

# TRAUMATIC INTRACRANIAL HEMORRHAGE\*

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INTRACRANIAL hemorrhagic collections resulting from injury have been classified upon the basis of an anatomic location within the cranial cavity. The main groups include: (1) Epidural hemorrhage, produced by tearing of the middle meningeal vessels or dural sinuses, (2) subdural collections resulting mainly from disruption of cortical pial vessels, (3) subarachnoid hemorrhage produced by cerebral contusion and laceration and (4) intraparenchymatous hemorrhage due to intracerebral bleeding.

The type of hemorrhage may be correlated to some degree with the nature of the injuring force. When a direct blow or force strikes the non-moving or slower moving head, localized vascular injury is produced, the middle meningeal vessels may be torn, a venous sinus disrupted or a pial vessel ruptured as cerebral tissue is bruised and lacerated. The forces of indirect injury, when the head is decelerated as it rapidly moves against a slower moving or non-moving object, produce more complex, diffuse and combinations of vascular damage. The greater the velocity of the energy involved, the more extensive the pattern of vascular disruption.

The frequency of combinations of both vascular and parenchymatous damage must be emphasized since both the diagnosis and management are influenced by this circumstance. Thus, an extradural hemorrhage may co-exist with cerebral contusions, intracerebral petechial hemorrhages, subarachnoid and subdural bleeding. (Fig. 1.) However, one lesion is usually predominant and its clinical-surgical characteristics are sufficiently typical to warrant separate classification and discussion.

The following classification of vascular lesions is used in this analysis with a review of the pertinent findings in surgically treated and autopsied cases.

1. Epidural hemorrhage
2. Subdural hemorrhage
  - (a) Acute type
  - (b) Subacute chronic type
  - (c) Acute and chronic types in infants
3. Subarachnoid hemorrhage
4. Intraparenchymatous hemorrhage
  - (a) Petechial
  - (b) Massive
5. Subdural accumulation of spinal fluids

## EPIDURAL HEMORRHAGE

The most common type of extradural hemorrhage is of middle meningeal vessel origin. (Table 1.) Epidural hemorrhage from the sagittal sinus is occasionally seen. The latter may occur from depressed, comminuted fractures near the midline of the vault. In one of the cases studied an ice pick perforated the sinus, causing extensive extradural and intradural hemorrhage. A large clot collected between the two hemispheres in this instance and overlay the corpus callosum. Extradural hemorrhage of occipital emissary vein origin was noted in several cases of depressed fracture posterior and superior to the mastoid region. Occasionally the lateral sinus was involved, an occurrence associated with penetrating wounds.

Since extradural hemorrhage is usually of middle meningeal origin, the terms middle meningeal, epidural and extradural hemorrhage are used interchangeably. This type of collection is usually unilateral. Two cases of bilateral extradural clots have been

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seen in this series. One patient was operated upon and the other was seen at the post-mortem table.

**Etiology.** Falls, bicycle accidents and direct head blows are frequently the cause of extradural hemorrhage. In a series of 158

TABLE I  
THIRTY-FIVE OPERATED CASES OF MIDDLE MENINGEAL  
HEMORRHAGE

State of consciousness	
Lucid interval.....	13
Unconscious throughout.....	13
Drowsy and disoriented.....	8
Short unconsciousness followed by normal conscious state.....	1
Pupils	
Larger on same side.....	25
Larger on opposite side.....	1
Equal.....	9
Extraocular palsy	
Third.....	4
Fourth.....	1
Sixth.....	1
Focal signs	
Present.....	30
Not present.....	5
Vital functions	
Pulse.....	45-60
Respirations.....	18-26
Temperature.....	100-102
Spinal-fluid findings	
Pressure.....	45-650
Bloody.....	In 17 cases
Clear.....	In 1 case
Associated massive lesions	
Subdural hemorrhage.....	-
Temporo-sphenoidal-lobe clot.....	4
Subdural accumulation of spinal fluid.....	2
Recovered.....	26
Died.....	9

autopsied cases of cranial injuries, seven out of eleven fatal cases of epidural hemorrhage were the result of such low velocity, direct injuries. High velocity automobile injuries involving indirect forces appear to cause a smaller number of middle meningeal hemorrhages.

**Clinical Observations.** Although the classic "lucid interval" characteristic of the clinical syndrome of extradural hemorrhage may occur, it is the exception rather than the rule. An already unconscious state gradually deepening is commonly observed. A "lucid interval" may be absent due to co-existing pathologic damage within the cranial cavity. Occasionally hemorrhage from the meningeal vessels superimposed

upon a concussive state may be so rapid as to preclude a conscious period.

A frequent clinical finding is dilatation of the pupil on the side of the lesion. Extra-ocular palsies occur. The enlarging clot may compress the ocular nerve or nerves as they traverse the superior orbital fissure or the pressure may occur by the bulging inner aspect of the temporal lobe medially, involving and interrupting the nerve or nerves in their intracranial course from brain stem to the cavernous sinus. A dilated pupil without other signs of oculomotor paralysis may also be attributed to the above described etiology or to paralysis of the cortical pupillary constricting mechanism. In favor of a peripheral mechanism is the fact that when a clot is located at the base, pupillary manifestations are the rule. (Fig. 2.) When the clot covers the parietal and temporal areas, the pupils are likely to be equal in size. (Fig. 3.) A dilated pupil is usually on the side of the lesion. A constricted pupil on the side of the lesion was seen only once in this series. Paralysis of the third nerve was observed in four cases.

Increasing weakness and paralysis result from compression of the clot over the motor centers. The paralysis is usually on the opposite side from the clot. Occasionally it may be seen on the same side as the clot. In the untreated case the patient fails rapidly; the deep tendon reflexes become diminished, then lost; the plantar reflexes become unobtainable; the corneal response becomes imperceptible; generalized or Jacksonian convulsions may occur. The importance of constant observation cannot be overemphasized since these changes may follow one another in rapid succession with eventual fatal outcome.

**Roentgen Observations.** Survey roentgen studies of the skull may be of assistance in the diagnosis of an extradural hematoma. A fracture line crossing the temporoparietal region suggests this possibility. (Fig. 4.) Depressed temporal fractures may be associated with hemorrhage; five cases occurred in this series. Because of this association, patients with simple depressions, even if

