Intra-articular pressure changes in rheumatoid and normal peripheral joints

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Abstract

Objective—To investigate the intra-articular pressure (IAP) dynamics of a spectrum of joints in rheumatoid and normal subjects in order to determine whether a reperfusion event is likely to occur at these sites.

Methods—IAP was measured in the metacarpophalangeal (MCP) (n = 8), wrist (n = 8), ankle (n = 4), and elbow joints (n = 4) of rheumatoid subjects, in addition to the MCP (n = 8), wrist (n = 6), and ankle joints (n = 1) of normal healthy controls, using the hand held portable 295-1 Intra-Compartmental Pressure Monitor System (Stryker, UK).

Results—Resting IAP was positive in all rheumatoid joints, and subatmospheric or weakly atmospheric in normal subjects (p < 0.01). Exercise produced an increase in IAP in rheumatoid subjects only (p < 0.01). The addition of saline to normal joints mimicked the IAP changes seen in the rheumatoid group.

Conclusion—These observations suggest that increased resting IAP is a marker for chronic joint inflammation. The IAP increase seen in the rheumatoid group during exercise supports the concept of hypoxic reperfusion mediated joint injury.

In normal knees, resting IAP is at or slightly less than atmospheric pressure, becoming sub-atmospheric during periods of exercise.670 Rheumatoid subjects with chronic knee effusions have significantly greater resting pressures; during exercise, these may increase in excess of capillary perfusion pressure, and sometimes of systolic blood pressure.13

Artificial volume expansion of normal and rheumatoid knees (mimicking the effect of an effusion) also increases resting and exercise related pressures, the greatest pressures being generated in rheumatoid subjects.5-7 In addition, when an external pressure cuff is applied to rheumatoid knee joints with effusions, even modest increases in IAP (20 mm Hg) compromise synovial blood flow.8

This may result in a synovial perfusion-metabolic demand mismatch, manifest by increased synovial fluid lactate concentrations, Pco2 and reduced pH.9

Although IAP has been extensively studied in the knee joint, there is limited information available regarding other human synovial joints in either normal or rheumatoid subjects, and the effect of normal joint usage on IAP is largely unknown. Eyering et al demonstrated low resting IAP in the normal joints (two wrists, one ankle) of living subjects, and greater resting IAP in rheumatoid subjects with a joint effusion (one elbow, one shoulder, one ankle); however, the methods they used were unable to distinguish between atmospheric and sub-atmospheric IAP recordings.10

We have therefore measured IAP in a spectrum of rheumatoid and normal joints in order to evaluate the IAP dynamics, and determine whether a reperfusion event is likely to occur at these sites.

Patients and methods

After obtaining ethics committee approval and informed patient consent, we measured IAP in metacarpophalangeal (MCP), wrist (radio-carpal), and ankle (talocrural) joints (normal and rheumatoid subject), and in elbow (radio-humeral) joints (rheumatoid subjects only), using the hand held portable 295-1 Intra-Compartmental Pressure Monitor System (Stryker, UK). This device was developed specifically to measure compartment pressure and is used as an adjunct to diagnosis in compartment syndrome. For the purpose of this study, a digital, hand held multimeter (ISO-TECH IDM 63) was attached to the pressure monitor in order to determine
whether recorded pressures were atmospheric or subatmospheric.

The pressure monitor system consists of two principal components: a digital monitor containing a solid state transducer and case with auto zeroing device, and a sterile disposable pressure monitor set containing a prefilled saline syringe and diaphragm chamber to maintain a sterile fluid pathway. After assembly, the pressure monitor device was attached to a standard 21 gauge needle. Subcutaneous tissues were infiltrated with 2% lignocaine, sterile care being taken to avoid capsular or intra-articular contact. The pressure monitor set was assembled and primed by slowly forcing saline through the chamber to the needle tip, in order to ensure hydrodynamic communication with the joint lumen. The monitor was then set at zero and the needle introduced into the joint cavity. Accurate intra-articular positioning was supported either by successful aspiration of synovial fluid after IAP measurement, or observing consistent pressure fluctuations during exercise (rheumatoid subjects) and after injection of saline (normal subjects). When equilibrium had been established, IAP was recorded at rest and during periods of isometric exercise (resisted joint flexion and extension). At least six cycles were recorded in each individual and the mean value reported. In normal individuals, 0-9% normal saline (MCP, 0-2 ml; wrist and ankle, 2 ml) was injected into the joint space, to mimic the effect of an effusion. Sterile precautions were observed throughout. All operators were experienced in arthrocentesis.

**Statistical analysis**

As the data were not normally distributed, non-parametric statistical methods were applied. Within each patient group, resting and exercise related pressure changes were compared using the Wilcoxon signed rank test. The non-parametric Mann-Whitney U test was used to assess the differences between RA and normal subjects.

**Results**

Twenty patients who fulfilled the American College of Rheumatology (formerly, the American Rheumatism Association) criteria for the diagnosis of RA were recruited. They comprised 14 women and six men with a median age of 64 years (range 43–72). A spectrum of clinically inflamed joints (pain, swelling, tenderness, and limitation of movement) were studied. These included eight MCP, eight wrist, four ankle and four elbow joints. In addition, 15 healthy volunteers (median age 30 years, range 23–41; four women, 11 men; eight MCP, six wrist, one ankle) were studied. Recorded pressures are expressed in mm Hg and quoted as medians (interquartile range).

