M.[17]) has repeatedly stressed in the past: The clinical picture of extradural hemorrhage is extremely variable and cannot be depended upon from a diagnostic point of view. Even with the associated aids of history, roentgenographic and cerebrospinal fluid findings, this diagnosis remains one of the most difficult in the whole field of craniocerebral injuries. The impression of simplicity of diagnosis that one gains from the usual text-book description is very dangerous and misleading. The so-called classical picture is extremely rare.

Since Jacobson's article in 1885, little has been added to the list of conditions that may be confused with extradural hematoma. They include cerebral lacerations and contusions; localized cerebral edema; subdural hematoma; depressed skull fracture; and intracerebral hematoma (traumatic or spontaneous). Any of the preceding conditions may be accompanied by an extradural hematoma, however, which, when added to the underlying brain damage, tips the scales in favor of a fatal outcome. The problem of separating the former from the latter is frequently made much more difficult by such added complicating factors as intoxication from alcohol or other drugs, surgical shock and toxic dehydration, all of which alter the clinical and cerebrospinal fluid picture. Thus, it is obvious that in many instances the final diagnosis can only be made after a diagnostic exploratory trephination. This can be performed under local anesthesia with but little risk to the patient. It is justified because when treating a desperate situation such as an extradural hematoma and, moreover, one in which the unoperated mortality is 100 per cent, the risk to which a patient without a clot is thus exposed shrinks into insignificance in comparison with the increase in therapeutic efficiency made available to the individual who has been so unfortunate as to be suffering from extradural bleeding.

Treatment.—As soon as the diagnosis of extradural hemorrhage *cannot be disproved,* the only treatment is an immediate emergency operative procedure.

It is important that the surgeon who undertakes to operate upon these cases be well-prepared both as to mechanical equipment and skilled help. He must have available an adequate suction apparatus and a minimal supply of cranial surgical instruments. A reliable method of administering therapy intravenously must be set up at once by a special team who will have this as their sole responsibility. At least two and usually more compatible blood donors must be available as transfusions are always necessary to treat shock and to replace lost blood. They will have to be given through the intravenous set-up. Directness and speed of action are the deciding factors in bringing these cases to a successful conclusion. These preliminary precautions must be taken in every such case. If the patient is in a hospital that lacks adequate operative equipment or personnel, he should be moved at the earliest possible moment to one that has such equipment. The risk of moving even the sickest patient, is minimal when compared with the danger of operating upon an extradural hemorrhage without adequate equipment and help.

The operation can usually be performed with only local anesthesia, but if the patient is resistive and restless a few centimeters of sodium pentothal intravenously will give adequate relaxation. The ideal approach is the usual subtemporal route. This should be modified to meet individual requirements. After the bone has been removed widely enough to give adequate exposure, the clot should be evacuated as completely as possible by suction. The bleeding point must be adequately controlled. This implies technical exactness and certain occlusion of the bleeding vessel. Arteries should be clipped or tied with silk, and venous bleeding points controlled by the careful placing of adequate muscle stamps. Attempts to stop bleeding in these cases with electrocoagulation is dangerous and will usually lead to recurrence of the clot. If the artery has torn close to its entrance to the skull, the external carotid artery should be tied in the neck if there is any doubt about recurrence of the bleeding because of a slipped ligature. Having safely checked the bleeding, the dura should be opened widely in order to give an adequate decompression. We consider this latter procedure important as a protection against the untoward effects of the usual postoperative edema. The wound should be drained with one or two rubber drains led out through the bottom of the incision. These are adjusted according to the size of the cavity that remains after the clot has been removed. One may be placed inside the dura under the temporal lobe and one in the extradural cavity. They provide drainage for whatever oozing there may be after closure and should be removed in 24 hours. On occasion, quite large amounts of bone may have to be removed to adequately control bleeding, especially when it is coming from a lateral sinus. We feel that the absence of this bone is of no importance in the light of saving the patient's life, and especially so if it is removed in an area in which the temporal muscle can be employed as a covering for the opening.

