Synovial rupture

Experiments on cadaveric knees

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Rupture of a joint occurs when the intra-articular synovial pressure fluid increases excessively and the capsule lacks sufficient resistance. Fluid is released into the soft tissues and may produce an acute inflammatory reaction. This has been described most frequently but not exclusively in rheumatoid arthritis of the knee (Dixon and Grant, 1964; Dixon, 1964; Tait, Bach, and Dixon, 1965; Hall and Scott, 1966; Jayson, Swannell, Kirk, and Dixon, 1969), and less often in the elbow (Goode, 1968) and wrist (Jayson and others, 1969).

Quadriceps contraction normally produces negative or small positive intra-articular pressures in contrast with the very high values found in the presence of rheumatoid disease and of effusion (Jayson and Dixon, 1970). Significant fibrosis and resistance to these stresses develops only in more advanced rheumatoid involvement so that the clinical syndrome of acute knee joint rupture is most common in early disease (Jayson and others, 1969).

A series of experiments was therefore performed to determine the pressures needed to rupture cadaver knees. Observations were directed at determining the effects of age and sex of control and osteoarthrotic (OA) joints.

Experimental material (Table)

Experiments were performed on one or both knees of cadavers, usually within 6, and always within 24 hours of death. Refrigerated specimens were not used.

OA knees were defined as those in which the diagnosis had been made during life and was clinically and radiologically documented. Control joints were those with no

Table No. of joints examined, by sex

Sex	Male	Female	Total
Osteoarthrosis	60	21	81
Control	42	62	104
Total	102	83	185

FIG. 1 Apparatus used.

preceding history of joint disease and which moved fully and without crepitus after two or three trial flexions to relieve any rigor mortis. The first 25 joints were opened and examined after rupture to confirm the diagnosis and the reliability of these criteria.

Method (Fig. 1, opposite)

A Braun size 2 cannula was passed through a small incision behind the patella into the joint space and connected via a three-way tap to a 20-ml. syringe and to a reservoir containing 4.3 per cent. dextrose and 0.18 per cent. sodium chloride in water. A mercury manometer

was connected to the joint space via a second cannula. 10-ml. increments of fluid measured with a syringe were added to the joint. The intra-articular pressure was recorded at each increment. A sudden fall in the intra-articular pressure and loss of signs of an effusion indicated joint rupture. The pressure at which this occurred was noted.

Results (Figs 2-9)

Correlations for age and sex were found in the control joints. In both male and female knees there was a significant increase in the rupturing pressure with

FIG. 2 Male control knees. Regression line for rupturing pressure and age.

FIG. 3 Female control knees. Regression line for rupturing pressure and age.

FIG. 4 Control knees. Rupturing pressures, male and female, by age group.

FIG. 5 Male OA knees. Rupturing pressures.

FIG. 6 Female OA knees. Regression line for rupturing pressure and age.

FIG. 7 OA knees. Rupturing pressures, male and female, by age group.

age (Figs 2 and 3) and, when stratified by age, the mean rupturing pressures of male joints were higher than for female and the differences were usually significant (Fig. 4). An age correlation was found for female but not for male OA knees (Figs 5 and 6). Only in the young age groups were the rupturing pressures higher for the male than for the female OA joints (Fig. 7).

In each age group and in both sexes the OA joints ruptured at higher mean pressures than the controls and the differences were statistically highly significant (Figs 8 and 9).

SITE OF RUPTURE

After rupture of fifteen of the OA knees, methylene blue was injected into the joint space and by dissection, the site of the rupture was determined. In fourteen it was in the postero-lateral aspect of the joint, and in only one did it occur in the suprapatellar pouch.

Discussion

The increase in the rupturing pressure occurring with advancing age is suggestive of a progressive alteration in the collagen fibres of the joint capsule. Fibrosis of the capsule in later years could produce this resistance to rupture as well as the limitation of joint mobility commonly noticed. Elasticity measurements of human skin suggest a similar age change (Ridge and Wright, 1966a; Grahame, 1969) although this is in contrast with biochemical analysis of skin

collagen content (Shuster and Bottoms, 1963; Shuster, Raffle, and Bottoms, 1967; Stevenson, Bottoms, and Shuster, 1970).

The male joints ruptured at higher pressures than the female joints. This is in contrast with the natural sex incidence of acute joint rupture which is more common in male rheumatoid knees. Although male control joint capsules would appear to be more resistant to this complication, it may be that male rheumatoid tissue does not possess this advantage. Alternatively, male rheumatoid subjects may undertake more strenuous activity so that they develop higher intra-articular pressures than females.

The high rupturing pressures in osteoarthritis indicate that changes are not confined to the articular surfaces but occur also in the joint capsule. Indeed, pain receptors are absent from articular cartilage, so that changes here alone would be insufficient to explain the symptoms commonly developed. Sensory nerve endings are found in the joint capsule (Wyke, 1967), so that stretching this stiff tissue would produce abnormal stresses and symptoms of pain. Jayscn (1969) described three patients with no clinical or radiological evidence of joint disease but with severe arthralgia, who had considerably increased stiffness of the joint capsule.

The site of rupture in fourteen of the fifteen OA knees was not similar to that observed by arthrography in the clinical syndrome of acute joint rupture, and did not correspond with the previous experimental work of Dixon and Grant (1964) when

FIG. 8 Male knees. Rupturing pressures, OA and control in each age group.