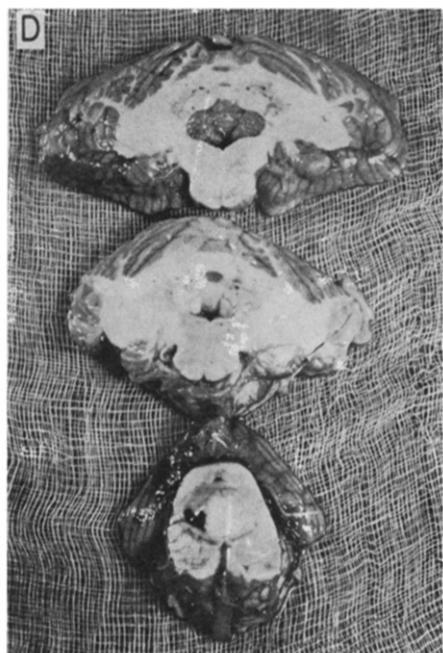
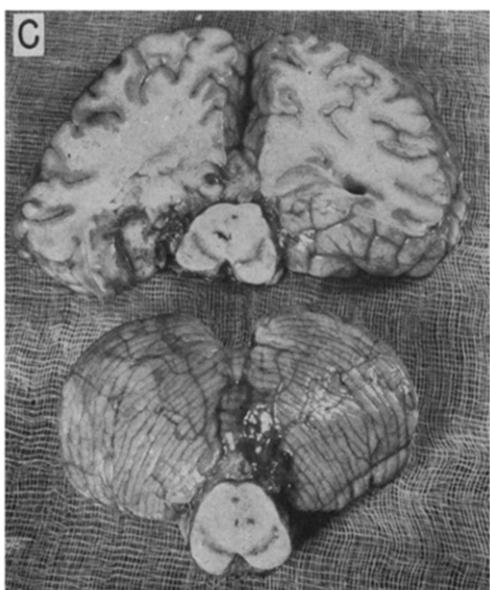
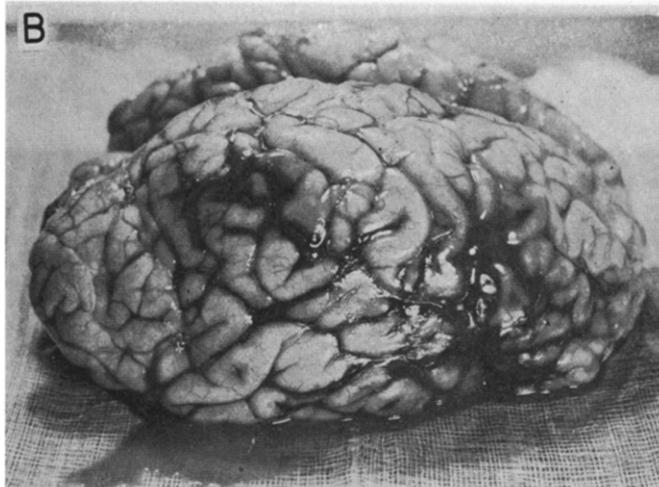


FIG. 1. Autopsy findings in a case with right epidural hematoma (A); subarachnoid hemorrhage (B); mid-brain hemorrhages and contusion of right occipital lobe (C); hemorrhages in the cerebellum and pons (D). That a combination of pathologic conditions co-exist is well known. However, usually a single pathologic entity predominates, producing fairly typical signs and symptoms.

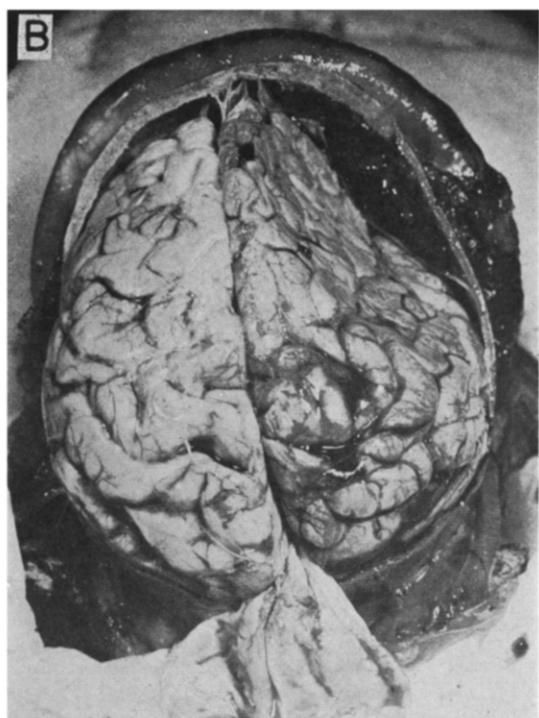
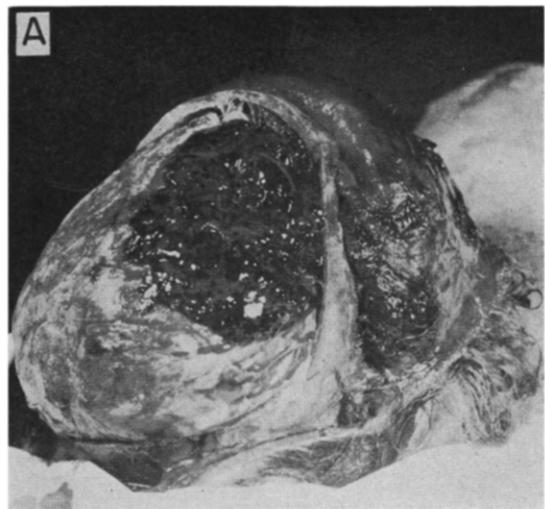


FIG. 2. Epidural hematoma over the convexity (A) causing marked compression of brain (B), dissecting its way into the middle fossa all the way to the region of the superior orbital fissure (C), and dissection showing the extent of the hemorrhage in the superior orbital fissure (D). This explains the usual involvement of the third nerve by compression in its course through the orbital fissure resulting in widely dilated pupil or ocular motor palsy.

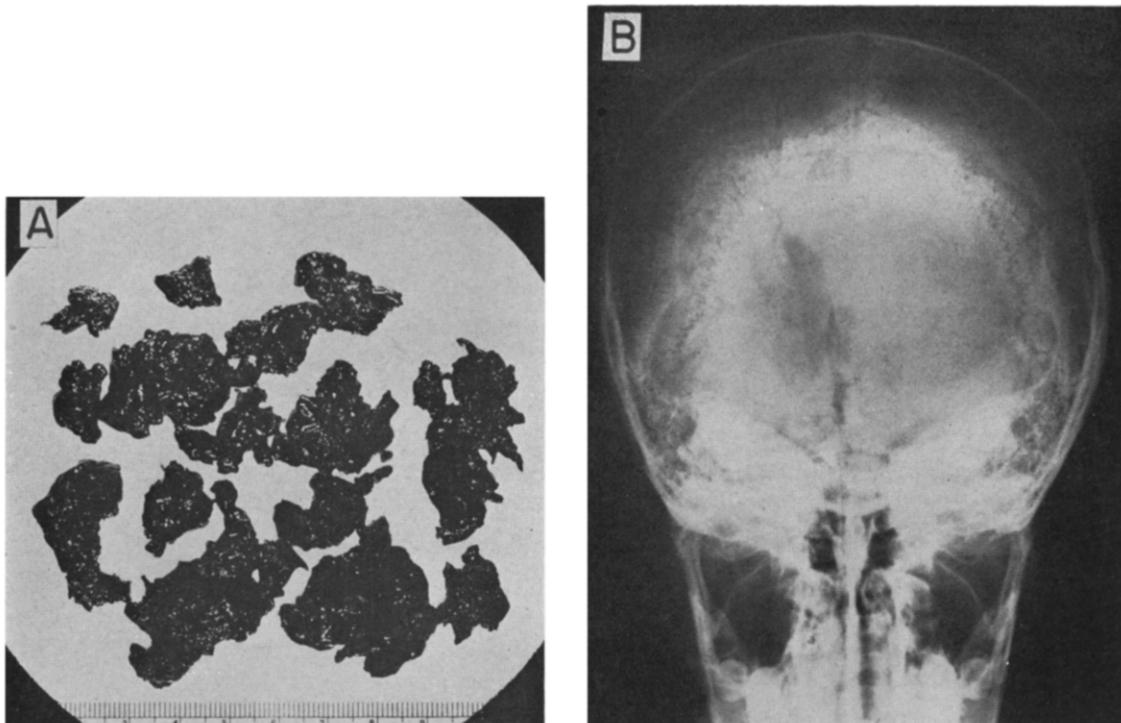


FIG. 3. A and B, encephalogram showing compression of right ventricle from epidural hematoma; hemorrhage only over the convexity with no inequality of pupils.

minor, deserve close observation, particularly if continued unconsciousness and focal signs exist. In two cases, demonstrated by postmortem examination, the fractures were of such degree that roentgen studies could not have revealed them. In one case of bilateral middle meningeal hemorrhage both temporal areas were explored because of the extensive fracture from one temple to the other. The patient presented bilateral neurologic signs. In patients with a pineal calcification, the presence of a shift may be diagnostic and may locate the lesion accurately. (Fig. 7.)

