A Study of Some Factors Modifying Response of Cerebral Tissue to Subdural Hematomata*

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The divergent clinical paths pursued by patients harboring subdural hematoma suggest that there are factors, other than the magnitude of the original violence, capable of altering the degree, direction and quality of this clinical response. The present study was undertaken to assess the influence of a few simple parameters, such as volume of subdural extravasation and age, upon the resultant clinical behavior and prognosis. The 80 cases of substantiated subdural hematoma (volume of 15 cc. or more) constituting this report were derived from the autopsy records of the neuropathology laboratory, Kings County Hospital Center, and represent all cases of subdural bleeding in patients over 25 years of age during the period 1954-1962 (Table 1). The interval of time between injury and death, or commencement of symptoms and death, was very variable, ranging from 2 days to 6 months. Cases in which coexistent cerebral contusion, laceration or intracerebral hematoma of an appreciable extent occurred were not considered because it was felt not possible to divorce the effects of extracerebral from the intracerebral bleeding. Instances of subdural bleeding associated with blood dyscrasias or local neoplasia also were excluded.

The existence of subdural hemorrhage compressing the cerebral hemisphere often provokes certain immediate and delayed responses within the subjacent tissues. One of the early effects of acute hemispheric compression, in many instances, appears to be an ipsilateral swelling of the white matter.2,3 The hemispheric swelling has been attributed to an impedance of venous drainage caused by the pressure of the extracerebral mass.1 It seems probable that the expanded cerebral hemisphere itself may contribute further to the impaired hemodynamics of venous return. Ishii and associates,4 in a study of cerebral changes induced experimentally by supratentorial cerebral compression, observed that ipsilateral cerebral swelling, particularly of white matter, was a common finding. Histologic analysis of the affected white matter in their experimental animals showed interstitial and perivascular accumulations of fluid, especially in the centrum semiovale, distention, and degeneration of endothelial cells of the capillaries and veins, and some loss of myelin. In autopsy studies of patients with prolonged periods of survival, the vessels of the elaborate transcerebral venous system, described by Kaplan,5 frequently are dilated, congested, and occasionally the site of focal, acute phlebitis. Acute perivenular demyelination within the cerebral white matter also has been demonstrated, accompanied by a corresponding, reactive phagocytosis.

Herniations of cerebral tissue accompany the extrinsic hemispheric compression and the cerebral swelling. The herniating mesial temporal lobe compresses the posterior cerebral artery against the free edge of the tentorium. The effectiveness of this extrinsic compression seems to bear little relationship to the degree of antecedent vascular sclerosis of the artery. Acute, often

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hemorrhagic, infarction occurs in the temporal and occipital lobes serviced by the affected posterior cerebral arteries in an appreciable fraction of fatal cases of subdural hemorrhage (17.5 per cent). Less commonly, branches of the middle cerebral artery appear to be compressed by the reactive cerebral swelling; a resultant acute encephalomalacia of the lateral frontal or parietal lobes ensues.

After a variable period of time following the initial extracerebral hemorrhage, the level of consciousness may become depressed suddenly and the pupils dilated paralytically. The patient may assume an attitude of decerebrate rigidity. The emergence of these serious clinical signs usually portends the death of the patient and reflects the development of fresh, secondary hemorrhages or less frequently infarcts, within the midbrain and upper pons. This secondary, and usually fatal, rostral brain-stem damage is believed to be caused by mechanically induced alterations of the local blood vessels concomitant with the lateral compression and the caudal displacement of the rostral brain stem by the herniated hippocampus. Those very rare individuals who survive the immediate effects of secondary brain-stem damage generally remain in a vegetative state with persisting decerebration and usually die of some intercurrent infection. Autopsy in such individuals shows old hemorrhagic or ischemic foci within the midbrain and pons. The inciting swelling of the supratentorial tissue may no longer be present in such cases of extended survival.

