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SYNOVIAL FLUID IN THE CALF*

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SUMMARY

While awareness of synovial rupture of the knee joint confirmed by early arthrography increases, concomitant deep-vein occlusion may be being overlooked. Two cases of this are described, illustrating different mechanisms of venous occlusion by synovial rupture. Early thrombosis occurred in one patient from direct pressure as a result of haematoma following anticoagulant therapy and in another due to presumed external irritation of the vein. Although isolated bursae do not apparently rupture, tendon sheaths occasionally do. An example of lateral popliteal mononeuritis as a result of rupture of a tense peroneal tendon sheath is also given. Synovial rupture is primarily a clinical diagnosis but in selected cases, deep calf venography may be useful rather than arthrography. Almost a century ago, William Morrant Baker published his work on synovial protrusions showing his awareness of the consequences of free fluid in the calf. Some comments are made on his life and work.

An important rheumatological emergency is the appearance of irritant synovial fluid in the soft tissues of the leg and there are four possible sites of origin. The commonest is the knee joint or a synovial protrusion which has developed from it in the popliteal region or down the calf, as far even as the ankle. This was carefully described by William Morrant Baker almost a century ago (Baker, 1877). Rarely, an engorged tendon sheath can rupture, but never one of the numerous bursae about the knee, which although subject to fluid collection seem not to rupture unless connection with a joint generates sufficient pressure. Many clinical combinations are possible and among the consequences of synovial rupture with release of toxic fluid into soft tissues are venous thrombosis from external irritation or pressure from a nearby haematoma or, as in the following case, mononeuritis involving the lateral popliteal nerve.

In an advanced rheumatoid patient who was immobilized by limb-joint deformities, the peroneal tendon sheath was enlarged with synovial fluid and, in spite of rest and elevation, ruptured upwards with intense inflammation spreading to the upper fibula simulating a venous thrombosis. Soon after, a neurological lesion developed corresponding to complete mononeuritis of the lateral popliteal nerve at the level of the head of the fibula. A year later there had been no functional recovery.

The following cases illustrate the need to regard a deep venous thrombosis as an uncommon event in an ambulant rheumatoid patient; synovial rupture, leaking joint protrusion or traumatic rupture of calf muscle, especially soleus (Trenwith and Douss, 1975) being more likely. Furthermore, a careful search for any circumstantial evidence of previous knee-joint effusion is required and it should be borne in mind that arthrography is likely to be of value only in the first day or so as a rupture often seals off quickly and that the diagnosis of a 'burst knee' is essentially a clinical one. The most relevant investigation as regards management may well be venography of the deep venous system.

An elderly gardener developed a swollen knee after digging and was hospitalized four days later with a painful swollen calf, at which time, his small knee effusion was

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thought to be due to the effect of a high leg vein occlusion. He was anticoagulated and the leg was rested because the venogram showed almost complete occlusion of the deep veins in the calf, most of the contrast draining through the long saphenous system. However, after a few days the knee swelled again in conjuction with a generalized inflammatory polyarthritis and it was clear that the commencement of this was a knee effusion which had burst and externally irritated the deep veins in the calf, causing secondary thrombosis.

Another consequence of a synovial leak from the knee occurred in a woman with mild knee osteoarthritis and occasional effusion, who noticed a gradually swelling calf for which she was anticoagulated on clinical grounds. Lack of improvement led to venography which surprisingly showed patent deep veins. Then a haematoma appeared in the popliteal fossa of considerable size and with further calf swelling. A repeat venogram now showed blockage of the deep veins low in the calf. The sequence of events would appear to be synovial leakage, damaged calf muscle with haematoma formation from anticoagulation and venous obstruction from external pressure. Recovery was slow and she required tendo-achilles lengthening for calf contracture.

Free fluid in the calf was first described by Foucher (1856) but its origin from synovitis of the knee joint or related protrusions was first appreciated by Morrant Baker in 1877. The observations recorded in his first paper about to enjoy its centennial year make his name synonymous with this entity. This distinguished man died in 1896 aged 57 in the prime of life and enjoying well deserved success, from locomotor ataxy, which condition ironically, for the last 25 year of his life, he believed may have caused the form of knee osteoarthritis with which many synovial cysts were associated. He was described as a singularly handsome man of commanding presence, fond of boxing, a good change slow bowler, consistent and kind almost to a fault. His advice was often sought though he was regarded as a good opinion rather than a good operator. As a writer, he bestowed so much care in putting pen to paper that as a consequence whatever he wrote was so speedily amalgamated into the growing mass of literature that his connection with it was all too speedily lost.

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