*Pathologic Observations.* The initial hemorrhage from the middle meningeal vessels may be instantaneous and rapid or delayed and slowly progressive. In progressive lesions, various venous channels of the dura and bone contribute as the separation of the dura from the bone proceeds. Accompanying subdural bleeding was noted in eleven out of thirty cases. In two patients there was an associated subdural accumulation of spinal fluid. In three instances, a

massive temporo-sphenoidal clot was found to accompany the extradural collection.

*Treatment.* In our experience the operation of choice is that of a subtemporal decompression on the side of the lesion. If the clot is beyond the area of the temporal bone, a small bone flap may be required. Bleeding points are controlled. The middle meningeal artery is ligated or occluded by silver clips or cautery. Deep hemorrhage at the base may require retraction of the temporal lobe for exposure of the foramen spinosum into which a plug of cotton or wood may be introduced. The dura is opened, if necessary, to remove a subdural or intraparenchymatous clot. Usually a gauze drain is used for hemostasis. Local or general anesthesia may be used. In our experience pentothal sodium has been dangerous.

#### ACUTE SUBDURAL HEMORRHAGE

Acute subdural hemorrhage usually results from a tear of pial vessels associated with contusion and laceration of cerebral

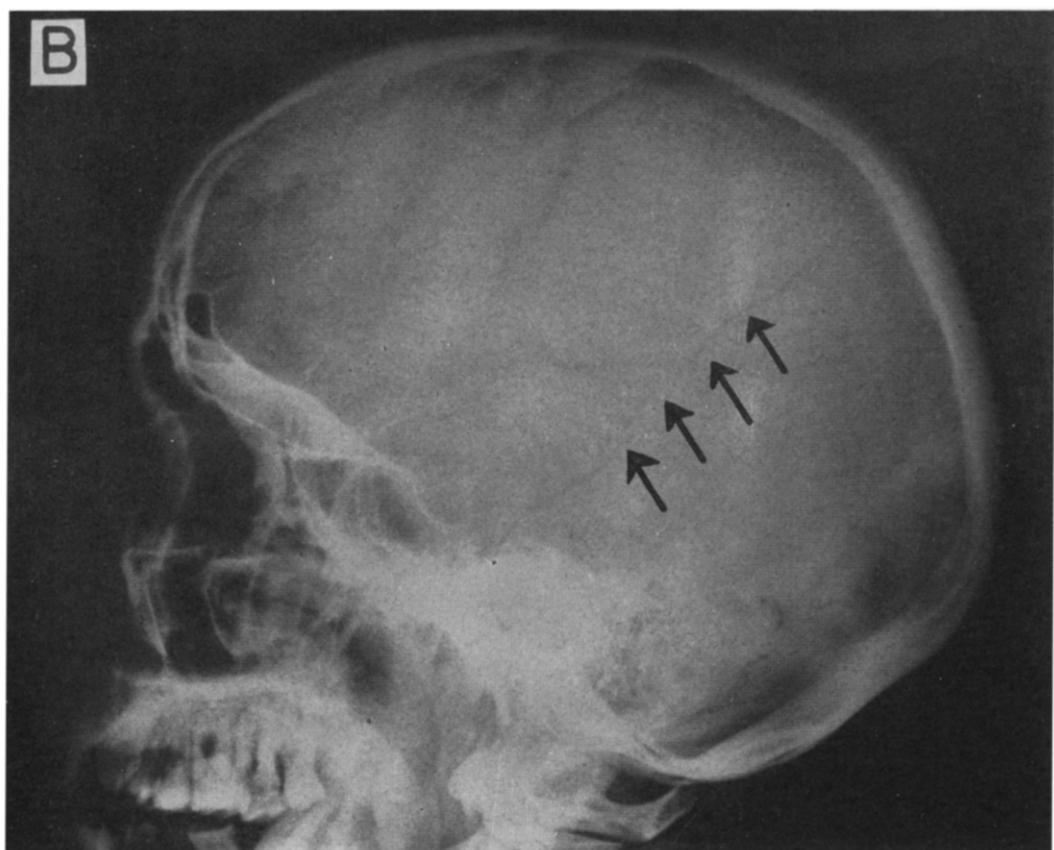
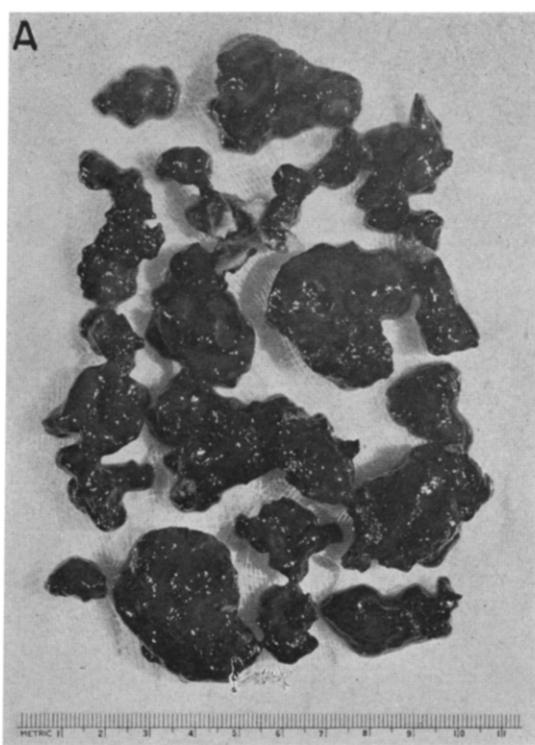


FIG. 4. A and B, epidural hematoma in a patient with a linear fracture of the left parietal bone extending into the middle fossa.

tissue. (Table II.) At times it is extensive, covering the surface of a hemisphere. Bilateral collections may be present. As a rule, the hematoma is located in the fronto-parieto-temporal region of one or both sides. Occasionally an unusual localization

TABLE II

## ACUTE SUBDURAL HEMORRHAGE

Lucid interval.....	8
Continued unconsciousness with disorientation.....	19
Dilated pupil.....	17
Hemiparesis or hemiplegia.....	20
Dilated pupil with contralateral paralysis or paresis.....	13
Convulsions (Jacksonian).....	6
Convulsions (generalized).....	4
Total cases.....	39
Deaths.....	15

of the clot is seen. (Fig. 5.) In two patients there was an extensive subdural hematoma between the two hemispheres.

The common fronto-parieto-temporal clot site can be explained on the basis of anatomic relations between brain and bone. The frontal and temporal lobes are snugly fitted into a bony encasement having projecting and irregular surfaces. In indirect blows when the rapidly moving head strikes a non-moving or slowly moving object, the intracranial contents by a mass movement abut against the irregular surfaces with resulting vascular disruption in this region.

