The normal synovium occupies a tissue space confined by the joint capsule. This outer limit is sometimes clear, as in the volar plate of digital hinge joints, and sometimes imperceptible, as in the normal suprapatellar region where areolar synovial tissue appears to blend into the interstitial spaces of the thigh. The limit becomes obvious in chronic synovitis, however, when the capsule clearly confines the distribution of radiolabelled albumin and serves as a relatively non-compliant rind in the presence of pressurised effusions.1–3

Within the capsule lies a synovial fluid filled articular cavity invested by the synovial intima which is in turn surrounded by areolar, fibrous, or fatty subintimal tissues. None of these contiguous regions (cavity, intima, and subintima) is separated by a basement membrane, and all of them are served by the same interstitial fluid. Thus they should share essentially the same concentration of physiological solutes and they should experience the same pressure fluctuations as the joint is used.4

We know very little about the dimensions of those fluctuations in normal joints. Observations from a number of centres in a number of species agree that resting pressures in the normal knee are slightly subatmospheric—usually at around −4 mm Hg.3,5 This baseline may differ in other joints; the average canine wrist, for example, had a resting pressure of +2.9 mm Hg, but that finding was the rare exception to the subatmospheric rule. In this issue of the Annals, Gaffney and colleagues greatly expand our meagre database in normal human subjects by reporting ‘slightly subatmospheric’ resting pressures in eight metacarpophalangeal (MCP) joints, six wrists and one ankle.6 All of these data are interesting, but the resting joint should be only a brief chapter in the physiological story. What happens to intra-articular pressure when the joint is put to work? Perhaps surprisingly, Gaffney’s group found no change with periods of isometric exercise.8 In contrast, Jayson and Dixon found that intra-articular pressure in the knee decreased by more than 100 mm Hg during isometric quadriceps contractions.7

There is a logical explanation for these discrepant findings. The synovial fluid within each normal joint is a highly viscous, bubble free film interposed between and adherent to the mobile surfaces of cartilage and synovium. When the joint moves, that film is subjected to shear, with resultant tensile stress in the fluid itself. A pressure manometer within the joint space reflects this tension as subatmospheric pressure. As in other viscoelastic phenomena, the magnitude of the change increases dramatically with increasing speed of joint usage. This phenomenon was easily demonstrated in our studies of cadaveric and anaesthetised dogs and, moreover, must be carefully guarded against as a potential artefact in the measurement of resting pressure.5 The discrepant human findings of no change in smaller joints but marked change in the knee are best explained by the fact that ‘isometric’ knee exercises are not isometric at the patellofemoral joint. It seems most likely that the prominent pressure dips observed by Jayson and Dixon represent the fluid stress imposed by patellar motion—a factor not present in the simpler joints studied by Gaffney’s group.

When the substantial tensile strength of synovial fluid is overcome, it fractures with a resulting audible ‘crack’ and a radiographically demonstrable gas bubble as the legacy of the resultant cavitation.8 The phenomenon is the subject of interesting experimental studies in third MCP joints subjected to increasing increments of purely tensile force,9,10 but is rarely achieved in this way by recreational knuckle crackers; they, instead, develop the requisite synovial fluid tension by short, sharp shocks of hyperextension or hyperflexion. These manoeuvres place the fluid under motion induced tension and then fracture it with lever induced distraction.

Knuckle cracking amuses many people in the non-rheumatological community and its logical explanation is fun. The point of this essay, however, is more fundamental. It seems reasonable to suggest that synovial fluid is regularly subjected to substantial cyclic tensile stress (and subatmospheric pressure) in the course of normal joint use. The more rapidly the joint moves, the more effective is the mechanism. The most interesting aspect of this is not its occasional failure (and resultant joint crack), but its usual integrity. This means that the simple movement of one joint surface over another draws these tissues toward each other and thus imparts a formidable stabilising factor which opposes distracting forces and promotes normal tracking.

In the presence of an effusion, this stabilising influence is lost as joint motion increases rather than decreases the intrasynovial pressure. This, of course, is the aspect of interest to Gaffney and colleagues, who found that mean resting pressures are greater than atmospheric in rheumatoid MCP, wrist, ankle, and elbow joints.9 In these joints, and in their saline filled normal counterparts, isometric contraction led to substantial further increases in intra-articular pressures. Despite the much greater age in the rheumatoid group (median 64 years compared with the median normal group age of 30) and, presumably, some degree of disease related debility, the contraction induced...
pressure increases were consistently greatest in the rheumatoid subjects. Presumably, the positive pressure pulse is brought about by extrinsic compression of the closed articular space. The greater responses in the rheumatoid subjects suggest that, as others have shown in the rheumatoid knee, their articular capsules are significantly less compliant.\(^2\)\(^3\)

These observations are long overdue and clearly most welcome. To the clinician, they should mean that the rubbery, firm feeling synovitis sometimes present in rheumatoid hands implies that these joints have increased articular pressures and that their vascular supply may be compromised.\(^1\) One hopes that these pilot observations on pressure will be followed by corresponding metabolic data that will, for the first time, give us a feeling for the prevalence of ischaemic impairment in the smaller peripheral joints where so much rheumatoid destruction is found. In time, too, we will learn whether increased baseline pressures correlate with the rate of disease progression and the extent of articular impairment. The authors believe their findings offer support for their hypothesis that hypoxic reperfusion injury is a significant factor in rheumatoid joint destruction.\(^1\) Maybe so; maybe not. It should now be beyond doubt that sustained pressure promotes articular ischaemia.\(^12\)\(^13\) It could well be, however, that the brief pressure pulses induced by normal joint usage confer a positive benefit through pumping of lymphatic and venous vessels within the synovial tissues. More work will be needed to resolve this issue. For the present, the authors are to be commended for providing all of us with thought provoking data. If more rheumatologists take time to think about the pressures within the joints they palpate, perhaps more will be stimulated to make the basic observations that are so much needed in this field.

Department of Medicine, RG-28,
University of Washington,
Seattle, WA 98195, USA

Supported by NIH grant AR32811.