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Review Article

Pathogenesis and biomechanics of traumatic intracranial haemorrhages

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Summary. Intracranial haemorrhage is frequently seen by the general pathologist in the context of neural trauma. Thus, the differential diagnosis, pathogenesis and biomechanics are of practical interest in the routine work. Extradural haematomas are produced when branches of the middle meningeal vessels are lacerated. They are commonly located in the temporal fossa, and other intracranial haematomas may be present. Skull fractures occur in a high percentage of cases and play a key role in the pathogenesis of this type of bleeding. Acute subdural haematomas commonly arise from tearing of the bridging veins. They are often located in the temporal and frontal regions, and the morbidity and mortality are related to the extent of the underlying brain damage. The visco-elastic behaviour of the bridging veins and their lack of reinforcement by arachnoid trabecula in the subdural space explains why they tear under high rates of acceleration during trauma. Subacute and chronic subdural haematomas are weakly correlated with trauma. The less striking onset of symptoms may be related to the rate of blood accumulation and the capacity of the brain to accommodate the mass effect of the bleeding. Intracerebral haematomas are probably due to the direct rupture of the intrinsic cerebral vessels. The mortality rate shows no correlation with location, but those located in the basal ganglia are compatible with a good recovery when occurring in isolation. Traumatic subarachnoid haemorrhage, when in isolation, is usually associated with evidence of injury elsewhere, such as the neck muscles or the ligamentary system of the cervical spinal column. It may be secondary to intraventricular bleeding due to tearing of the tela choroidea, or associated with contusions.

Key words: Extradural haematoma – Subdural haematoma – Intracerebral haematoma – Traumatic subarachnoid haemorrhage – Biomechanics

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For the general pathologist, neuropathological injury is generally seen as a result of falls or road traffic accidents (RTAs). Intracranial haemorrhage is a major component of the damage found and its pathogenesis is therefore of wide general interest. This review considers certain general aspects of the pathogenesis and course of various types of bleeding.

Major differences are seen between extradural and intradural haematomata, both in cause and effect.

Extradural haematomas

These haematomas frequently occur from laceration of branches of the middle meningeal artery or vein, or both. They have an incidence that has been estimated between 5% and 15% in fatal head injuries (Freytag 1963; Maloney and Whatmore 1969). Less frequently, laceration of the sagittal or the lateral venous sinuses, or of emissary veins, or any combination of the above may give rise to bleeding in the space between dura and skull (Gallagher and Browder 1968; Leestma 1988). Skull fracture demonstrated by X-ray, surgery or autopsy was found in 91% of 167 patients with extradural haematoma by Gallagher and Browder (1968). The temporal fossa is the commonest location of extradural haematomas, since in 83% of cases the bleeding source is the middle meningeal artery or one of its branches; only 20% of haematomas are located outside the temporal fossa (McKissock et al. 1960). Extradural haematomas may be associated with intradural haematomas. In a series of 125 cases (McKissock et al. 1960) extradural haematomas presented concurrently with intracerebral haematoma in 2 cases and with subdural haematoma in 12 cases. The subdural haematomas in these cases were ipsilateral in 8, contralateral in 2, and bilateral in 2. RTAs cause 45% of extradural haematomas, but the cause of injury varies with age. Falls at home account for most of those in children; RTAs are prominent in the second and third decades, and domestic accidents increase in

importance as a cause with increasing age (Jamieson and Yelland 1968).

Posterior fossa extradural haematoma occurs more commonly among children and teenagers. It is less common in the elderly, who fall onto the back of their head more frequently (Jamieson and Yelland 1968). Male predominance (3.6 to 1) is clear and a fracture of the occipital bone is seen in 84% of the patients (Roda et al. 1983). Loss of consciousness either at the time of impact or before surgical intervention, or an associated intracranial lesion are factors indicating a poor prognosis in this location. The overall mortality rate is 26% while the surgical mortality is 11% with excellent results after surgery in 65% of cases (Roda et al. 1983).

Prognostic factors for extradural haematomas include the level of consciousness at the time of operation. Patients who lost consciousness from the outset and remained unconscious before treatment had a 57% mortality rate in the now old series of McKissock et al. (1960). Age is an important factor, since in this series all patients over 60 years died and all under 7 years survived. The rate of evolution of the clinical picture shows that mortality is highest in the most acute cases, but is still considerable until the end of the 2nd week (McKissock et al. 1960).