*Clinical Observations.* The conscious state may manifest changes which correspond with those observed in patients with extradural hemorrhage. The "lucid interval" is common and is seen as frequently as in middle meningeal hemorrhage. The interval may vary from a few hours to several days or weeks. Since expansion of the subdural lesion occurs more slowly than the epidural, resulting physiologic events are usually less prompt in appearance. The longer the period of lucidity and freedom from major symptoms, the better the prognosis. Patients may remain semiconscious or comatose from the time of injury. Focal neurologic signs may be meager, those present being the result of increased intracranial pressure. Frequent examinations may reveal a gradually deepening stupor

with a dulling of responses to stimuli. A slowing of the pulse may occur. Occasionally the blood pressure rises with increasing stupor but as a rule it shows little or no change. Convulsions are common. Pupillary inequality and extraocular palsies occur. A dilated pupil, if present, is usually on the same side as the lesion. Unilateral extraocular muscle paralyses are also usually ipsilateral. There is most commonly an absence of papilledema. Increasing paresis of one-half of the body may be seen. This paresis or paralysis may be on the same side as the subdural collection, resulting in false localization. The spinal fluid pressure is usually elevated but surprisingly low pressures have been noted. The cerebrospinal fluid is often bloody but may be xanthochromic or clear. Severe bloody spinal fluid most frequently indicates diffuse intracerebral injury but exceptions to this conclusion are commonly encountered. Failure of a patient who has sustained a cranial injury to improve normally suggests the possibility of an existing complication which may well be a subdural collection. By means of pneumoencephalography or multiple burr holes the diagnosis may be made.

*Roentgen Observations.* A fracture of the skull may be present on the same or on the side opposite the hematoma with equal frequency. (Fig. 6.) A fracture is commonly absent. The presence of a fracture line does not locate the collection in cases of acute subdural hemorrhage. The presence of a shifted pineal shadow may be diagnostic. (Fig. 7.)

*Pathologic Observations.* As a rule, the disruption of pial vessels is the source of a subdural hemorrhage. Occasionally a massive intraparenchymatous clot may extrude through the cortex and seep into the subdural space. The latter is more common in non-traumatic cases. Contusion and laceration of the cerebral substance may accompany pial tears; deeper multiple small hemorrhages may be seen. These associated lesions complicate the syndrome of the subdural collection and serve as a cause for



FIG. 5. A and B, unusual localization of subdural hematoma. Air studies suggest a large mass between the two hemispheres which was a subdural hemorrhage from tear of connecting veins between cerebrum and superior sagittal sinus.

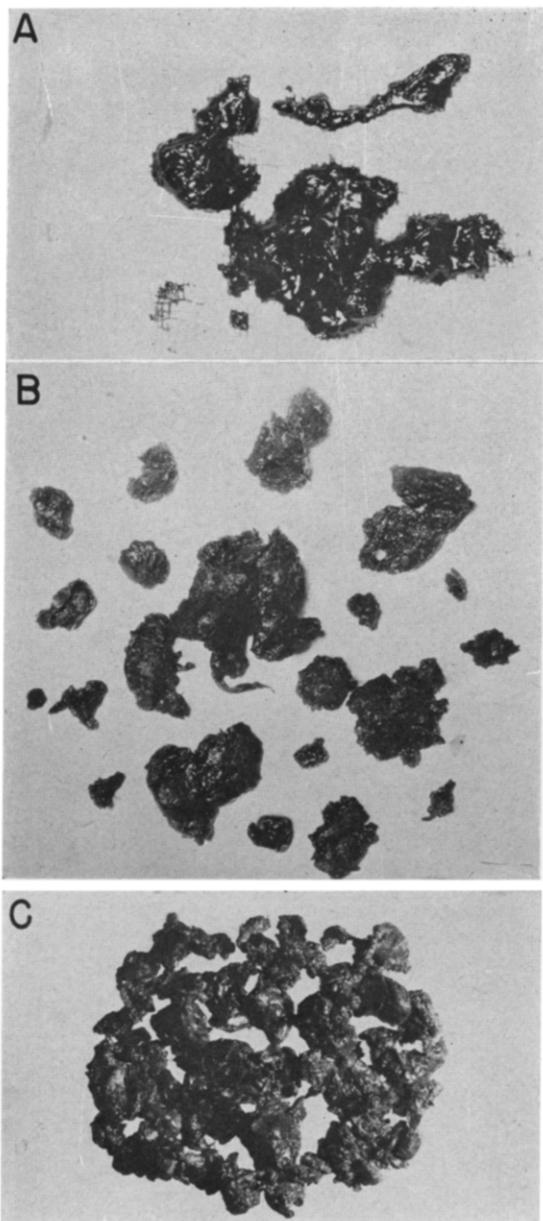


FIG. 6. Subdural hemorrhage (A) in a case with a fracture on the same side as the hemorrhage; (B) in a patient with a fracture on the opposite side; (C) in a patient with no fracture.

prolonged disability or death. A subdural hemorrhage may be of non-surgical proportions producing minor or no symptoms and signs. Upon the initial size and source of the hemorrhage depends the expanding nature of the mass with slow or rapid progression of symptoms and signs. A disruption of a large pial vein may produce practically instantaneous signs and symp-

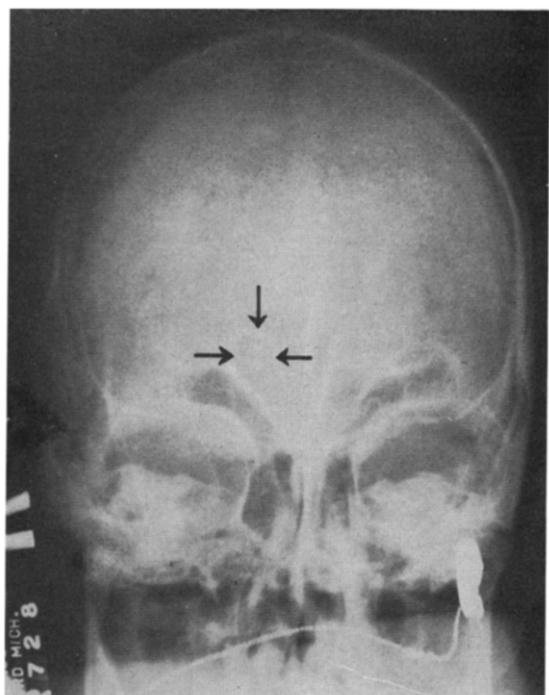


FIG. 7. Pineal shift in a patient with acute subdural hematoma.

toms. In the slowly expanding forms, the absorption of tissue fluids and cerebrospinal fluid by the collection may only gradually add volume and only insidiously result in a dynamic intracranial condition. This process becomes characterized by its chronicity and is so described by the term chronic subdural hematoma. In instances in which the localizing signs are on the wrong side there probably occurs compression of the brain stem on the opposite side from the hemorrhage against the tentorial border, compressing the pyramidal tract on the opposite side.

