SUBDURAL HAEMATOMA FROM ARTERIAL RUPTURE*

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The source of bleeding in subdural haematoma commonly is thought to be venous in origin, although there are well recognised exceptions to such a statement, such as the subdural clots associated with a ruptured aneurysm or those associated with contre-coup injury to the brain. Cases in which the bleeding arises from a single, small rent in a cortical artery are becoming recognised with increasing frequency. One hundred cases of subdural haematoma have been admitted to the Neurosurgical Service in London, Ontario; in 11 of these the bleeding definitely was arterial in origin, spurting from a small rent in a surface cortical artery, without evidence of surrounding brain injury. These cases form the basis of this presentation.

This arterial source of bleeding has received little emphasis in the past, and, with a few exceptions, the descriptions have been of postmortem specimens from untreated cases.

Hey reported the case of a football player who was struck on the head by a ball and died 2 hours later. At autopsy the fatal subdural haemorrhage apparently arose from a torn artery near the vertex of the hemisphere. Werkgartner described a 71-year-old man who fell and exhibited signs of cerebral compression in 3 hours. Death occurred 3 days later and histologic examination revealed the source of the fatal subdural haemorrhage to be a ruptured artery on the right temporal lobe. Stevenson, in a review of 80 cases of chronic subdural haematoma treated surgically in the Toronto General Hospital, cited 2 cases (12 days and 2 months following injury) in which the haemorrhage apparently resulted from a pin-point opening in an artery lying on the surface of the brain.

He postulated trauma as the cause, the artery having been adherent through the arachnoid to the dura mater. On the other hand, Scott felt in his case of chronic subdural haematoma in a 66-year-old clergyman, that the bleeding from a pin-point opening in the left Sylvian artery was spontaneous and not the result of trauma. Jaeger, in his discussion of Scott’s paper, mentioned that he had uncovered a spurting arterial rupture after wiping away a large clot from a Sylvian artery in a case of vascular hypertension. In a study of 102 cases of fatal subdural haemorrhage Vance found 6 undoubted instances in which rupture of a surface cerebral artery was the cause of the bleeding. Five were acute subdural clots and the initial loss of consciousness was profound and persistent in only 1 case, transient in another and absent in 2 cases. In the 5th case as well as in the case of a subacute clot the patients were admitted in a presumed alcoholic stupor with no history of injury. Dividing these cases into two groups, he presumed that in 2 cases, arterial branches were present in communicating or bridging vessels from the subarachnoid space to the dura mater and were ruptured during the trauma which probably caused oscillation of the brain. He had examined a few communicating vascular stalks and had found that while some contain only veins, others contain both arteries and veins. In the remaining 4 cases, the arterial lesion was termed a “fire hose” rupture, as it occurred in arteries on the lateral cerebral surface which possess small arterial twigs coming off the outer wall of the vessel at right angles just under the arachnoid. He felt that a point of weakness was present which was ill-fitted to withstand any increase of pressure within the vascular lumen. He considered that the brain was...
probably forcibly distorted against the inside of the dura mater, the vascular blood pressure was raised locally and the arterial twigs and overlying arachnoid were ruptured, causing the subdural haemorrhage. Krauland described 4 cases post mortem in which subdural haemorrhage resulted from injury to arterial twigs on the surface of the cerebrum by blunt injury without fracture. One case was acute (24 hours), 2 subacute (3 days and 15 days), and 1, a fresh subdural clot superimposed on an encapsulated chronic subdural haematoma. He felt that the mechanism was a contre-coup blow to the surface of the brain where the ascending ramus of the middle cerebral artery are not buried in sulci, the vessels being stiffened by age and high blood pressure.

Our experience has been with 11 cases (Table 1), 3 of which are presented in more detail as typical examples of the problem.

**CASE REPORTS**

**Case 1.** In June 1952, Cpl. S., aged 38, was admitted in deep coma with a fixed and dilated right pupil. Two hours previously he had returned from swimming complaining of headache. He became confused and had a generalised convulsion following which he remained stuporous.

Carotid angiography demonstrated subdural haemorrhage on the right.

At operation, in the semisitting position, through a subtemporal craniotomy a massive fresh clot was removed. Brisk bleeding continued into the field and by packing the subdural space vertically it was evident that it was arising from the parieto-occipital region. A large bone flap was turned down to uncover this area. Upon opening the dura mater another mass of freshly accumulated subdural clot was removed. This uncovered the source of the haemorrhage—a tiny stream of blood pumping out of a surface cortical artery in the region of the supramarginal gyrus. The field remained dry following the application of a silver clip. Neither the artery nor the adjacent brain appeared abnormal.

This patient has remained well for 6\(\frac{1}{2}\) years and has been unable to recall any injury although his wife felt he had “bumped his head” while swimming.

