KNIGHT CONTRECOUP

PRINCIPLE

*“Major impact right side of head causing swelling underneath.  Skull fractures radiating out from point of impact.  Brain damage underneath.  Fractures on opposite side of skull - contra coup damage.*

*Think of a hoop (skull) impact on one side forces skull in with fracturing away from impact travelling back into the point of impact.  Hoop bows out and fractures around that point travelling back in. (Struck hoop theory of skull fracturing around since the 1960s)*

*Rational was that low level falls do not cause fracturing of skull prevalent in the paediatric school of an injured child is a battered one but enough evidence that low level falls (3 foot or less from head height to ground (i.e. a child standing on a single step and falling back can fracture their skull -usually linear and without brain damage.  Does not need much more height i.e. 4 -5 foot before you get this sort of damage”*

Chapter 5 , Head and spinal injuries ***P.156-197***

The mechanism of brain damage

The brain may be injured:

* By direct intrusion, either by a foreign object such as a penetrating weapon, bullet or other missile---- or fragment of skull in a compound fracture where the skull is disrupted. In these open wounds the mechanism of the damage to brain is obvious, though of course it may be compounded by the second type of injury described below.
* By deformation of the brain in closed head injuries. Here the mechanism of injury is complicated and variable, with several competing theories of causation that have been put forward since the eighteenth century (Morgagni 1767, and LeDran 1751). A public debate was held in 1766 in Paris, where rival theories of coup and contrecoup brain damage were hotly defended at the Academy of Surgeons.

The brain is almost incompressible and purely axial impact may give rise to little or no damage. It is extremely rare, however, for an impact not to impact some rotatory movement and it seems agreed that this component is the main culprit in causing brain damage. What is now clear is that no actual blow or fall need be suffered by the head to cause severe and even fatal brain damage. It is the change in velocity ---- either acceleration or deceleration ---- with a rotational rater than solely axial element, that leads to damage: the surface of the head need never contact any hard object or surface. A prime example is the now universally accepted causation of infant subdural haemorrhage by shaking, a relatively common tragedy in child abuse.

In most head injuries ---- notably traffic accidents and falls ---- there is marked deceleration of the moving head on contact with a fixed surface, but in many criminal and combat injuries the head is accelerated by a blow. In either case the initial sudden change in velocity is applied to the scalp and skull, the latter then transmitting the change to the brain via the anatomical suspensory system within the cranium. This system is slightly flexible and consists of the falx and tentorium, which divide the cranial cavity into three major compartments; these contain the two cerebral hemispheres, the cerebellum and the brainstem. When violent relative movements take place between the brain and the dura forming the partitions of the cranium, the cerebral tissue can become damaged against both the sharp edges and the flat surface of these membranes. In addition, vessels traversing the subdural and subarachnoid spaces can be born by such relative movements, especially in old people where cerebral atrophy may have widened these spaces.

Among the competing theories of impact brain damage are:

* + - The rotational shear force theory.
		- The pressure gradient theory.
		- The vibration theory.
		- The transmitted wave force theory.
		- The brain displacement theory.
		- The skull deformation theory.

These hypotheses overlap and most are correct in some aspect. Because experiments on primates and mechanical models have been pursued vigorously in recent years, it is now widely accepted that there are marked pressure changes within the skull on impact[**(Yanagida, Fujiwara and Mizoi).**](http://hpe.h.kobe-u.ac.jp/~yasuyosi/headinjury2.html)**When a head falls against the ground, pressure momentarily increases at the impact point but falls to a negative value diametrically opposite. As these suction or cavitation effects are more damaging to neural and meningeal tissue than pressure, this is good evidence for claiming that contrecoup damage is largely a result of this vacuum effect.**

**The actual physical disruption of cerebral tissue is caused, according to both Gurdjian and Holbourn, by one or more of the following processes:**

**a, Compression of the constituent units, by their being forced together.**
**b, Tension of the units, which pulls them apart.**
**c, Sliding or ‘shear’ strains, which move adjacent strata of tissue laterally. The usual homely example is**
**given of a pack of playing cards being displaced, so that each card slides upon its neighbour.**
**Transient deformation of the skull almost certainly contributes to brain damage (Rowbotham). The area of the skull beneath an impact becomes momentarily depressed even if it does not fracture, and therefore may impinge on the underlying brain causing compression, as in (a) above. This is responsible for the typical cone-shaped contusions on the cortex, with the base at the surface, as the impact -----p possibly via short-lived oscillations of decreasing amplitude ----- injures the cortex and passes a diminishing force down into the deeper layers.**

**Simultaneously, other areas of the skull must bulge outward to accommodate the deformation ----- the so-called ‘struck-hoop’ action ---- when it is suggested that a ‘rarefaction’ remote from the impact may cause tension damage, as in (b) above.**

**More important is (c), being laminar deformity or ‘shear stress’ caused by the angular rotation of the head. As the head is pivoted on the first cervical vertebra almost any impact on jaw, face, or cranium will produce an angular momentum, the acceleration being conveyed first to the skull.**