*Treatment.* The acute subdural hemorrhage presenting dynamic signs usually necessitates operative intervention. Both hemispheric surfaces should always be explored. A trephine opening at the frontoparietal junction 2 inches on either side of the midline uncovers the greatest majority of collections. If the two initial openings fail to reveal a hematoma, an air study may be employed in place of further random openings. If a subdural hemorrhage has been located, a subtemporal decompression

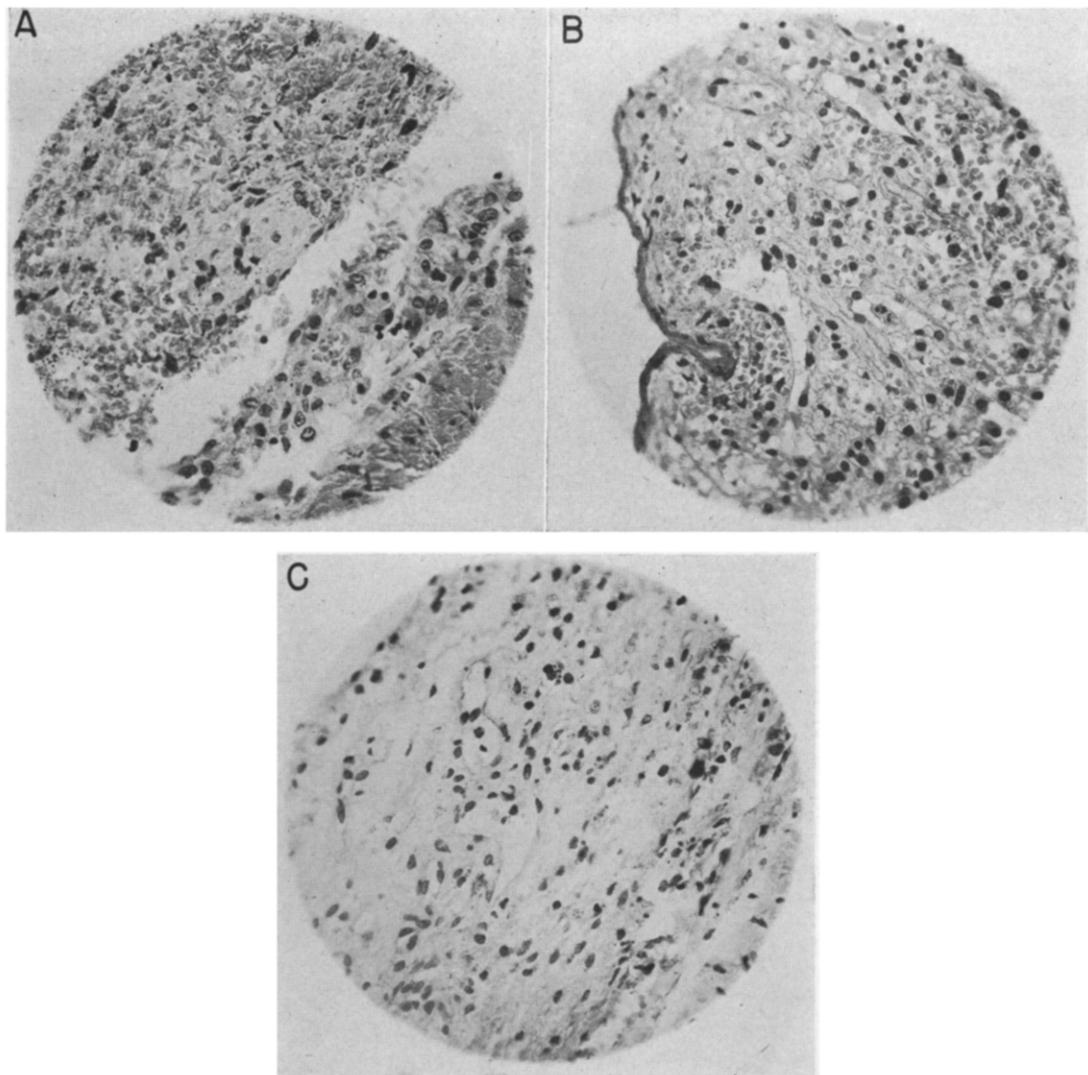


FIG. 8. Connective tissue proliferation in a patient with acute subdural hemorrhage eighteen hours after injury (A). Thick wall on the dural side of chronic subdural hematoma six weeks after injury (B). Thick wall on the dural side of chronic subdural hematoma several months after injury (C).

is then performed on the side of the lesion. The liquid and/or semi-solid clots are irrigated from the subdural space by means of saline and a brain spatula. The subdural space is drained from twelve to eighteen hours.

#### SUBACUTE AND CHRONIC SUBDURAL HEMATOMAS

When hemorrhage into the subdural space is small in amount or of quantity which is not sufficient to jeopardize the spacial relationships and requirements

within the cranial cavity, symptoms may be absent for weeks or months. By osmosis and the diffusion of cerebrospinal fluid and other tissue fluids into the collection (having a higher specific gravity), the subdural hematoma gradually expands. Additional volume is contributed by occasional hemorrhages into the clot from vessels in the proliferating granulation tissue on the dural side of the collection. (Fig. 8.) Ultimately the expansion results in cerebral symptoms. The prolonged, lucid, symptom-free interval can be explained upon these circumstances. (Table III.)

**Clinical Observations.** Chronic subdural hematoma may follow minor types of cranial injury. In some instances the trauma may be entirely forgotten by the patient and relatives. On the other hand, severe cerebral damage may be accom-

TABLE III  
SIXTY-FIVE OPERATED CASES OF CHRONIC SUBDURAL  
HEMATOMA (ADULT)

Headaches	
Present over the lesion.....	42
Not complained of, or no history.....	12
Generalized.....	11
Pupils	
Dilated on side of lesion.....	13
Equal.....	49
Dilated on side opposite lesion.....	3
Extraocular palsy	
Third.....	4
None.....	61
Fracture of skull	
Present.....	21
None.....	44
Side involved	
Left.....	32
Right.....	27
Bilateral.....	6
Spinal fluid findings	
Pressure above 300.....	40
200-300.....	12
100-200.....	5
Blood or xanthochromic.....	
Operation of choice:	
Bilateral, exploratory, trephine subdural temporal decompression on the side of lesion or osteoplastic flap if clot is solid	
Recovered.....	59
Died.....	6

panied and complicated by this condition. In such cases there may be fracture of the skull with disability from the onset of the injury.

If chronic subdural hematoma complicates a severe injury, the patient may remain unconscious or semi-conscious for varying periods from days to weeks, progressing from the unconsciousness caused by the initial severe cerebral damage into a semi-consciousness caused by the cerebral compression of the subdural hematoma. A number of patients in this series had associated severe, diffuse cerebral injury.

In the more typical case the patient may be normal following a cranial injury for varying periods. Then in a brief period of but several days headache, previously of mild order, becomes intense. Conduct and

personality defects appear and within a week the patient progresses into a stupor, and finally coma if recognition of this complication has not been made.

Pupillary inequality may occur with the enlarged pupil on the ipsilateral side. The fundi may exhibit papilledema although this is the exception rather than the rule. Visual field studies in those patients who are cooperative have proved of little help. Headache is a constant complaint and may be localized to the side of the lesion. An overlying hyperpathic zone of the scalp may be present. In some patients increasing drowsiness may be the only finding. As a general rule, focal neurologic signs obtain in the advanced cases. Bilateral focal signs and findings implicating the "wrong side" are common. This is the result of peduncular compression against the tentorium on the side opposite the lesion. The cerebrospinal fluid pressure is usually elevated. The fluid itself may be clear but is frequently xanthochromatic. The grossly clear fluid may contain an elevated total protein with minimal pleocytosis.

Changes in pulse, respiration, blood pressure and temperature are of little diagnostic significance until advanced cerebral compression has occurred. At times a bradycardia may be observed usually accompanied by headache. The electroencephalogram may be of important diagnostic assistance. (Figs. 9 and 11.)