FIG. 9 Female knees. Rupturing pressures, OA and control in each age group.

the joint usually ruptured at the apex of the suprapatellar pouch.

These measurements were made usually several hours after death. There may well be *post mortem* changes that are extremely difficult to quantify. In addition there is loss of the normal muscular tone protecting parts of the capsule. For these reasons, the results obtained may well differ from those to be expected if the experiments had been performed during life. We suggest, however, that the correlations with age and sex, and the difference between control and osteoarthritic joints reflect similar trends in joint capsule structure in living joints.

Summary

Measurements were made of the rupturing pressures of cadaver, control, and osteoarthrotic knee joints. Higher pressures were required in male than in female joints and the rupturing pressures showed significant correlations with age. Higher pressures were required in the osteoarthrotic joints than in the controls, indicating the presence of changes in the joint capsule as well as in the articular cartilage.

This work was performed with the aid of a grant from the Arthritis and Rheumatism Council. One of us is in receipt of a research grant from the Medical Research Council (M.I.V.J.).

We acknowledge with thanks the help of Mr. G. M. James of the Department of Medical Illustration, Bristol Royal Infirmary.

Discussion

PROF. V. WRIGHT (*Leeds*) I am unhappy about bending the knee to overcome rigor mortis, since the tissue is visco-elastic not elastic. We have demonstrated in the finger that if you bend it cyclically the force needed diminishes with time because it is a plastic substance (Ridge and Wright, 1966b). Secondly, I should like to

know your criteria for the diagnosis of osteoarthrosis. Thirdly, I think one has to be cautious about drawing parallels between these results and the stiffness of collagen elsewhere because, if you correlate age with either the tensile strength of skin collagen, or with the load required to rupture the aponeurosis of the abdomen, both follow a very similar curve! This does not decrease with age as Dr. Jayson has found, but rises until the age of 40 and then falls. I was interested that Dr. Jayson did not find this, and I wonder if he has any comments about the disparity between his results and those usually found when one is studying the tensile strength of collagen?

DR. M. I. V. JAYSON The trial flexions before the experiment were performed equally in both control and osteoarthritic knees, so that I think the effects would be similar in both. I agree that there may well be *post mortem* changes, but these would have applied equally to both groups. The diagnostic criteria for osteoarthrosis were that the diagnosis had been made during life and was confirmed by *x* rays. Regarding the tensile strength of the tissue, we were in fact examining the bursting of the capsule. I am not clear whether this can be equated with tensile strength, which is the resistance to an elongating force. One can imagine something of low tensile strength which could burst relatively easily or alternatively extend considerably.

DR. R. GRAHAME (*London*) There is a difference between the actual force at which rupture occurs and previous stress-strain response. As far as skin is concerned, one is dealing in the latter case with elasticity, or the resistance to stretch, and in the former with ultimate tensile stress, which is the breaking point and well beyond the elastic limit.

PROF. V. WRIGHT (*Leeds*) If my comments had been based solely on the skin I should accept that, but in the experiments with aponeurosis we imposed an impact load to determine the breaking point. What intrigued us was that the age/breaking correlation was exactly the same as for skin, and I think that this was a comparable situation.

References

- DIXON, A. ST. J. (1964) *Proc. roy. Soc. Med.*, **57**, 1129 (Acute rupture of the knee in rheumatoid arthritis).
- AND GRANT, C. (1964) *Lancet*, **1**, 742 (Acute synovial rupture in rheumatoid arthritis).
- GOODE, J. D. (1968) *Ann. rheum. Dis.*, **27**, 604 (Synovial rupture of the elbow joint).
- GRAHAME, R. (1969) *Ann. phys. Med.*, **10**, 130 (Elasticity of human skin *in vivo*).
- HALL, A. P., AND SCOTT, J. T. (1966) *Ann. rheum. Dis.*, **25**, 32 (Synovial cysts and rupture of the knee joint in rheumatoid arthritis).
- JAYSON, M. I. V. (1969) *Ibid.*, **28**, 677 (A new syndrome?).
- AND DIXON, A. ST. J. (1970) *Ibid.*, **29**, 401. (Intra-articular pressure in rheumatoid arthritis of the knee. III. Pressure changes during joint use).
- , SWANNELL, A. J., KIRK, J. A., AND DIXON, A. ST. J. (1969) *Ann. phys. Med.*, **10**, 175 (Acute joint rupture).
- RIDGE, M. D., AND WRIGHT, V. (1966a) *Gerontologia (Basel)*, **12**, 174 (The ageing of skin—A bio-engineering approach).
- (1966b) *Ann. rheum. Dis.*, **25**, 509 (Rheological analysis of connective tissue; a bioengineering analysis of skin).
- SHUSTER, S., AND BOTTOMS, E. (1963) *Clin. Sci.*, **25**, 487 (Senile degeneration of skin collagen).
- , RAFFLE, E. J., AND BOTTOMS, E. (1967) *Lancet*, **1**, 525 (Skin collagen in rheumatoid arthritis and the effect of corticosteroids).
- STEVENSON, C. J., BOTTOMS, E., AND SHUSTER, S. (1970) *Ibid.*, **1**, 860 (Skin collagen in osteogenesis imperfecta).
- TAIT, G. B. W., BACH, F., AND DIXON, A. ST. J. (1965) *Ann. rheum. Dis.*, **24**, 273 (Acute synovial rupture).
- WYKE, B. (1967) *Ann. roy. Coll. Surg. Engl.*, **41**, 25 (The neurology of joints).