Pathogenesis

The strength of dural attachment to the inner table of the skull plays a key role in the development of the extradural haematoma, and the strength of this adherence is age dependent. In infants and the elderly, the dura is strongly adherent to the bone and difficult to separate, which explains the lower incidence of extradural haematoma in the very young and in the aged (Gallagher and Browder 1968). The role of skull fracture has been emphasized; when a blow strikes the cranium, it produces detachment of the dura directly beneath the site of impact, and once the haemorrhage has begun, the blood fills the extradural pocket. With progressive bleeding, this pocket acquires an ever-widening perimeter as the dura is stripped away, and these haematomas may eventually extend all the way from the frontal to the occipital regions (Gallagher and Browder 1968).

Acute subdural haematomas

Post-mortem examination of non-missile head injuries reveals a widely differing frequency for these haematomas, estimated between 26% and 63% (Maloney and Whatmore 1969; Freytag 1963). They arise from laceration of cortical arteries and veins that occurs when penetrating injuries also lacerate the brain, and from closed head injuries resulting in large contusions. However, the most common type arises from tearing of the bridging veins that traverse the subdural space in their course from the brain's surface to the various dural sinuses (Gennarelli and Thibault 1982). The haematomas are unilateral in 90% of cases and are commonly found in the temporal and frontal lobes. The most frequent location is over the lateral portions of the cerebral hemispheres in the sylvian region; other sites include the far anterior, posterior, inferior, midline, and posterior cerebral areas or posterior fossa (Leestma 1988). Rare spontaneous cases, probably due to cortical arterial haemorrhages, have been reported (O'Brien et al. 1974).

Different patterns of clinical response are evoked in different age groups by the same volume of subdural haemorrhage (Aronson and Okazaki 1963). Rapid deterioration of the level of consciousness, pupillary paralysis, decerebrate posture and clinical evidence of brainstem compression are seen more commonly in patients under 65 years. They are rarely seen after 66 years and have not been recorded in patients over 75 years. The compressive effects of an extracerebral haemorrhage will be reduced as the volume of intracranial tissues decreases and the available free intracranial space increases (Aronson and Okazaki 1963).

The greater mortality and morbidity of subdural haematoma when compared with epidural haematoma is related to the associated underlying brain damage (Graham 1990). Mortality rates vary widely between different series, partly because different criteria have been taken into account for their estimation. Lewin (1949) estimated a 62% mortality in his series without specifying the parameters, while Fell et al. (1975) calculated a mortality rate of 48% for those patients treated within 24 h of injury and 45% for those treated within 72 h. Seelig et al. (1981) estimated a 30% mortality rate for those patients undergoing surgery within the first 4 h, whereas those who had surgery after 4 h had a mortality rate of 90%, stressing that the delay between injury and operation was the factor of greatest importance in the management. Age also influences the mortality rate. Patients under 10 years of age had a 33% mortality, while 69% of those over 60 years died (Fell et al. 1975). Analysis of the causes of acute subdural haematoma (ASDH) indicate that 72% are due to high strain-rate falls and assaults and that 24% are due to lower strain-rate vehicular injuries (Gennarelli and Thibault 1982).

Pathogenesis

ASDH occurs in association with high rates of acceleration during the trauma. It is the acceleration induced by an impact and not the head contact per se that causes ASDH, although the most common cause of clinical ASDH is impact to the head (Gennarelli and Thibault 1982).

The parasagittal bridging veins which are generally ruptured exhibit strong viscoelastic mechanical behaviour. Their elongation capacity is strongly dependent on the elongation rate at which the vessel is strained. The strain at which mechanical failure occurs is low when strain-rates are high and increases as strain-rate decreases. This means that the strain to produce vein rupture decreases by almost one order of magnitude if the strain-rate is increased from zero to 300 s^{-1} (Löwenhielm 1973). At static loading, the elongation may be