**Roentgen Observations.** A fracture of the skull may or may not be present and its presence does not indicate the side of the hematoma. As a rule, the complication follows minor trauma to the head and thus a majority of patients show no fracture. Pneumoencephalograms are very helpful and patients with subdural hematomas tolerate this procedure surprisingly well. If the ventricles are visualized, there is a midline shift and cortical air markings on the affected side are characteristically absent. (Figs. 9 and 10.) A collection of air may show medial to the hematoma and this column may appear as a "pointer" within the cranial cavity. This finding is pathog-

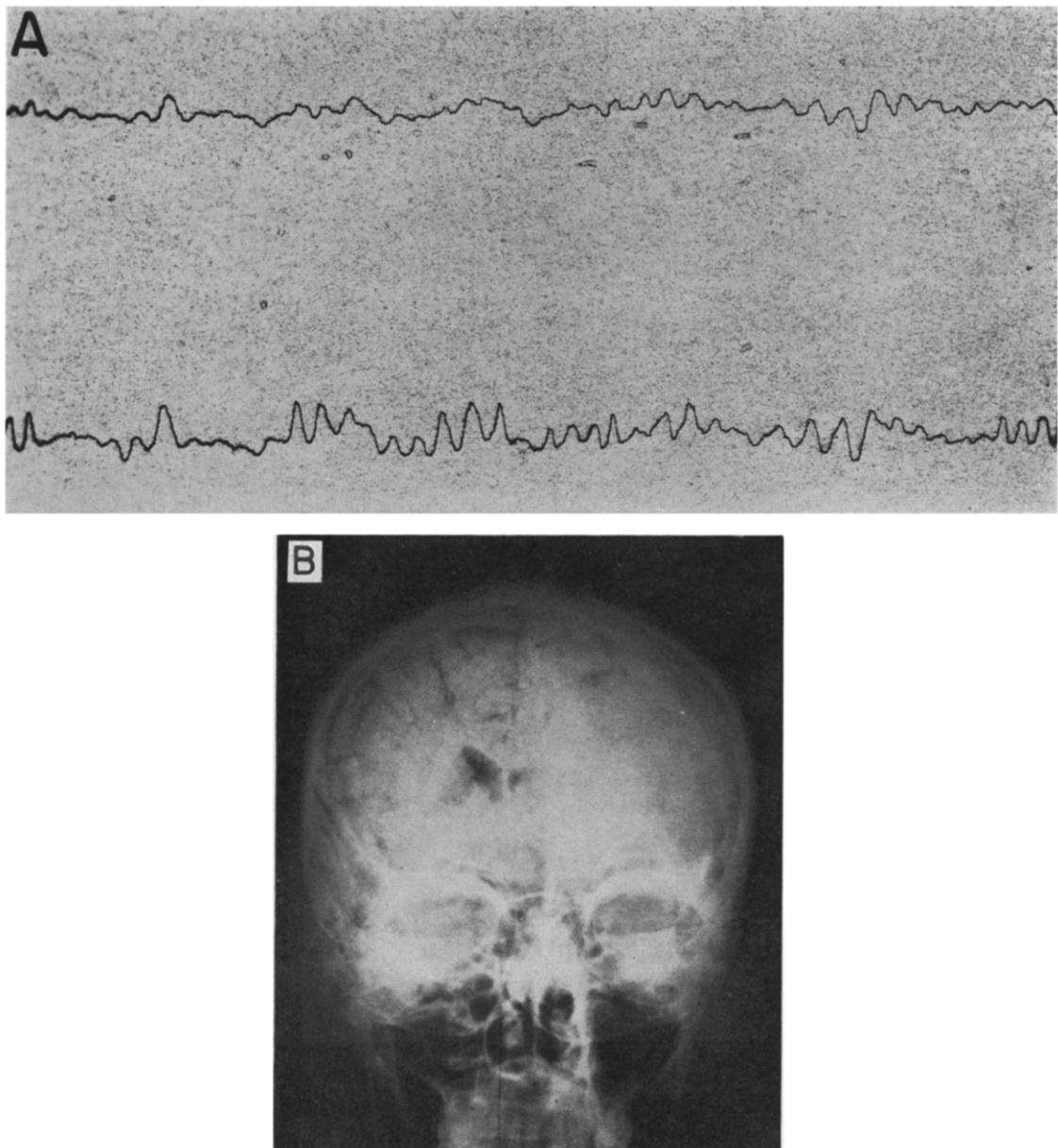


FIG. 9. A and B, air study and electroencephalogram in a patient with right subdural hematoma. Note absence of subarachnoid spaces on the affected side. Electroencephalographic record with disorganization of the electrical pattern on the affected side.

nomic of a subdural hematoma. Pineal shadow shift may be diagnostic of the location of the lesion. (Fig. 7.) Electroencephalographic studies may be diagnostic (Fig. 11), although in our experience correct localization of the lesion has been accomplished by this technic in about 40 per cent of the cases.

***Pathologic Observations.*** In the genesis of chronic subdural hematoma there is a

prompt cellular reaction with connective tissue proliferation on the dural side of the collection. (Fig. 8.) On the arachnoid side a limiting membrane forms. The subdural mass increases in size by the absorption of tissue fluids and spinal fluid through the process of osmosis and diffusion. The inner layer of the sac serves as a semi-permeable membrane. Thus an expanding process occurs (usually covering a wide area of the

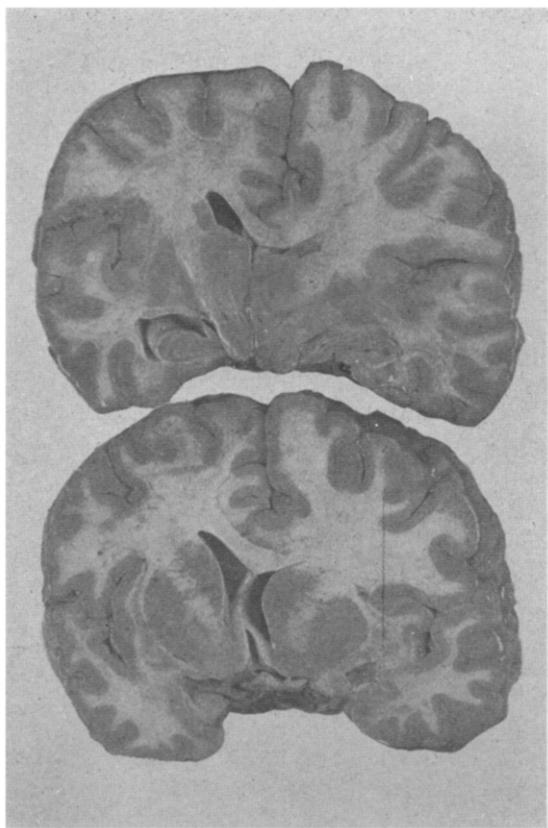


FIG. 10. Marked compression of the brain with ventricular distortion in a case of acute subdural hematoma.

cerebral surface, since the subdural space is not limited) rather than an absorption of the collection. Within the sac of the hematoma the blood may partially or completely hemolyze. It is not unusual, however, for the clot to remain semi-solid.

*Treatment.* Bilateral trepanation at the frontoparietal junction about 2 inches on either side of the midline followed by a subtemporal decompression on the side of the collection is the method of choice if the lesion is mostly fluid. A small osteoplastic flap is used if the clot is mostly solid. A single or two trephine openings may be adequate for the evacuation of unclotted hematomas when combined with irrigation. Changing the position of the head may also be of assistance. It is now generally admitted that the membrane of the hematoma sac need not be removed. After evacuation of the sac contents the ability of

this tissue to retain its characteristics of a semi-permeable membrane is lost.

