THE DIAGNOSIS OF TRAUMATIC INTRACRANIAL HEMORRHAGE BY ANGIOGRAPHY*

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(Received for publication November 27, 1950)

The diagnosis of massive intracranial bleeding in the seriously injured patient is sometimes difficult, and exploratory multiple trephine openings and diagnostic air studies may be dangerous and inconclusive. Since patients in this group may have diffuse brain damage with contusions and non-surgical hemorrhagic collections, accurate localization of the lesion and estimation of its dimensions have importance. Such a diagnostic procedure may be angiography. Although its use is not without danger, a successful angiogram accurately may locate a massive lesion and also differentiate between subdural, epidural and intracerebral hematomas.

The patients included in this study were from the neurosurgical services of the Detroit Receiving and Grace Hospitals over a period of 6 months (January to June 1950). Approximately 350 head injury patients are admitted during this period. Of these, 45 per cent or 160 have major cranial injuries consisting of fracture of the skull and brain contusions and laceration with or without fracture.

Angiography was employed in those patients with major cranial injuries who either did not improve under conservative management or who presented evidence of a "dynamic syndrome" of increasing stupor, new focal signs, and cardiac, thermal, or respiratory evidence of progressive cerebral dysfunction. Thirty arteriographic studies were made.

Under local or pentothal anesthesia, a percutaneous angiogram was obtained by the injection of 35 per cent diodrast and satisfactory anteroposterior and lateral studies were made. Although to begin with, local anesthesia was used, most of the patients required additional pentothal administration. A routine survey set of roentgenograms was obtained at the same time. If a surgical lesion was demonstrated, the patient was removed to the operating room for treatment. All of the patients in groups A, B, C, G, and H were operated upon. In group F, all but 2 were trephined (Table 1).

RESULTS

Table 1 summarizes the results of the angiographic studies of 30 patients. The mortality is also included. There were 9 subdural hematomas, 2 epidural hematomas, 4 temporal lobe intracerebral collections and 1 "extravasation" lesion, 2 aneurisms, and 10 cases in which there was no evidence of a complicating surgical lesion. It is of note that there was 1 false negative and

* Aided by the Kresge fund.
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TABLE 1
Summary of results
(30 cases)

<table>
<thead>
<tr>
<th></th>
<th>No.</th>
<th>No.</th>
<th>Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Subdural Hematoma</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Acute</td>
<td>4</td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>2. Subacute</td>
<td>3</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>3. Chronic</td>
<td>2</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>B. Epidural Hematoma</td>
<td>2</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>C. Intracerebral Hematoma</td>
<td>4</td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>Etiology</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Traumatic</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Primary vascular disease</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>D. Aneurisms</td>
<td>2</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>E. Extravasation Phenomenon</td>
<td>1</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>F. Brain Contusion with Normal Angiograms</td>
<td>10</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>G. False Normal Angiogram</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>H. False Positive Angiogram</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severe diffuse temporal lobe contusion and laceration</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>30</td>
<td>11</td>
<td>36.6%</td>
</tr>
<tr>
<td>Total Mortality</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Fig. 1.* R.M., 23-year-old male, beaten in fight 10 days before operation. Was not unconscious, but became progressively stuporous. Had right frontal headache, vomiting, dizziness and diplopia. Admitted day before operation in semicomatose state. Had bruise about right eye with subconjunctival hemorrhage, left 3rd nerve palsy, ptosis of lid, fixed pupil, and bilateral Babinski reflexes. Roentgenograms normal. Collection outlined at periphery of brain. Anterior cerebral artery shifted. Liquid and solid subdural hematoma (150 cc.) evacuated. Lived.

* Illustrations have been retouched for clarity.
false positive angiogram. The former occurred in a profoundly injured patient who was found to have a bilateral subdural hydroma by burr openings. The latter occurred in a patient with severe damage to the temporal lobe (Fig. 8).

The patterns of typical subdural hematoma studies are shown in Figs. 1, 2 and 3. A marked shift of anterior cerebral to the opposite side is noted. The middle cerebral may be depressed. If the finer radicals of the middle cerebral are also visualized, the space occupied by the hematoma may be evaluated by the distance between the skull and cerebral surface vessels.