**Case 2.** In August 1952, H.D., aged 44, was seen by his physician, complaining of headache of 1 week's duration. Several days later he became ataxic and at the end of the second week he rapidly became stuporous and was admitted with a fixed dilated right pupil and right hemiparesis.

Carotid angiography revealed the typical deformity of a right subdural haematoma.

A semiliquid clot was removed through a subtemporal craniotomy. Slight bleeding persisted from the parietal region, but finally stopped with irrigation and gentle packing with cottonoids. Recovery was prompt and several hours later he was able to recall being struck on the head by a pole, knocked over by a horse about 2 weeks previously.

The following morning he became confused, then stuporous, with spasms of extensor rigidity and dilation of the right pupil. No recurrent clot was found at the site of the subtemporal craniotomy, but with a finger a large reaccumulation could be felt posteriorly. A parieto-occipital bone flap was turned down and a deep recurrent subdural clot was gently removed. This uncovered brisk bleeding from a tiny rent in a surface parietal artery with no evidence of surrounding brain injury. The tentorium was sectioned to relieve this second episode of uncal herniation.

The patient made an excellent though not un-

<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>Injury</th>
<th>Initial Coma</th>
<th>Interval to Coma</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. H.S.</td>
<td>38</td>
<td>Diving</td>
<td>No</td>
<td>2 hrs.</td>
<td>Well</td>
</tr>
<tr>
<td>2. H.D.</td>
<td>44</td>
<td>Struck by pole</td>
<td>No</td>
<td>14 days</td>
<td>Well</td>
</tr>
<tr>
<td>3. R.F.</td>
<td>3</td>
<td>Fall</td>
<td>No</td>
<td>½ hr.</td>
<td>Well</td>
</tr>
<tr>
<td>4. M.A.</td>
<td>49</td>
<td>Fall (epilepsy)</td>
<td>Yes</td>
<td>4 days</td>
<td>L. hemiparesis</td>
</tr>
<tr>
<td>5. G.H.</td>
<td>70</td>
<td>Fall</td>
<td>No</td>
<td>9 days</td>
<td>Died</td>
</tr>
<tr>
<td>6. C.W.</td>
<td>60</td>
<td>Motor vehicle</td>
<td>No</td>
<td>3 wks.</td>
<td>Well</td>
</tr>
<tr>
<td>7. M.J.</td>
<td>70</td>
<td>Fall</td>
<td>No</td>
<td>24 hrs.</td>
<td>Died</td>
</tr>
<tr>
<td>8. G.M.</td>
<td>67</td>
<td>Fall</td>
<td>Brief</td>
<td>4 hrs.</td>
<td>Died</td>
</tr>
<tr>
<td>9. M.B.</td>
<td>59</td>
<td>Unknown</td>
<td>No</td>
<td>---</td>
<td>Well</td>
</tr>
<tr>
<td>10. R.F.</td>
<td>68</td>
<td>Fall</td>
<td>No</td>
<td>7 hrs.</td>
<td>Died</td>
</tr>
<tr>
<td>11. M.W.</td>
<td>54</td>
<td>Blow</td>
<td>No</td>
<td>8 days</td>
<td>Well</td>
</tr>
</tbody>
</table>
eventful recovery, for a postoperative extradural clot developed following cranioplasty. He has remained well since.

Case 7. M.J., a 70-year-old known hypertensive, slipped on an icy sidewalk on the way to church. She told a neighbour that she had hurt her left elbow and left side of her head and did not feel like going to church. The neighbour visited her at 2:00 p.m. and found her to be drowsy and confused. By the following morning she was deeply comatose without lateralising signs.

A fresh subdural clot was removed from the left side. Arterial bleeding continued into the field, but by enlarging the craniotomy posteriorly and depressing the brain with a spatula, blood could be seen to be spurting from a pin-point opening in a small artery which branched downward from the Sylvian fissure. A silver clip dried up the field.

The patient remained unresponsive and died 12 hours later. Autopsy revealed midbrain haemorrhages but no recurrent subdural clot.

**DISCUSSION**

These 11 patients fall into a group principally because of the origin of the haemorrhage from a tiny rent in a surface cerebral artery without evidence of surrounding brain injury (Fig. 1). The trauma for the most part was trivial, for in only 1 case was there sustained initial loss of consciousness. This occurred in Case 4, an epileptic who struck her head against a table during a seizure. A simple depressed fracture in Case 3 was the only fracture in the group. The sites of arterial bleeding were around the lateral aspect of the Sylvian fissure (Fig. 2).