#### SUBDURAL HEMATOMA (ACUTE AND CHRONIC) IN INFANTS

Newborn infants occasionally are afflicted with subdural hemorrhage. Birth injuries, injury by forceps, falls and other etiologic factors may be responsible. In acute subdural hemorrhage, the infant does not react normally to its environment, presenting often a lethargy and dulled response to stimuli. A weak, inconstant cry replaces the lusty ability of the normal infant. Cyanosis appears with weak, irregular respirations. High concentrations of oxygen may have little effect. Neurologic findings may be of a minor order. Occasionally spasticity is present on the contralateral side. Pupillary irregularities occur. Convulsions are common, being either localized or generalized. An important finding is a tenseness, fullness or bogginess of the anterior fontanel. Lumbar puncture usually shows a grossly bloody fluid under increased pressure.

The infant with a chronic type of subdural hemorrhage or hydroma has progressed satisfactorily in its early months until a parent or pediatrician notices enlargement of the head. In other patients, a convulsion first draws attention to an interruption of normal progress. In all other respects, the growth, sleep, feeding, vision, etc., may be normal. Head measurements indicate an enlargement above normal. A cracked pot sound may be noted on percussion of the head. The fontanel is wide and the sutures may be separated. Roentgen studies may show this same suture separation. Focal neurologic signs and fundus changes are usually absent.

The lesion may be accurately diagnosed by tap with a No. 18 gauge needle through the lateral aspect of the fontanel on both sides. When present, the collection may be evacuated through appropriate trephine openings on the affected side. Occasionally aspiration alone has been sufficient to drain the collection.

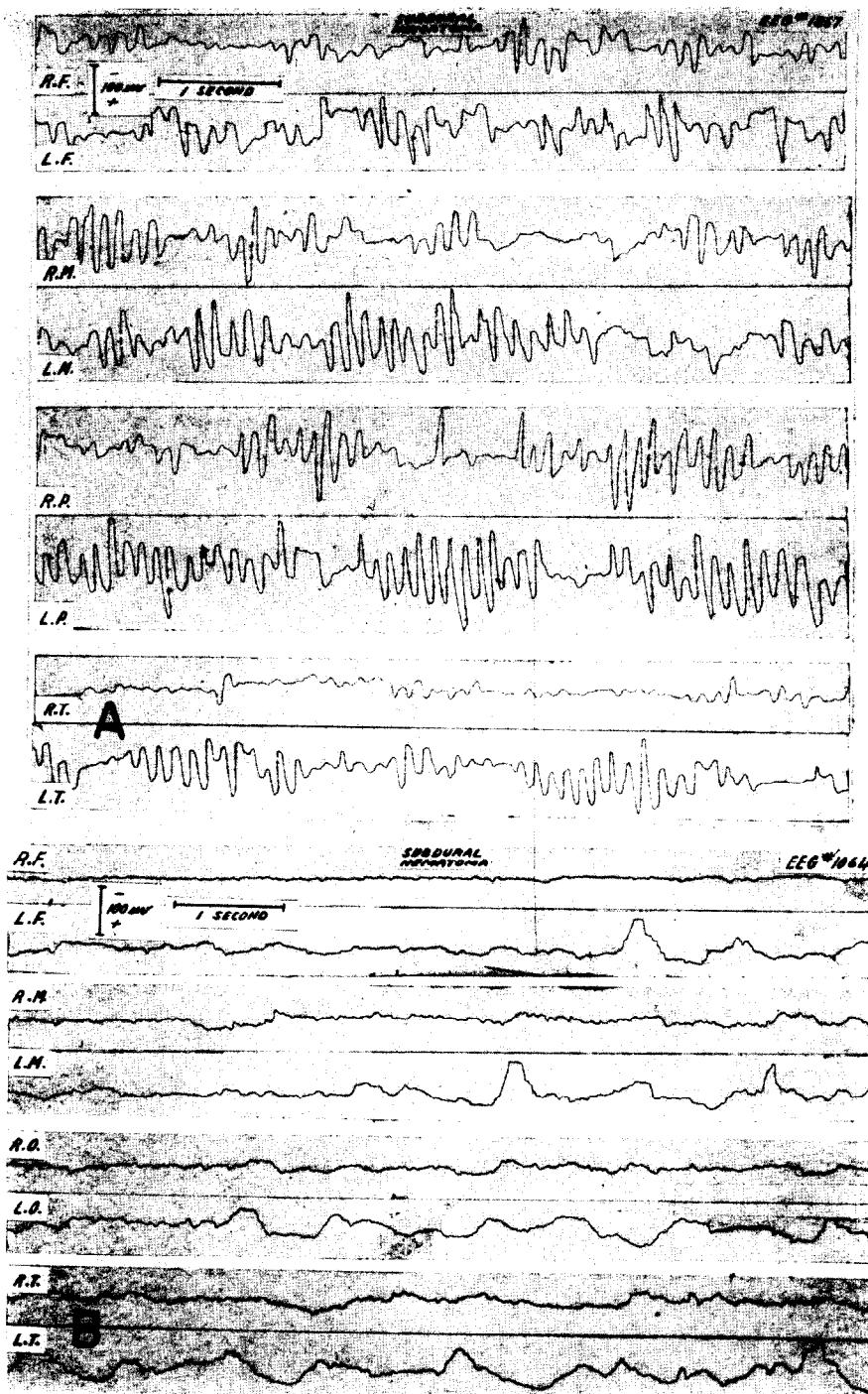


FIG. 11. Electroencephalographic records in two cases of subdural hematoma. In (A) there is disorganization of the electrical pattern over the right temporal region. In (B) there is delta activity over the left hemisphere.

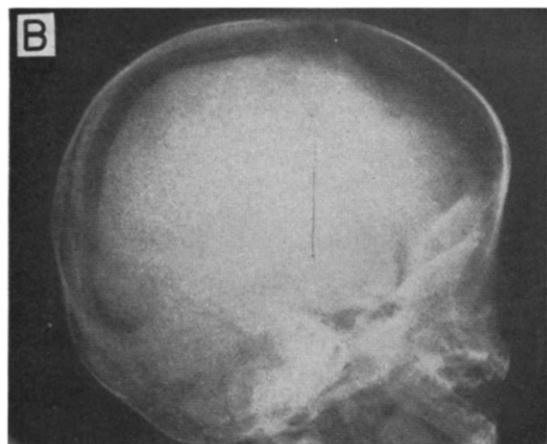
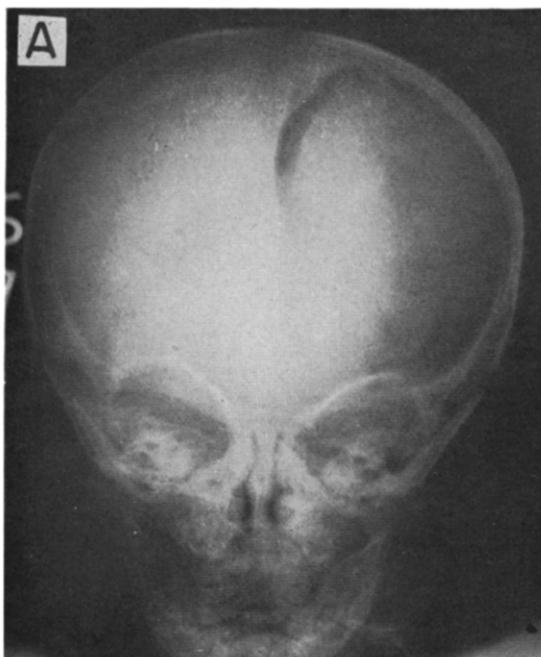


FIG. 12. A and B, subdural hematoma in a four months old infant visualized by injecting air after drainage of fluid contents. Note that the entire hemisphere was compressed by the fluid mass.