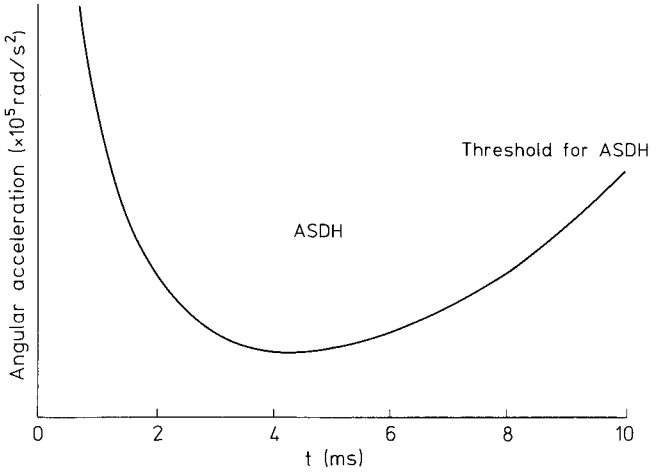

Fig. 1. Threshold curve for acute subdural haematoma (adapted from Genneralli and Thibault 1982)

more than 100%, while at elongation rates of about 500 s^{-1} the elongation capacity is reduced to 20%. Deformation of the brain close to the brain surface is considerable when the head undergoes angular acceleration. During the deformation, the parasagittal bridging veins undergo an elongation which is proportional to the relative angular displacement between the brain and skull (Thibault and Ginnarelli 1982), and if this elongation is more than 20%, rupture of the vessels will occur (Löwenhielm 1973).

Strain-rate sensitivity of the bridging veins was confirmed in an experimental model for ASDH, whereby holding angular acceleration constant but increasing its duration beyond 5 ms no ASDH occurred. If the acceleration pulse was greater than 5 ms and the acceleration magnitude was increased beyond 1.75×10^5 rad s^2, ASDH was produced (Gennarelli and Thibault 1982). The viscoelastic behaviour of bridging veins precludes an ultimate strain or stress criterion for failure. This tolerance should reflect an increased threshold for injury as the strain-rate is reduced because the ultimate strain which causes mechanical failure is inversely related to the strain-rate (Gennarelli and Thibault 1982). Tolerance level for long pulses depends only on the acceleration, whereas for short pulses it depends on both the acceleration and the pulse duration, becoming proportional to the angular velocity. In general, the critical change in angular velocity attains a constant value as the pulse length increases and the same is valid for the angular acceleration when the time (t) becomes small (Löwenhielm 1975). A tolerance curve for the bridging veins indicating the threshold for the occurrence of ASDH should have an upward concave shape, as shown in Fig. 1.

Subdural segments of the bridging veins are more fragile than their subarachnoid portions as they have thin walls of variable thickness, a circumferential arrangement of collagen fibres and absence of outer reinforcement by arachnoid trabecula when examined under electron microscopic observation (Yamashima and Friede 1984).

Subacute subdural haematomas

These are those haematomas that have remained undetected or unoperated upon for at least 3 days and up to 2 weeks after onset (Leestma 1988). The nomenclature of subacute subdural haematomas is not uniform (Jennett and Teasdale 1981), but they are best defined at operation when there is a mixture of clotted and fluid blood (Teasdale and Galbraith 1981). The mortality rate for these haematomas is much lower than that for ASDH. Nonetheless, they carry an increased risk of misdiagnosis because the onset of symptoms is less striking and symptoms may be non-specific, and because there is a weak association with severe head trauma (Leestma 1988).

Pathogenesis

Essential pathophysiological variables are the rate of blood accumulation, the compensation capacity of the brain for the increasing mass effect of the haematoma, and the co-existence of associated lesions that may modify the compensating capacity (Leestma 1988).

Chronic subdural haematomas

These haematomas represent a phase distinct from the subacute subdural haematomas when a neomembrane, sometimes tenuous, has formed over the undersurface of the haematoma (Leestma 1988). These haematomas can be recognized at operation when the contents consist of dark, turbid fluid and varying amounts of fresh blood (Teasdale and Galbraith 1981). They are at least 2 weeks old (Leestma 1988) but can present clinically after weeks or months following an otherwise trivial head injury (Graham 1990).