**Influence of Hematoma Volume**

In determining the possible influence of volume of subdural bleeding upon the subsequent clinical course, only patients examined at autopsy were evaluated. The majority (90 per cent) did not have surgical treatment. In the few who did, the volume of hemorrhage was measured at the time of intervention. In these latter patients, death ensued generally within a few days of operation. A positive correlation between the volume of subdural hemorrhage and the percentage of patients showing neurological symptoms is evident (Table 2). Only a small fraction of individuals with subdural bleeding measuring less than 25 cc. showed any clinical alterations possibly referable to such extracerebral hematomata. In patients harboring hematomas measured to be 26-50 cc., about one-half showed pertinent symptoms commencing after the traumatic episode. In cases in which the subdural hematoma

**TABLE 1**

*Volumes of subdural hemorrhage in relation to age in 80 autopsied cases*

<table>
<thead>
<tr>
<th>Age (Years)</th>
<th>Volume (cc.)</th>
<th>Head Trauma* (Per cent)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>15-25</td>
<td>26-50</td>
</tr>
<tr>
<td>26-45</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>46-65</td>
<td>10</td>
<td>5</td>
</tr>
<tr>
<td>66-75</td>
<td>7</td>
<td>1</td>
</tr>
<tr>
<td>76-90</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>30</td>
<td>7</td>
</tr>
</tbody>
</table>

* Only patients with accumulations of 26 cc. or more are included in these means.

**TABLE 2**

*Relationship of volume of subdural hemorrhage to appearance of symptoms*

<table>
<thead>
<tr>
<th>Volume (cc.)</th>
<th>No. of Cases</th>
<th>Symptoms Probably Caused by Hem. (Per cent)</th>
<th>Hem. Major Cause of Death (Per cent)</th>
</tr>
</thead>
<tbody>
<tr>
<td>15-25</td>
<td>30</td>
<td>13.3</td>
<td>0</td>
</tr>
<tr>
<td>26-50</td>
<td>7</td>
<td>57.1</td>
<td>42.9</td>
</tr>
<tr>
<td>51-100</td>
<td>96</td>
<td>97.2</td>
<td>94.4</td>
</tr>
<tr>
<td>Over 100</td>
<td>7</td>
<td>100</td>
<td>85.7</td>
</tr>
<tr>
<td>Total</td>
<td>80</td>
<td>62.5</td>
<td>51.3</td>
</tr>
</tbody>
</table>
measured more than 50 cc., neurologic symptoms were an invariable occurrence (with the exception of 1 patient with Alzheimer's disease in whom assessment of symptoms was extremely difficult). For practical purposes, it was concluded that a subdural hematoma of 50 cc. or more produced some form of neurologic deficit or psychiatric alteration. Similarly, some correlation between volume of bleeding and percentage of fatality, attributable largely to the hematoma, could be determined. In patients with subdural hematoma greater than 100 cc., a review of the terminal clinical course indicated the strong likelihood that the hematoma was the immediate cause of death.

Some relationship between the volume of subdural bleeding and incidence of secondary brain-stem damage also was apparent (Table 3). An over-all incidence of 28.8 per cent secondary brain-stem change was present in the entire autopsy series of subdural hematoma. In those cases in which the volume of subdural hemorrhage was less than 25 cc., no instances of secondary brain-stem hemorrhage were uncovered. The frequency of this belated brain-stem damage increased progressively as the volume of the subdural hematoma increased, but diminished when the volume exceeded 100 cc. The reason for this paradox is clarified when the frequency of secondary brain-stem hemorrhage is equated with age (see below). The incidence of secondary cerebral infarction, particularly within the occipital lobes, was maintained at a steady rate in cases in which the volume of the subdural hematoma was more than 25 cc.

**Factor of Age of Patient**

The most striking factor influencing the biological behavior of subdural hematomata became apparent when the velocity and character of nervous-system reactions were assessed in relation to the age of the host. The clinical course, in patients beyond the age of 75, almost invariably was longer and without the acutely developing states of decerebration characteristic of patients in the middle decades of life. In many instances, a torpid clinical evolution was more suggestive of degenerative dementia than traumatic subdural bleeding, particularly in the absence of a history of injury. While severe depression of the state of consciousness, convulsions and motor deficits were predominant in the patients below the age of 75, these features were not as notable in patients beyond this age. In contrast, such symptoms as confusion, altered social behavior and disorientation were more paramount. It should be stressed that this comparison of symptoms referable to age was confined to those patients with subdural hematomata measuring more than 50 cc. and without significant degrees of associated parenchymal injuries such as contusions or intracerebral bleeding.