![Fig. 2. W.D., 49-year-old male, fell in bathtub 13 days before admission, striking head. Was not unconscious. Nine days later lethargy developed with a short period of unconsciousness. At admission he was semiconscious. Right pupil was slightly larger than left. There was left facial weakness. Angiogram revealed anterior cerebral artery displaced to left with depression of middle cerebral, indicating a subdural hematoma on the right side. Burr holes evacuated a large (150 cc. liquid) right frontotemporal subdural hematoma. Lived.](image)

The pattern of the intracerebral hematoma of the temporal lobe is shown in Fig. 4. The anterior cerebral is shifted to the opposite side in part of its extent. In the lateral views, the Sylvian vessels are dislocated upward and forward.

In one of these patients, J. D., the fact that the angiogram pre-operatively localized a temporal lobe lesion accounted for his survival. Routine burr openings most probably would have revealed only the associated subdural extravasation. His critical state may then have been wrongly ascribed to non-surgical type of brain damage. This patient’s findings were as follows:
J.D., a 50-year-old white male, was admitted on Feb. 6, 1950, having fallen and
struck his head on a curb. He was uncooperative and confused, with evidence of
alcoholism. A left Babinski reflex was the only abnormal neurologic sign. B.P. was
140/80. Roentgenograms of the skull showed a left occipital linear fracture measur-
ing 3 inches. A calcified pineal gland was in normal position. An EEG at admission
showed a severe right frontotemporal disturbance with a delta focus of 1.5–3/sec.
waves. Spinal puncture yielded bloody CSF under normal pressure. The patient

![Diagram](image)

Fig. 3. R.E., 32-year-old male, beaten in
fight 2 months prior to trephine. Progressive
disorientation with papilledema; roentgeno-
grams normal; CSF clear. Arteriogram showed
shift of anterior cerebral artery to the right with
depression of left middle cerebral artery (note
normal pattern of right side for comparison),
typical of frontotemporal collection. Liquid
hematoma (300 cc.) evacuated. Death.

improved although he remained confused. An EEG on Feb. 10, 1950 showed "im-
provement of the basic rhythm with the delta focus not so pronounced. Paroxysmal
activity resembling psychomotor patterns are present in the right temporal area."
Two days later, on Feb. 12, 1950, the patient became apathetic, then stuporous.
His neurologic status rapidly changed, resulting in a dilatation of the right pupil
and a left hemiparesis, with return of pyramidal tract signs. The fundi remained
normal.

An angiogram was done, showing the pattern presented in Fig. 4. Operation fol-
lowed. Exploratory burr openings were converted into use for a small temporal
craniotomy flap, only when a pancake type of subdural hematoma was found cover-
ing the frontotemporal area. Incision of the temporal dura resulted in extrusion of an
intracerebral hematoma, measuring about 100 cc.

The patient improved and was discharged on Feb. 23, 1950, without neurologic
deficit. A final EEG on Feb. 20, 1950 showed "disappearance of the delta activity."
Fig. 4. E.B. Pattern of intracerebral hematoma similar to J.D. (see case report). Shift of anterior cerebral to left with elevation of Sylvian vessels. Evacuation of right temporal intracerebral collection by small bone flap. Considered a subdural suspect. A small subdural collection was present and the massive intracerebral clot evacuated might have been overlooked without angiogram.

Fig. 5. C.W., 47-year-old male, struck on head 9 days before operation. Was briefly unconscious. Severe right frontal headache developed. Roentgenograms showed linear fracture in right parietotemporal area. Shortly after admission had dilated right pupil with ptosis and slight weakness of left arm. Angiogram showed upward displacement of middle cerebral and shift of anterior cerebral. Temporal epidural collection of 100 cc. of solid blood evacuated. Lived.
Examples of the findings in *epidural hematoma* are shown in Figs. 5 and 6. In Fig. 5 the shifted middle cerebral vessel suggests a temporal mass. In temporal lobe hematoma, the branches of the middle cerebral are more likely to be locally deformed in a direction upward and anteriorly. In Fig. 6 there is marked separation of the anterior and middle cerebral group of vessels. An intracerebral frontal lobe mass could also result in such a finding. In the case illustrated, there was a large frontal extradural hematoma.