During craniotomy for other reasons, it is not uncommon to find a knuckle of a surface cerebral artery protruding through the arachnoid to be adherent to the dura mater or, more rarely, to be the origin of an arterial twig to the dura mater. In well-known ex-

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**Fig. 1. Case 10. Spurting arterial rupture on frontal operculum.**

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**Fig. 2. Sites of arterial bleeding. Square = right side; circle = left side.**
experiments Pudenz and Shelden have demonstrated under lucite calvaria the movement of the cerebrum with head injury. In 2 animals, massive "sub-lucite" venous bleeding occurred from tearing of parietal veins from the sagittal sinus. It is reasonable to assume that the arterial bleeding in the present series of cases stemmed from the gliding rotatory movement of the brain within the skull upon injury, tearing an artery or arterial twig from a dural attachment, leaving a tiny rent in the vessel. The universal absence of adjacent cortical bruising would exclude blunt injury and the common history of trivial injury would make a spontaneous origin only a remote possibility. A clear-cut history of head injury was obtained in all but 2 cases. Case 1 collapsed after swimming in a shallow river. He cannot recall an injury but his wife felt he had struck his head while diving. The bleeding in Case 9 apparently occurred during an episode of serum sickness but it is not known whether he struck his head against the bed. A spontaneous origin would imply local arterial disease such as a small malformation or atherosclerotic plaque. No remnant of an aneurysm or angioma was seen and degenerative disease was not evident in the arteriogram or at direct inspection at operation. Known hypertension was present in only 2 patients (Cases 7 and 10) and did not appear to be a factor in the bleeding. There was no visual evidence of an arterial twig at the bleeding point as described by Vance and Krauland, although such a twig may have been avulsed from its origin and not seen. In one patient (Case 7), a thread-like filament, a few millimetres in length, was attached to one side of the arterial opening. Histologic examination proved this to be a segment of dura mater undoubtedly torn at the moment of injury and avulsed from the membrane as the enlarging clot separated the brain from the dura mater.

The unusual frequency of this type of bleeding in this centre prompted critical thought whether the arterial injury could be the fault of the surgeon. This seems unlikely in view of the fact that 8 of the 11 bleeding sites lay on the brain exposed directly beneath the temporal craniectomy, where the clot extrudes spontaneously or is simply removed by suction. In only 3 instances was the arterial rent hidden posteriorly under the vault where a not too gentle finger might tear an arterial adherence or twig. In addition, following personal communication, several neurosurgical colleagues informed the writer that they have seen one or two cases of this type of bleeding (Drs. Alexander, Cluff, Fleming, Horsey and Rasmussen).

One would expect that arterial bleeding into the subdural space would mimic, in many respects, the classic syndrome of extradural haemorrhage, but it is remarkable that only 5 patients were operated upon within 24 hours. That this haemorrhage does not always result in early secondary coma is probably because of the tiny opening in the artery. The rapidity of formation and size of the clot must depend on the size of the rent, the bleeding ultimately ceasing because of local arterial factors and the clotting mechanism. Removal of the clot has opened the rent and persistent bleeding has been encountered in the subdural cavity. The source of bleeding in a few instances was not apparent immediately even when most of the clot had been removed. However, after a particularly firm adherent fragment was removed from the surface of the brain, the tiny stream of arterial blood jetted into the field. Fortunately in 8 cases the origin of the bleeding could be seen through the small temporal craniectomy. In 3 cases, however, persistent bleeding into the field required a bone flap to be made posteriorly from where the bleeding seemed to be coming. In all cases a dry field was obtained after the bleeding point was occluded. It is possible that this type of arterial bleeding might not be recognised as the cause of a haematoma when a small adherent fragment, occluding and hiding an arterial rupture, is left behind. By inspection the age of the clot seemed commensurate with the interval from injury and in no case was there evidence of repeated episodes of bleeding. One patient (Case 4) had a recurrent subdural clot from a large vein entering
the transverse sinus, torn by the operator's finger during removal of the original haematoma. This bleeding was thought to have been controlled.

Four deaths occurred in these 11 patients. It is well known that the greatest surgical mortality in subdural haemorrhage occurs when the interval to coma is brief. The bleeding is often both arterial and venous from bruising and laceration of the brain and with the associated cerebral swelling irreversible coma soon results from tentorial coning. One would expect that surgical treatment of subdural bleeding from simple arterial rupture without associated brain injury, would be rewarding. However, delay in recognition and treatment resulted in death in 3 of the 5 early cases. The patients with longer intervals to delayed coma did well, except for a 70-year-old lady whose death occurred 2 months after removal of a subacute clot. She remained bedridden because of confusion and hemiparesis.

CONCLUSIONS

1. A small but definite proportion of acute and subacute subdural haematomas result from a tiny rent in a surface cortical artery where it has been avulsed from a dural attachment by the motion of the brain upon injury.

2. Only 5 of 11 patients showed early delayed coma. In the remainder the bleeding stopped because of local arterial factors, and clotting about the tiny opening. There was no evidence of repeated bleeding.

3. Persistent arterial bleeding into the subdural space should be investigated for this cause. It may be necessary to turn a bone flap over the pietrotetoral region.

4. The mortality in the patients with brief interval syndromes was high (3 deaths in 5 patients) because of delay in recognition and treatment.

REFERENCES