Pathogenesis

The pathogenetic mechanisms behind the formation of chronic subdural haematomas are unknown, and more than half of the cases lack a clear-cut traumatic aetiology (Leestma 1988). Best evidence to date suggests that the probable pathogenesis is related to persistent bleeding from capillary vessels in the neomembrane (Friede and Schachenmayr 1978; Schachenmayr and Friede 1978). The dural-arachnoid junction is nearly devoid of collagen, but it is populated with connective tissue cells called dural border cells. No capillaries are present normally, but injury stimulates their growth into this zone. These capillaries have a very thin wall and are often delicate and of large calibre; hence they are prone to spontaneous rupture without apparent trauma (Friede and Schachenmayr 1978; Schachenmayr and Friede 1978). Border cells proliferate following injury; then neomembrane formation occurs. This is followed by many cycles of injury, capillary and dural border cell proliferation, bleeding, and more proliferation until an equilibrium

is reached or the lesion is treated (Friede and Schachenmayr 1978; Schachenmayr and Friede 1978).

Intracerebral haematomas

Intracerebral haematomas are present in about 15% of fatal head injuries (Freytag 1963). They may be single or multiple and occur more frequently in the subfrontal and temporal regions. The cerebellum is affected less frequently (Graham 1990). Frontal haematomas are most often caused by occipital blows (46%) and rarely by frontal blows (8%). With lateral blows, they are ipsilateral to the trauma in 64% of the cases (Jamieson and Yelland 1968). Temporal haematomas occur most commonly as a result of lateral blows (61%) and are ipsilateral to the trauma in 58% of the cases; frontal and occipital blows each cause about 20% of the haematomas. The mortality rate has no correlation with the site of the haematoma (Jamieson and Yelland 1968).

Basal ganglia haematomas are deep intracerebral haematomas involving the striatum, the pallidum or the thalamus and can be classified as "large" if they are more than 2 cm in diameter or as "small" if they measure less than 2 cm in diameter (Adams et al. 1986). In patients with basal ganglia haematomas, associated contusions are more severe, the incidence of a lucid interval is lower, the incidence of gliding contusion and diffuse axonal injury (DAI) is higher than in patients without basal ganglia haematoma, and there is a strong association with RTAs (Adams et al. 1986). Survival time is strikingly different in patients with this type of haematoma; the range is from 2 h to 3 months, whereas in patients without these haematomas the survival is 2–3 days (Adams et al. 1986). When occurring in isolation they are compatible with a favourable recovery. The haemorrhage determines the clinical signs which are related to the side and subcortical structures involved. The overall cognitive impairment, and the speed and quality of recovery are strongly related to the associated DAI (Katz et al. 1989).

Pathogenesis

In intracerebral haematomas, the direct rupture of intrinsic cerebral vessels at the moment of injury is a plausible mechanism, but the physical principles involved remain elusive (Graham 1990). In contrast, basal ganglia haematomas are thought to arise from the shear strains elicited by acceleration/deceleration forces (Adams et al. 1986).

Burst lobe implies the concomitant presence of cerebral contusions, hamorrhage from superficial cortical vessels in the subdural space and an intraparenchymal haematoma, most frequently in frontal and temporal poles where contusions tend to be more severe (Adams 1984).

Traumatic subarachnoid haemorrhage

Trauma may produce more cases of subarachnoid haemorrhage than any other cause, with or without clinical manifestations of the process. Clinically, however, the most common form of subarachnoid haemorrhage is due to an intrinsic vascular disorder such as aneurysm, vascular malformation, or to haemorrhage due to hypertension (Leestma 1988). There are no characteristic differences in the type of artery damage which might indicate whether the damage is spontaneous or traumatic. Identifying trauma as the cause of an isolated subarachnoid haemorrhage is justified only when there are other distinct traces of the injury, such as in the neck muscles or on the lateral surface of the neck, as well as impairment of one of the anatomical elements of the upper cervical spinal column or ligamentary systems (Marek 1981).

Subarachnoid haemorrhage may also arise when blood follows the path of the cerebrospinal fluid. This occurs in cases of intraventricular bleeding due to tears in the tela choroidea resulting from closed head injury with acceleration/deceleration (Grĉević 1983).