The postoperative care of the patient is extremely important. He must be followed very closely and the so-called malignant postoperative edema combated with adequate dehydration and lumbar punctures. Coma may persist

for several days postoperatively. Every effort must be made to prevent pulmonary congestion and resulting pneumonia. Adequate fluids must be administered. Not uncommon complications of extradural clots are certain midbrain signs which are probably due to the gross shifting of the entire intracranial contents. Perhaps the most dreaded of these is hyperthermia. In the treatment of this condition we have used frequent tepid sponges with remarkable success. Tepid sponges seem to cause dilatation of the peripheral vessels and facilitate heat loss more readily than the alcohol and ice water sponges previously employed. Removal of all covers, playing an electric fan on the patient while still wet, and cold water or shaved ice enemata have also helped. Erickson[18] has recently given an excellent summary of the possible pathogenesis and treatment of so-called neurogenic hyperthermia. He stresses the importance of recognizing the condition in its inception by the accompanying tachycardia and hyperpnea and instituting treatment early. In the extradural cases, however, the usual first sign is the excessive and steady rise in temperature.

Mortality and Complications.—The mortality from extradural hematomata has always been very high except for the unbelievably good results of J. Hill[1] in 1772. The figure usually quoted to-day is around 50 per cent. This comes from clinics in which there are well-equipped neurosurgical units. There can be no question but that the general mortality is much higher. Our 44 extradural hematomata show a total or case mortality of 59 per cent, and an operative mortality of 52 per cent.

Thirty-eight (86 per cent) were operated upon. The six unoperated cases all died. Twenty of the operated cases died. Table IV gives the mortality statistics for the years covered in this study.

TABLE IV

MORTALITY

Year	Total Mortality	No. of Cases	No. Operated
1932	66	3	3
1933	50	4	3
1934	66	6	6
1935	66	6	5
1936	75	4	3
1937	37	8	8
1938	60	5	4
1939	62	8	6

In the six cases that died without being operated upon, the mortality was 100 per cent. One died before operation because of uncontrollable hemorrhage from some unknown source in the nasopharynx. We consider this an unavoidable fatality. Two cases died almost immediately after admission to the accident floor and before even the barest preparations could be made for operation. The remaining three cases died because of the delay in operating. In one, difficulty was encountered in obtaining operative permission. Of the other two, one was kept on the General Surgical Service for 36 hours before the condition was recognized and neurosurgical consultation requested, and the other died on the Neurosurgical Service with the incorrect diagnosis of

lacerated brain and dehydration. In one-half of these six cases, the diagnosis of extradural hematoma was made before death, while in the other half either a diagnosis of lacerated brain or subdural hematoma was made. At least half of them could have been saved by earlier diagnosis and the more rapid institution of therapy.

In analyzing the 20 cases that died in spite of surgical intervention, we may immediately dismiss one that died several weeks after the operation from unrelated lobar pneumonia. The other 19 cases have been divided into two groups: In the first group, there were six deaths that could be ascribed to technical errors; and in the second, 13 that were caused either by the brain damage which accompanied the clot or by other complications which caused or contributed to the fatal outcome. Among these complications were pneumonia, myocardial failure and uremia.

The six fatalities in the first group follow: In one case which was explored through frontal bur-holes, the clot was not found. In a second instance, the patient had a compound fracture as well as an extradural hemorrhage, neither of which was diagnosed. Both of these cases were seen in 1932–1933, before we had developed our present standard of technic and therapy. The third case was one in which the diagnosis was made and bilateral exploration undertaken, but the exposure was inadequate and failed to demonstrate a large posteriorly-located clot. There were, also, two cases which developed postoperative meningeal infection. The final case in this group was one in which a recurrent extradural hemorrhage developed. Failure to recognize this complication early enough resulted in a fatality. It is important to be on the watch for recurrent extradural hematomata; on one occasion we had removed such a recurrence with ultimate recovery of the patient. The possibility of recurrent bleeding in extradurals has recently been emphasized by Ellis.[20]

The records of the remaining 13 fatal cases demonstrate that death was traceable to the concomitant brain damage (contusion, laceration, and intracortical clot), which was so severe as to make recovery improbable, or to some extraneous complication which of itself prevented recovery.