**Alternatively, if the head is moving and is suddenly arrested, then the skull will decelerate first and the momentum of the brain will cause it to continue in motion, against almost certainly with some rotatory component.**

**In either the deceleration or acceleration mode, the skull and brain cannot change their velocities simultaneously, and the brain will speed up or slow down only by virtue of the restraint provided by the dural septa and the configuration of the interior of the skull. In other words, the brain is either retarded or set into motion secondarily by the skull, especially by the dural septa and the bony prominences.**

**This restraint will occur first --- and with maximum effect ---- on the most superficial layers of the cortex. These in turn will drag on the next deepest layer and so on until the difference in velocity is equalised ---- but this will have been at the expense of laminar tearing of the cerebral tissue and its associated blood vessels. In addition to this shearing damage, the brain may be forced against the sharp edge of the tentorial opening and the lower edge of the falx, causing damage to the base of the cerebrum, the corpus callosum, and the brainstem. Impact against the wide wall of the skull and against the falx may cause diffuse contusion of the cortex. The cerebellum tends to suffer less damage, as it is much smaller and lighter than the cerebrum and there is less room for relative movement in the more tightly-enclosed posterior fossa. The configuration of the interior of the cranium is thought to be partly responsible for the common localisation of cerebral damage at the tips and undersurface of the frontal and temporal lobes. The rough floor of the anterior fossa, the sharp edge of the wing of the sphenoid, and the massive bar of the petrous temporal bone are in contrast to the smooth inner surface of the vault of the skull.**

Coup and contrecoup damage

Whatever the underlying mechanics of cerebral damage, one aspect is of considerable practical importance to the pathologist. When a mobile head is struck with an object, the site of maximum cortical contusion is most likely to be beneath or at least on the same side as the blow. This is the so-called ‘coup’ lesion. When a moving head is suddenly decelerated, as in a fall, though there might still be a ‘coup’ lesion at the site of impact, there is often cortical damage on the *opposite* side of the brain---the ‘contrecoup’ lesion [**(Fig.5.34)**.](http://hpe.h.kobe-u.ac.jp/~shintai/headinjury1.jpg)

The mechanism of the ‘coup’ and ‘contrecoup’ injuries has long been dabated --at least since the time of the famous Paris meeting of 1766. In recent years the controversy has been continued, especially by Courville and by Holbourn, but no satisfactory resolution has been agreed though the recent work on intracranial pressures by **[Yanagida, Fujiwara, and Mizoi](http://hpe.h.kobe-u.ac.jp/~yasuyosi/headinjury2.html)**seems to provide proof that a ‘vacuum’ occurs at the contrecoup site.

The following practical points should be considered:

* There may be no coup damage at all, only contrecoup.
* There need be no fracture of skull, even in the presence of severe coup and contrecoup lesions.
* The most common site for contrecoup injury is in the frontal lobes (Fig.5.35). It is often at the tips of the frontal poles and may be symmetrical, if a fall on the occiput has occurred.
* In temporal or parietal impacts, the contrecoup lesions are likely to be diametrically opposite on the contralateral surface of the brain, but exact geometrical correspondence is not necessarily present.
* It is virtually unknown for a fall on the frontal region to produce occipital contrecoup. This is thought to be due to the anatomical configuration of the floor of the cranium, but the reasons are by no means understood.
* In a temporal impact, the contrecoup damage may be not be on the contralateral hemisphere, but on the opposite side of the ipsilateral hemisphere from impact against the falx cerebri.
* The degree of contrecoup damage may be severe, sufficient to cause blood-filled cavitation in the deep cortex and underlying white matter, especially in the frontal lobes and tips of the temporal lobes.
* With severe frontal contrecoup from a fall on the occiput, the transmitted force may be sufficient to fracture the thin bone of the floor of the anterior fossa. Such cracks in the roofs of the orbits may allow meningeal haemorrhage to speed into the orbits and appear as ‘black eyes’. In assaults where a fall has occurred, care must be taken not to attribute such periorbital bleeding to direct punches.
* Though contrecoup contusion is classically caused by deceleration of a falling head, it can also occur when a fixed head is struck. If the victim is already lying on the ground or against some other unyielding surface, a heavy blow on the upper side may cause typical contrecoup lesions either in the contralateral temporal or parietal cortex, or against the falx on the inner side of the ipsilateral lobe. In these circumstances, there is often coup damage as well.
* The interpretation of contrecoup lesions is most reliable in the form of cortical contusions or lacerations. Meningeal haemorrhage, either subdural or subarachnoid, may also arise as a contrecoup lesion. Its diagnostic value is, however, much less than that of cortical damage when interpreting a falling or fixed head injury, as they are more diffuse and mobile lesion. When no associated cortical contusion is present, it is unsafe to rely upon a unilateral meningeal haemorrhage as a definite indicator of the type of head injury.