An inverse association between frequency of brain-stem hemorrhage and age exists in patients with subdural hematomata greater than 50 cc. In patients below the age of 65,
for example, about 77 per cent show secondary brain-stem hemorrhage (Table 4). Beyond the age of 66, the incidence decreases to about 6 per cent despite the fact that the average volume of subdural accumulation is somewhat greater in the older group.

This inverse correlation between age and incidence of secondary brain-stem hemorrhage is not unique to subdural hematoma. Rapidly growing, intrinsic neoplasms of the nervous system also are responsible for such secondary brain-stem hemorrhages. In reviewing 64 consecutive cases of glioblastoma of the cerebral hemispheres studied at autopsy at Kings County Hospital a comparable inverse association again is apparent (Table 4).

The extreme infrequency of secondary brain-stem hemorrhage in individuals beyond the age of 65 years, regardless of the inciting agency, naturally suggests that cerebral atrophy may play a major role in precluding this secondary phenomenon. Measurements of the weights of fresh brains at autopsy show a continuous decrease with increasing age. There is an estimated 4.0 per cent loss of brain substance between the ages of 60 and 80 years in males, and a 5.4 per cent loss in females. In contrast, however, the volume of the intracranial cavity does not alter appreciably with age. It may be assumed, therefore, that the amount of extracerebral space within the cranial cavity increases progressively. Korenchevsky’s statistics regarding the weight of the human brain indicate a similar decrease with age, i.e. 5.5 per cent loss in males and 4.2 per cent loss in females between the ages of 55 and 75 years.

Weights of brain, determined at autopsy, were recorded in most of the patients with subdural hematoma. In males below the age of 65 years the weights of the brains averaged about 4 per cent above normal values, were approximately of normal values in patients between 66 and 75 years of age, and were slightly less than normal (compared with controls of comparable age) in patients over 75 years of age. The number of female patients in the current series was not sufficient to permit an evaluation. The absence of brain-weight increment in the elderly patients with subdural collections would lend further support to the contention that enhanced cerebral volume in patients without prior cerebral atrophy is a feature in the pathogenesis of subdural hematoma.

The degree of cerebral vascular atherosclerosis, in relation to age, was also studied. While increasing sclerosis of the major arteries at the base of the brain characterized the older patients, there were no examples of significantly extensive encephalomalacia in any of the present cases.

**Comment**

Increased difficulties in the clinical recognition of subdural hematoma harbored within elderly patients have been noted repeatedly. Rabiner and Schacter have described 6 illustrative cases of patients with subdural hemorrhages varying in age from 64 to 80 years with symptoms mimicking those of diffuse cerebral vascular insuffi-
ciency. There was a notable absence of any signs suggesting brain-stem compression in their cases. Perlmutter\textsuperscript{7} also has been impressed with the gradual progression of symptoms in elderly people with subdural hemorrhage. The outstanding sign, in his series, was a deterioration in the state of consciousness. He attributed the altered clinical presentation in the elderly to the fact that the pre-existing atrophy of the brain prevented prompt and intensive compression of the underlying cerebral tissues.

The currently summarized data suggest that the same volume of subdural hemorrhage, in different age groups, may evoke different patterns of clinical response. Rapid deterioration in the state of consciousness, pupillary paralysis, decerebrate posture and clinical evidence of brain-stem embarrassment are noted more commonly in patients with subdural hematoma who are younger than 65 years, are rarely seen in patients beyond 66, and are never recorded beyond the age of 75 years. In contrast, prolonged periods of disturbed social and emotional demeanor, disorientation and slowly developing lethargy more commonly characterize subdural hematoma in patients who are older than 75 years.

Autopsy findings in the elderly patient with subdural hemorrhage indicate a notable absence of secondary lesions such as cerebral encephalomalacia or brain-stem hemorrhage.

It seems logical to suppose that the compressive effects of an extracerebral hemorrhage will be lessened as the volume of intracranial tissues diminishes and the free intracranial space increases.

References