An analysis of these mortality statistics warrants the following conclusions: There will probably be about 25 per cent of the cases of extradural hematoma in which either the severity of the accompanying brain damage or the general physical condition of the patient will prevent recovery, even with the most timely and ideal treatment of the clot. In another 15 per cent, recovery would be probable if all possible advantages were taken of our present diagnostic and therapeutic methods. Thus, providing ideally proper care had been given, there would have been a reduction of our case mortality from 59 to 44 per cent.

SUMMARY AND CONCLUSIONS

A series of 44 cases of extradural hemorrhage, seen and treated on the Neurosurgical Service of the Boston City Hospital, between the years 1932–1939, has been reviewed.

A statistical study of the clinical, cerebrospinal fluid, and roentgenologic findings has been presented.

A standardized method of treatment has been outlined.

We believe that the so-called classical picture of extradural hematoma is so rare that it is the exception rather than the rule, in practice. This fact must be more widely recognized if the mortality from this condition is to be reduced.

In the past, the possibility of venous bleeding as a source of extradural clots has been overlooked. Sufficient evidence has now been collected to demonstrate its actuality and to prove its importance.

REFERENCES

[1] Hill, J.: Cases in Surgery, R. Baldwin, Edinburgh, 1772.
[2] Bell, Sir Charles: Surgical Observations, 466–467, Longman, Herst, Rees, Arnie and Brown, London, 1816.
[3] Cock, Edward: Guy's Hospital Rep., **7**, 157, 1842.
[4] Hewett, Sir Percy: Holmes' System of Surgery. 2nd Ed., **2**, 258, 1870 (New York).
[5] Callender, G. W.: The Anatomy of Brain Shocks. St. Bartholomew's Hosp. Rep., **3**, 415–444, 1867.
[6] Hutchinson, Jonathan: Lectures on Compression of the Brain. London Hosp. Rep., **4**, 10–55, 1867–1868.
[7] Erichson, John E.: Injuries of the Head. Lancet, **1**, 1, 1878.
[8] Gross, Samuel W.: An Examination of the Causes, Diagnosis, and Operative Treatment of Compression of the Brain as Met with in Army Practice. Am. Jour. Med. Sci., **66**, 40–74, 1873.
[9] Jacobson, W. H. A.: On Middle Meningeal Hemorrhage. Guy's Hosp. Rep., **43**, 147–308, 1885–1886.
[10] Pringle, J. H.: Traumatic Meningeal Hemorrhage; with a Review of 71 Cases. Edinburgh Med. Jour., n.s., **4**, 741, 1938.
[11] LeCount, E. R., and Apfelbach, C. W.: Pathologic Anatomy of Traumatic Fractures of the Cranial Bones and Concomitant Brain Injuries. J.A.M.A., **75**, 501, 1920.
[12] Moody, W. B.: Traumatic Fracture of the Cranial Bones; Clinical Consideration with Special Reference to Extradural Hemorrhage. J.A.M.A., **74**, 511, 1920.
[13] Wood Jones, Frederic: On the Grooves upon the Ossa Parietalia Commonly said to Be Caused by the Arteria Meningea Media. Jour. Anat. and Physiol., **46**, 228, 1911–1912.
[14] Wood Jones, Frederic: The Vascular Lesion in Some Cases of Middle Meningeal Hemorrhage. Lancet, **2**, 7–12, 1912.
[15] Verbrugghen, A.: Extradural Hemorrhage. Am. Jour. Surg., n.s., **37**, 275–290, 1937.
[16] McKenzie, K. G.: Extradural Hemorrhage. Brit. Jour. Surg., **26**, 346, 1938.
[17] Munro, Donald: Craniocerebral Injuries. Oxford University Press, New York, 165, 1938.
[18] Erickson, T. C.: Neurogenic Hyperthermia. Brain, **62**, 172, 1939.
[19] Ellis, F. F.: Repeated Extradural Hemorrhage. Med. Jour. Australia, **25**, No. 1, 262, 1938.