The chronic subdural collection is suitably managed by means of two fronto-parietal openings of an exploratory type. If a clot is encountered with a thick membrane, it may be necessary by means of a small bone flap to remove this layer in order to allow expansion of the depressed hemisphere. (Fig. 12.) Clots may be irrigated from the surface through two openings. A hydroma may be evacuated through a single opening.

#### SUBDURAL ACCUMULATION OF SPINAL FLUID

Consideration must be given to the occurrence of a subdural accumulation of

TABLE IV

SUBDURAL COLLECTION OF SPINAL FLUID

Lucid interval.....	3
Continued unconsciousness with disorientation.....	16
Dilated pupil.....	9
Hemiparesis or hemiplegia.....	8
Dilated pupil with contralateral paralysis or paresis.....	5
Convulsions (Jacksonian).....	2
Convulsions (generalized).....	2
Total Cases.....	20
Deaths.....	-

spinal fluid. (Table IV.) This curious condition is seen in less severe grades of head

injury. It is almost never diagnosed prior to operation. That there is a cause and effect relationship between this condition and clinical findings is proved by the rather remarkable results following treatment in some cases. Pathologically, the collection has been stated to be caused by a rent in the arachnoid with a resulting valve-like action which permits cerebrospinal fluid to enter the subdural space and not return. When a patient fails to improve after drainage of a collection, air studies may be necessary to rule out a co-existent disturbance. We have seen middle meningeal hemorrhage occur with this lesion. In some cases this abnormality may be seen without a history of cranial injury.

The treatment is that of simple drainage of the subdural collection through trephine openings. Not infrequently the collections are bilateral.

#### MASSIVE TYPE INTRAPARENCHYMATOUS HEMORRHAGE

Massive intraparenchymatous hemorrhage or intracerebral hemorrhage in acute head injury is uncommon in autopsy ma-

terial. The most vulnerable area seems to be in the region of the temporosphenoidal lobe. Clinically the same rarity of such cases is evident. In our series of over 200 operations for head injury with massive hemorrhage, there have been five instances of massive intracerebral clot; three occurred in the left temporosphenoidal lobe.

The clinical findings observed were similar in each instance to left temporosphenoidal clot. A progressive failure characterized by a right lower facial weakness, right upper limb paresis, eventual aphasia and stupor was noted. Because of the localizing signs, trephine with a left subtemporal decompression was performed and in each instance a massive clot from the temporosphenoidal lobe with an overlying subdural hemorrhage was evacuated. Much necrotic brain tissue was encountered which was also removed. (Fig. 13.)

In the diagnosis of such cases, localizing neurologic signs aided by air studies should accurately locate the lesion.

#### SUBARACHNOID HEMORRHAGE

Subarachnoid hemorrhage is the most common variety of traumatic intracranial hemorrhage. It may co-exist with epidural, subdural and intraparenchymatous hemorrhage. The associated subarachnoid hemorrhage is usually brought about by laceration and contusions of brain surfaces.

TABLE V\*

Dilated pupil.....	11
Hemiparesis or hemiplegia.....	15
Lucid interval.....	7
Continued unconsciousness or disorientation.....	26
Dilated pupil with contralateral paralysis.....	10
Convulsions (Jacksonian).....	4
Convulsions (generalized).....	3
Total cases.....	34
Deaths.....	16

\* In thirty-four cases the above signs and symptoms led to exploration, but the latter revealed no massive clots. Usually edema of the brain, contusions and subarachnoid hemorrhage were seen.

At times it is extensive enough to cover one or both hemispheres. The diagnosis is confirmed by a lumbar puncture. The cere-

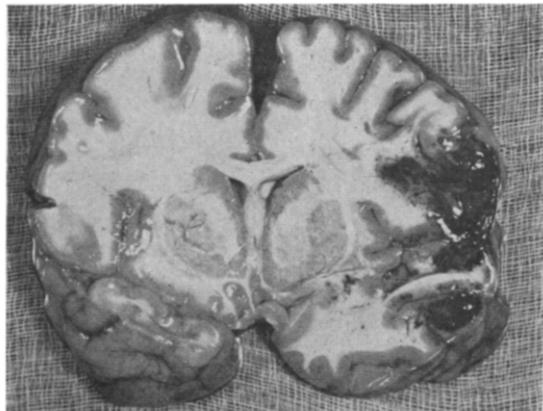


FIG. 13. Extensive contusion of brain in right parietal region. Such a lesion may eventually result in softening and hemorrhages causing an intraparenchymatous mass lesion. Traumatic intraparenchymatous hemorrhage is almost always associated with softening and necrosis of brain tissue resulting in severe cerebral deficit.

brospinal fluid obtained from the spine may contain much less blood than that obtained from the cisterna magna or the ventricles. Localized subarachnoid hemorrhage with edema may cause focal signs of a progressive nature so as to resemble closely the picture of subdural hematoma or massive intracranial hemorrhage. The presence of blood in the spinal fluid should not deter operative intervention, for in cases of epidural and subdural hemorrhage there is frequently associated subarachnoid hemorrhage. The patient with subarachnoid hemorrhage usually gradually improves, the conscious or semi-conscious state lightens and focal signs resolve. Conservative supportive treatment is thus wisely continued in the presence of gradual improvement.

#### PETECHIAL HEMORRHAGE

Brief mention of petechial hemorrhages is made to complete the subject. How often they occur in those with severe cranial injury is difficult to establish, but 34 per cent of cases seen at the autopsy table show these small hemorrhages throughout the cerebral tissue. The association of petechial hemorrhages with other forms of operative

conditions makes for longer convalescence and less complete recovery due to sequelae of a permanent nature.

#### COMMENT AND SUMMARY

A similarity of symptoms and signs is evident in the discussion of the various types of acute traumatic intracranial hemorrhagic collections. The state of consciousness and its alterations are significantly important. An increasing stupor, abolition of the conscious state after a lucid interval are indications for possible operative intervention. The neurologic status of the patient should be studied frequently. The progress of neurologic findings may be suggestive of an enlarging mass lesion. On repeated examinations, if the patient shows increasing weakness of one-half of the body, eventuating in paralysis, he may be afflicted with an enlarging blood clot. On the other hand, if a paresis or paralysis has been found to exist soon after injury, its causation by contusions and bruises of cerebral tissue is much more likely and, therefore, operative intervention may not be considered in such a case. The presence of inequality of pupils may be significant. The dilated pupil is usually on the side of the lesion. Papilledema may be present although this is rare. The vital functions may show alterations from normal. A slowing pulse rate and a lowering respira-

tory rate may accompany mass lesions. An increase in blood pressure, although rare, may signify increasing intracranial tension.

In acute hemorrhagic collection the one single important symptom justifying exploration is increasing drowsiness or stupor. Pupillary inequalities, changes in vital functions and focal neurologic signs may corroborate the presence of an enlarging clot. However, a dilated pupil, a low pulse rate or hemiplegia in a perfectly conscious patient does not justify operative intervention.

In subacute and chronic subdural hematomas the presence of unilateral headache and hyperpathia of the scalp over the pathologic lesion are important. The association of severe brain injury with subdural hematoma produces long disability and the recognition of a complicating subdural hematoma in long continued unconsciousness is important.

Post-traumatic epilepsy is rare in epidural and chronic subdural hematomas. In acute subdural hematomas it is seen in almost a third of the patients. This favors the assumption that in these patients the subdural hematoma is associated with contusions and bruises of the cerebral surface eventuating in cerebrodural cicatrization. Headaches, dizzy spells and personality changes are surprisingly uncommon as sequelae in postoperative mass lesions.