Concussion
Concussion**is a clinical, not a pathlogical entity, but the pathologist must consider it, as it is related to intracranial lesions and he is often questioned about it in court proceedings, Concussion, according to Wilson, is 'a disorder of cerebral function which follows immediately upon the impact of a force to the head'. A more full definition is offered by Trotter: 'A transient paralytic state due to head injury which is of instantaneous onset, does not show any evidence of atructural cerebral injury and is always followed by amnesia from the actual moment of the accident'.**
  Some neurologists would also include postconcussion symptoms within the definition of concussion, even in the absence of initial coma, following a head injury. There may also be evidence of depressed medullary function, which can affect cardiorespiratory action. Denny-Brown and Russell (1941) showed that the rate of change of velocity of the head was important in producing concussion, which rarely developed if the speed threshold as less than 28 feet/second.
   It is an extremely common, if not inevitable, sequel to any significant mechanical insult to the brain. Though in general terms its duration is loosely related to the severity of the injury, there are many exceptions. Gross skull and brain damage have occurred with little or no apparent concussion, though concussion may be so transient that the subject may not even fall to the ground. Relatively minor head injuries have given rise to prolonged unconsciousness so, once again, it is most unwise to be dogmatic about retrospactive estimates of concussion.
   There is considerable controversy about the cause of concussion, from the unacceptable 'traumatic neurosis' on the one hand (which cannot be true) to claims for the inevitable demonstration of phys-
ical lesions on the other.
   Courville(1953) has discussed the condition in depth and there seems to be no reason to doubt that some mechanical process does temporarily disrupt the function, if not necessarily the structure, of the neuronic apparatus. Changes in the nucleus and cytoplasm of neurones, the composition of the  cere-
brospinal fluid and in the electroencephalograph have all been inconstantly reported (see 'dffuse axo-
nal injury' below).
   True concussion may last for seconds or minutes. If prolonged unconscious- ness extends into hours, days, or longer, then there is likely to be some structural brain damage. Occasionally what appears to be simple concussion proves to be fatal, causing respiratory paralysis, though at autopsy no    signifi-
cant lesions are found (Keen).
   Where a victim of 'simple' concussion dies of some incidental non-neurological condition, autopsy usually reveals no macroscopic damage, though sometimes there is slight cerebral oedema and scat-
tered  non-specific petechial haemorrhages may be found. There seems to be a connection between concussion and rotatory movements of the head, which are usually responsible for obvious structural damage, because when a head is fixed before impact loss of consciousness may not occur. The classic example is trapping of a head against a wall or being jammed between buffers.
   That shear stresses are instrumental in causing neurone damage seems confirmed by the frequency in which concussion occurs in boxing contests, where a blow on the jaw is the ultimate in producing a rotational movement of the cranium.
   Concussion may be followed by a 'postconcussion state' characterised by headaches, unsteadiness, and anxiety. This seems a genuine phenomenon, though it has been pointed out that it may be over-
lain by a 'compensation syndrome' whilst civil litigation is in progress over responsibility for the acci-
dent, which often clears up rapidly once the claim is settled.
   Retrograde amnesia is almost inevitably associated with concussion, though like concussion itself it may be so transient as to escape notice. A protective mechanism, it seems to be caused by loss of sen-
sory input before the latter is transferred to permanent memory storage in the brain. Though  com-
monly only of minutes' duration, it can extend to several days before the head injury. Though there is often a later recovery of much of this lost period, the memory of events immediately before the inci-
dent rarely return, which may fortuitously be a protective device. Concussion has been attributed to several causes, included the undoubted vasomotor disturbances that take place after a head injury. Another theory is the  impaction of the brain into the foramen magnum or tentorial opening, but the most acceptable hypothesis is 'diffuse neuronal injury'

KOKOLU

See discussion at page 4 *“Skull fractures after minor traumas such as domestic falls are more common among infants than older children. This may be because infants have a different anatomy compared with older children. For example the heads of infants are larger relative to their body size) than for older children and adults. Therefore during a fall their head is proportionally more exposed to external impact than other body parts. The body’s centre of gravity being closer to the head region in infants may also increase the frequency of head trauma after falls for this age group. In infants cranial bones are softer than those of older children and therefore less resistant to traumatic impact. Since the skull of infants is spherical only a small surface area absorbs the while impact of an external force”*

NHS ALERT

The cranium and facial bones are laid down from membrane in fetal life. The anterior fontanelle closes functionally between 9 and 26 weeks after birth, though is not tightly sealed until about 18 months. The posterior fontanelle closes between birth and 8 weeks of age. Suture lines close by interdigitation during childhood and osseous fusion occurs irregularly at variable dates during adult life.

The adult cranium consists of two parallel tables of compact bone called the ‘diploë’, the outer being about twice the thickness of the inner. They are separated by a central zone of soft cancellous bone, which is often misnamed the diploë. This zone is interrupted at suture lines and vanishes where the bone becomes particularly thin, especially in the floor of the skull.