References

Adams JH (1984) Head injury. In: Adams JH, Corsellis JAN, Duchen LW (eds) Greenfield's neuropathology. Arnold, London, pp 85–124

Adams JH, Doyle D, Graham DI, Lawrence AE, McLellan DR (1986) Deep intracerebral (basal ganglia) haematomas in fatal non-missile head injury in man. J Neurol Neurosurg Psychiatry 49:1039–1043

Aronson SM, Okazaki H (1963) A study of some factors modifying response of cerebral tissue to subdural haematomata. J Neurosurg 20:89–93

Fell DA, Fitzgerald S, Moiel RH, Caram P (1975) Acute subdural hematomas. Review of 144 cases. J Neurosurg 42:37–42

Freytag E (1963) Autopsy findings in head injuries from blunt forces. Arch Pathol 75:402–413

Friede RL, Schachenmayr W (1978) The origin of subdural neomembranes. II. Fine structure of neomembranes. Am J Pathol 92:69–84

Gallagher JP, Browder J (1968) Extradural hematoma. Experience with 167 patients. J Neurosurg 29:1–12

Gennarelli TA, Thibault LE (1982) Biomechanics of acute subdural haematoma. J Trauma 22:680–686

Graham DI (1990) Trauma. In: Weller RO (ed) Symmers systemic pathology, nervous system, muscles and eyes. Churchill-Livingstone, London, pp 125–150

Grĉevič N (1983) Traumatic tears of the tela choroidea: a hitherto unrecognized cause of post-traumatic hydrocephalus. Acta Neurochir [Suppl] 32:79–85

Jamieson KG, Yelland JDN (1968) Extradural haematoma: report of 167 cases. J Neurosurg 29:13–23

Jennett B, Teasdale G (1981) Management of head injuries. Davis, Philadelphia

Katz DI, Alexander MP, Seliger GM, Bellas D (1989) Traumatic basal ganglia hemorrhage: clinicopathologic features and outcome. Neurology 39:897–904

Leestma JE (1988) Impact injuries to the brain and head. In: Leestma JE, Kirkpatrick JB (eds) Forensic neuropathology. Raven Press, New York, pp 184–253

Lewin W (1949) Acute subdural and extradural haematoma in closed head injury. Ann R Coll Surg Engl 5:240–274

Löwenhielm P (1973) On the mechenism of cortical bridging vein rupture. Proceedings of the International Conference on the Biokinetics of Impacts, IRCOBI, Lyon. pp 423–430

Löwenhielm P (1975) Mathematical simulation of gliding contusions. J Biomech 8:351–356

Maloney AFJ, Whatmore WJ (1969) Clinical and pathological observations in fatal head injuries: a 5-year survey of 173 cases. Br J Surg 56:23–31

Marek Z (1981) Isolated subarachnoid hemorrhage as a medicolegal problem. Am J Forensic Med Pathol 2:19–22

McKissock W, Taylor JC, Bloom WH, Till K (1960) Extradural haematoma: observations on 125 cases. Lancet II:167–172

O'Brien PK, Norris JW, Tator CH (1974) Acute subdural haematoma of arterial origin. J Neurosurg 41:435–439

Roda JM, Gimènez D, Pérez-Higueras H, Blazquez MG, Pérez-Alvarez M (1983) Posterior fossa epidural haematomas: a review and synthesis. Surg neurol 19:419–424

Schachenmayr W, Friede RL (1978) The origin of subdural neomembranes. I. Fine structure of the dura-arachnoid interface in man. Am J Pathol 92:53–86

Seelig JM, Becker DP, Miller JD, Greenberg RP, Ward JD, Chol SC (1981) Traumatic acute subdural haematoma: major mortality reduction in comatose patients treated within four hours. N Engl J Med 304:1511–1518

Teasdale G, Galbraith S (1981) Acute traumatic intracranial haematomas. In: Kragenbühl H, Macpes PE, Sweet WH (eds) Progress in neurological surgery. Karger, Basel, pp 252–290

Thibault LE, Gennarelli TA (1982) The development of intracranial tissue component failure criteria as a consequence of controlled inertial loading. Proceedings of the Advisory Group for Aerospace Research and Development. AGARD-Conference Proceedings-322, NASA, NATO, pp 1–11

Yamashima T, Friede RL (1984) Why do bridging veins rupture into the virtual subdural space? J Neurol Neurosurg Psychiatry 47:121–127