All rheumatoid patients registered positive IAP at rest, and an increase in IAP during exercise. Synovial fluid (median volume 3 ml, range 0–8) was aspirated from eight rheumatoid patients after joint cannulation and completion of IAP measurement (three wrist, three elbow, two ankle). Resting and exercise related IAP was subatmospheric or weakly atmospheric in control subjects. The administration of saline produced an increase in IAP to the atmospheric range in all control subjects. During exercise, a further increment in IAP was noted in this group; however, a trend towards reduction in the maximally generated exercise related IAP was observed during the period of intra-articular cannulation. The addition of saline was also associated with the development of a subjective sensation of joint heaviness.

**MCP joints**

Resting IAP was 14 mm Hg (10–17) and –2 mm Hg (–4 to 0) in rheumatoid (n = 8) and normal (n = 8) subjects, respectively (p < 0.001). In rheumatoid subjects IAP increased to 103 mm Hg (92–118) during exercise (p < 0.01); no significant change was seen in normal subjects. After the addition of intra-articular saline to normal joints, resting and exercise related IAP increased to 18 mm Hg (13–25) and 75 mm Hg (60–117) respectively (both p < 0.05) (fig 1).

**Wrist joints**

A resting IAP of 18 mm Hg (13–25) and –2 mm Hg (–3 to 1) was recorded in rheumatoid (n = 8) and normal (n = 6) subjects, respectively (p < 0.01). Exercise produced an increase in IAP to 85 mm Hg (56–103) in RA subjects only (p < 0.01). In normal subjects, the addition of intra-articular saline produced an increase in resting IAP to 17 mm Hg (16–22); this increased further, to 50 mm Hg (43–81), during exercise (both p < 0.05) (fig 2).
rheumatoid joints, and increases further during exercise. Previous IAP studies have almost exclusively been centred on the knee joint, in which it has been proposed that such changes in IAP compromise capillary perfusion. Because of the large surrounding muscle mass and unique weight bearing functions of the knee joint, observations made regarding IAP dynamics at that site may not be reliably extrapolated to other human synovial joints. This description of an identical phenomenon in non-weight-bearing joints supports the concept of an exercise related hypoxic reperfusion event.

Normal joints have subatmospheric or weakly atmospheric resting and exercise related pressures. Although the addition of saline produced an increase in IAP, smaller exercise related pressures were generated in these artificially volume expanded normal joints than in corresponding rheumatoid joints. In addition, during the subsequent period of isometric exercise, a gradual reduction in exercise related IAP was observed. This pattern has previously been reported in experimentally induced effusions in rabbit knees, and, together with the subjective feeling of joint heaviness experienced after administration of saline, may be explained by reflex muscle inhibition. This phenomenon has previously been described in patients with acute traumatic effusions, and may be due to the joint effusion exerting an inhibitory effect via capsular mechanoreceptors, thereby resulting in inhibition of the Hoffmann reflex. Ultimately, this protects against the generation of high exercise related IAP and may explain the low incidence of chronic synovitis in patients with acute traumatic effusions.

In this study, IAP measurements were recorded using a blind technique. Despite our initial concern regarding accuracy of joint cannulation, preliminary studies demonstrated that exercise related IAP fluctuations were not seen when the needle tip was placed in subcutaneous tissues at the time of failed joint cannulation. This, together with the fact that synovial fluid was successfully aspirated from eight rheumatoid patients immediately before completion of the procedure, strengthened our clinical confidence regarding accuracy of needle placement. In addition, great care was taken to ensure that local anaesthetic was not injected into the joint capsule or cavity, as this may increase joint effusion volume, increase IAP, and indirectly influence periarticular muscle tone via local anaesthetic effects.

In addition to likely implications for hypoxic reperfusion injury, pathologically increased IAP may have additional deleterious effects. These include impairment of synovial blood flow and nutrition, increased periarticular tension producing local discomfort, dilution of hyaluronic acid and other secreted macromolecules which may impair joint lubrication, induction of muscle weakness via reflex muscle inhibition, and chronic distortion of the joint capsule and ligaments. Ultimately, these mechanisms result in further impairment of joint function and mobility.

ANKLEJOINTS
At rest, IAP was 16 mm Hg (15–18) and −3 mm Hg in rheumatoid (n = 4) and normal (n = 1) ankle joints, respectively (p < 0.01). During exercise, IAP increased to 92 mm Hg (82–98) in the rheumatoid group (p < 0.05); there was no change in the normal subject. The addition of saline to the normal ankle joint increased resting and exercise related IAP to 26 mm Hg and 62 mm Hg, respectively (fig 3).

ELBOWJOINTS
IAP was 12 mm Hg (8–17) in rheumatoid elbow joints (n = 4) at rest, increasing to 88 mm Hg (66–142) during exercise (p < 0.05) (fig 4).

Discussion
Using an adapted intra-compartmental pressure measuring device, we have shown that IAP is increased in a spectrum of resting
We have demonstrated that increased resting and exercise related IAP is seen in a spectrum of rheumatoid joints. Similar IAP changes are likely to occur in other chronically inflamed rheumatoid joints. Our findings suggest that increased resting IAP is a marker for chronic synovitis. The additional IAP increase observed during joint exercise further supports the concept of hypoxic reperfusion mediated joint injury.

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