Two patients were admitted in a semiconscious state attributed to head injuries, having fallen as the result of *aneurismal rupture* with subarachnoid hemorrhage. Both were considered subdural hematoma suspects, but angiograms revealed the correct diagnosis (Fig. 7).

Of interest also has been the occurrence of an "*extravasation phenomenon,*" probably indicating cerebral contusion. A circumscribed area of diodrast extravasation was noted in 1 patient, unassociated with a subdural collection. This patient recovered. He had evidence of diffuse brain injury. This is an unusual finding in the diffusely damaged brain, even with fatal outcome, since the vascular pattern in the angiogram is usually normal.

In 2 instances, air studies gave false positive results (Fig. 8), while the angiograms were normal. Trephine of these patients confirmed the angiographic studies.
Fig. 7. V.J., 40-year-old female. On admission was conscious, confused, disoriented, aphasic, with right hemiparesis. Roentgenograms showed suggestive fracture of right squamosal suture and slight displacement of pineal to right. CSF xanthochromic. Considered as subdural hematoma suspect until angiograms revealed an aneurism, following air study. Lived.

Fig. 8. S.B., 57-year-old male, was struck by a car. Left occipital fracture, progressive deepening stupor with dilated right pupil and paresis of left lower extremity. Shift of anterior cerebral artery with upward displacement of middle cerebral artery. Death after exploration. Diffuse damage to temporal lobe with epidural, subdural and intracerebral collections not of surgical proportion. Classified as false positive angiogram.
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DISCUSSION

Many of the patients suspected of subdural hematoma were trephined in spite of normal vascular patterns in the angiogram. In but 1 instance, in which a hydroma occurred, did the angiogram fail to reveal a surgical type of lesion, if present. Autopsy studies also verified this accuracy. An important exception must be made with a subdural or epidural collection in the parietal or occipital area or, of course, in the posterior fossa. Such a lesion is uncommon and was not encountered in this group of patients. If doubt exists that such a lesion is present, posterior exploratory openings must be made with or without an air study.

The arteriogram is valuable in distinguishing temporal lobe intracerebral hematoma. Pre-operative knowledge that a massive collection avoids misjudging the significance of a coincidental subdural collection. A small temporal flap can be planned and the hematoma evacuated.

It is of note that the pattern for the epidural hemorrhage in temporal region is similar to that of the temporal lobe hematoma, but with the former, the trunk of the middle cerebral is more apt to be dislocated upward; whereas, with a temporal hematoma, a dislocation of the middle cerebral branches upward and forward is usually the case. The presence of a temporal skull fracture also may be evidence in favor of the epidural collection. Often the classical clinical syndrome clearly distinguishing middle meningeal hemorrhage is absent; consequently, a diagnostic procedure accurately delineating the lesion may be useful.

The shifted anterior cerebral artery indicates the presence and side of the hematoma. In large unilateral subdural hematomas, the entire vessel is shifted to the opposite side. The depressed or elevated Sylvian vessels may distinguish the subdural from an epidural or intracerebral clot. A surface collection may be outlined by the vascular compression—its size well estimated.

Unfortunately, there is a disadvantage in moving the patient for the roentgen studies. There is a further additional risk when pentothal anesthesia is necessary for the uncooperative patient. The time consumed in doing the arteriogram is a consideration. The question must be raised whether these risks are warranted in the interest of prompt and accurate diagnosis. The conventional use of multiple exploratory Burr openings under local or general anesthesia with or without the injection of air and ventriculography also have disadvantages and may lead to inaccurate localization. Arteriography appears to be useful and more experience will give it its proper place among the diagnostic procedures in the better management of the seriously ill patients following head trauma.

SUMMARY

1. Angiographic studies were made in a group of 30 seriously ill patients with head injury, suspected of massive intracranial hemorrhage.
2. Typical vascular patterns were found to distinguish the subdural, epidural and intracerebral hematomas.

3. Angiography was found to be reliable. The procedure is superior to the more complicated and possibly inaccurate "exploratory trephine."

4. There are several risks involved, chiefly related to anesthesia in head injury patients.

REFERENCE