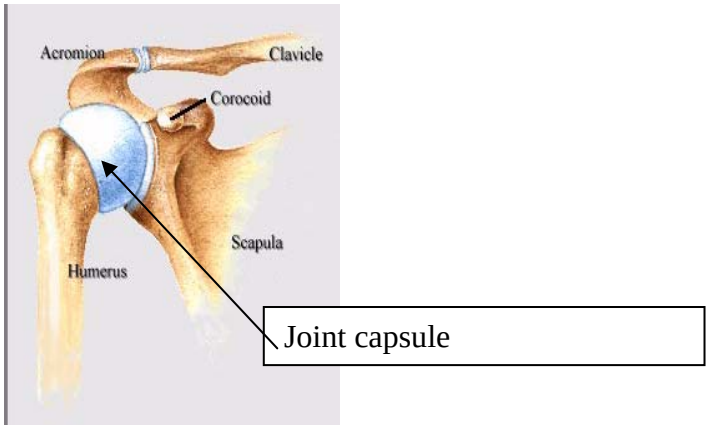
Injury Prognosis

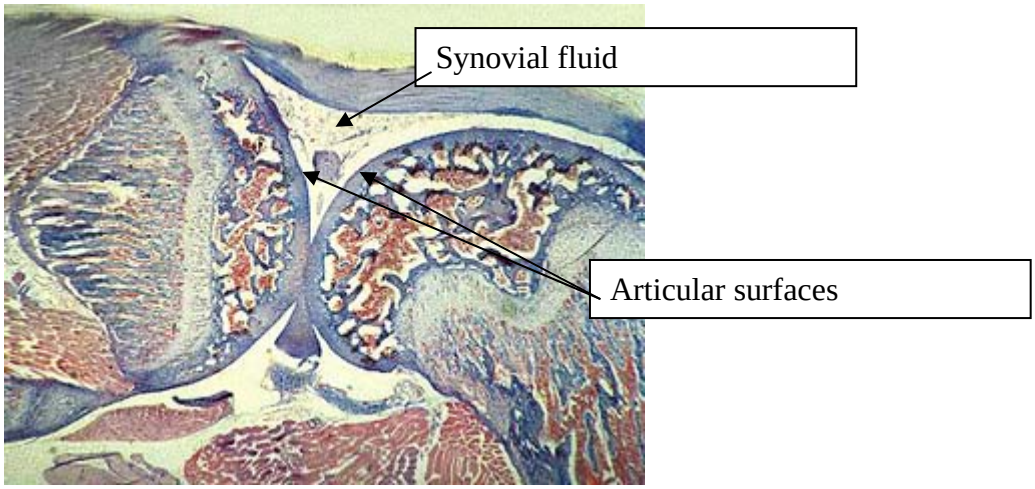
NWD Jansen et al. Arthritis and Rheumatism (2007) Vol.56#1 p 199 - 207
Exposure of Human Cartilage Tissue to Low Concentrations of Blood for a Short Period of Time Leads to Prolonged Cartilage Damage

The research demonstrates a mechanism whereby a joint injury could in principle aggravate, accelerate or cause a chronic joint disorder. Even where the injury does not directly damage the joint cartilages the proposal is that bleeding into the joint has the potential to have an irreversible damaging effect on that cartilage.

Joint trauma such as moderate sprain or joint fracture is often accompanied by bleeding into the sealed capsule that surrounds, and ensures lubrication of, the articulated surfaces. In the past 6 years, evidence has begun to emerge that a minor bleed into this part of the joint can provoke long term damage to the articular surfaces. The damage has been observed to have some characteristics of osteoarthritis and some of rheumatoid arthritis.

The white part of the shoulder diagram below indicates the joint capsule.





The present study was designed to assess the degree of bleeding required to lead to permanent changes in articular surfaces. The white part of the diagram immediately above is filled with a clear fluid [the synovial fluid] which acts a lubricant [it contains lubrication molecules known as proteoglycans]. It is thought that when blood contaminates this synovial fluid there is a possibility of damage to the articular surfaces.

Healthy human articular cartilage tissue was grown *in vitro* either in the presence or absence of diluted whole blood. Blood concentration, duration of exposure and effect of a recovery period after withdrawal of blood were the operating variables under study.

Outcomes were determined by measuring the rate of proteoglycan synthesis as well as the release and content of cartilage matrix proteoglycans and the activity of matrix metalloproteinases.

Exposure of cartilage to 50% (v/v) blood led to adverse changes that were largely independent of the exposure time. The adverse effects persisted for at least 12 days after an initial exposure of up to or exceeding 2 days. Proteoglycan synthesis was completely inhibited unless exposure was below one day. Exposure of cartilage to increasing concentrations of blood for 4 days led to concentration-dependent adverse changes.

These effects persisted when the concentration was ≥ 10% (v/v) blood. Moreover, after 2 days of exposure to a blood load of 10% (v/v), the adverse effects on cartilage were not reversible.

The authors propose that to prevent long term blood-induced joint damage aspiration [washing out] of blood from the joint within 2 days after bleeding should be considered.

Comment
The results provide preliminary evidence in support of permanent adverse changes in the joint following bleeding into a joint capsule. However, the duration of this experiment was insufficient to establish this with any certainty. Evidence of the loss of proteoglycan synthesising cells would support the view that the damage was permanent.

The actual significance of these in vitro changes cannot be assessed as yet. It could be that serious chronic debilitation could follow, or be accelerated, as a result of a reduced capacity for joint lubrication.

The number of injuries involving bleeding into the joint capsule is not readily known. Such bleeding would not show up on an X-ray image. It has been estimated that, in the UK, 10,000 patients each year may suffer cartilage damage warranting repair [NICE Technology Appraisal 89 (2005)]. The report does not state the proportion caused by impact injury.

In the neck there are synovial joints between the articular processes of the vertebrae. There is no intervertebral disc between the first and second cervical vertebrae. The articulations between these and between the skull and the first cervical vertebra are synovial.

The above mechanism could perhaps provide a non-psychosocial explanation for chronic neck pain following for example, whiplash neck injury. Osteoarthritis could perhaps be accelerated by such an injury. However, it is surprising that initial injury severity is not predictive of outcome after one year; if the above mechanism is valid this is what would be expected.

M Hooiveldt et al. American Journal of Pathology, Vol. 162, No. 3, March 2003 found evidence of permanent loss of chondrocytes in cartilage exposed to whole blood. Chondrocyte are the cells responsible for the production and maintenance of the collagenous matrix for the articular surfaces. Chondrocytes do not usually divide; once lost there is only limited replacement. Hooiveldt works with the same team as Jansen, so this may not be entirely independent evidence.