EXPERIMENTALLY PRODUCED FRACTURES OF ARTICULAR CARTILAGE AND BONE

THE EFFECTS OF SHEAR FORCES ON THE PIG KNEE

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Experimental injuries of cartilage and bone were produced by applying shear force to the articular surfaces of the lateral femoral condyles of six-month-old pigs under various loading conditions. The lesions were divided into two groups, 'open' or 'closed', depending on the presence of a crack on the articular surface. Each was further divided into four types according to the depth of penetrating injury: (1) splitting of uncalcified cartilage; (2) splitting at the subchondral plate; (3) subchondral fracture; and (4) intra-articular fracture.

When shear force was applied at high speed but with low energy, the articular cartilage surface was the first to crack. At low speed and low energy, splits occurred in the deeper layers first. As the energy increased, both loading conditions eventually resulted in similar open lesions.

Experimentally produced shear injuries are useful models for clinical osteochondral fracture, osteochondritis dissecans, and chondromalacia patellae.

The articular surfaces of joints are exposed to various forces which can cause different forms of lesions to articular cartilage and bone. The forces may be divided into two kinds, compression and shear. In normal joint function, only compression forces act on the articular surface, shear forces being negligible because of the low friction coefficient between two articular surfaces. In twisting and spraining injury and joint dislocation, however, convex articular surfaces are subject to large sideways forces which may result in a variety of shearing lesions of cartilage and bone.

Most osteochondral fractures are thought to be caused by shear forces (Milgram 1943, 1978; Kennedy, Grainger and McGraw 1966; Matthewson and Dandy 1978) and osteochondritis dissecans may be the result of

an ununited osteochondral fracture (Rosenberg 1964; Aichroth 1971; Matthewson and Dandy 1978) although it is not known under what circumstances an osteochondral fracture develops into osteochondritis. Shear damage to the articular cartilage has been suggested as a cause of chondromalacia patellae because of the association with malalignment of the patellofemoral joint (Hughston 1968; Insall, Falvo and Wise 1976).

Clinical observations alone cannot resolve these questions about the effect of shear forces at a joint. We therefore studied the effects of sideways forces on animal joints in the laboratory and attempted to correlate our experimental findings with clinical findings.

MATERIALS AND METHODS

Osteochondritis dissecans, chondromalacia patellae and osteochondral fractures occur mostly in adolescence. We therefore used the joints of six-month-old pig carcasses because their articular cartilage is similar to that of the human adolescent and the epiphyses are still open. The animals' body-weight was about 110 kg.

The lateral femoral condyles were removed and either used immediately or stored at -30°C and thawed to room temperature on the day of the experiment. Specimens were kept wet with physiological saline during testing.

Impact forces, or quasi-static forces at a low constant speed, were applied diagonally to the convex articular

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surfaces. The forces were similar to those generated in life by dislocations, sprains or excessive rotatory movements between the two articular surfaces.

The equipment used is shown diagrammatically in Figure 1. The loading box and the loading tower were connected by a sliding guide fitted with ball-bearings to minimise friction. The lateral femoral condyle was fixed inside the box so that only its articular surface was visible outside the box. A mould for the upper half of the articular surface was made using acrylic resin and placed in contact with the articular surface of the condyle. Its edges were smoothed off and it was aligned at an angle of 120°. The dimensions of the articular surface of the mould were: width 18 to 22 mm, height 7 to 9 mm, and depth 6 to 8 mm. At the end of the experiment, the projected area of the articular surface covered by the mould was measured from a photograph. If this was larger or smaller than between 75 and 100 mm² the specimen was excluded from the data.

The vinyl silicone rubber (G. C. Dental Industry Co, Tokyo, Japan) consisted of two pastes which, when mixed and allowed to solidify for six to eight minutes,

produced an elastic sheet, 3 mm thick. During the experiment, the specimen was constantly covered by this sheet (Fig. 1), which protected the cartilage surface from the irregular hard edges of the acrylic resin mould. The silicone rubber layer models the articular cartilage of the tibial condyle of the natural knee.

The tangential modulus of stiffness of the silicone rubber for 30% strain, at a cross-head speed of 2.4 mm/minute, was between $1.22 \times 10 \text{ MN/m}^2$ and $1.84 \times 10 \text{ MN/m}^2$ in five material testings. This is comparable with the 'short-term' stiffness of visually normal cartilage from the human hip (1.96 MN/m^2 to $1.44 \times 10 \text{ MN/m}^2$) (Freeman and Kempson 1973).

In the impact experiment, the weighted loading box was allowed to fall onto the specimen from different heights. In the quasi-static experiment, we used an Instron testing machine to lower the loading box at a constant cross-head speed of 2.4 mm/min. The cross-head was stopped at various displacements and damage to the specimen was examined in detail.

For the statistical analysis of data, we used Student's *t*-test.

Fig. 1

The lateral femoral condyle is held in the fixation box with plaster of Paris. A mould of the upper half of the articular surface is made from acrylic resin and fixed in the loading box. A layer of self-curing vinyl silicone rubber is inserted between the acrylic mould and the articular surface of the specimen.

RESULTS

Open and closed lesions. The lesions were classified into two types, according to whether they were open or closed. The articular surface of the specimen was examined by the naked eye for visible cracks on the surface. Small impressions and abrasions were ignored (Fig. 2).

Depth of lesion. Using a metal wheel cutter, three to five 1 mm-thick slices of the articular surface were taken from the vicinity of each lesion. These were examined by the naked eye, by stereoscopic microscopy, and by radiography, to determine the depth of the lesion, which was classified, as shown in Figures 3 and 4, into four types.

Type I. Splitting of the uncalcified cartilage. Radiography showed no involvement of the calcified tissues.

Type II. Splitting at the subchondral plate which consists of the calcified cartilage and the bone plate. Radiography showed separation of a thin layer of the calcified tissues.

Fig. 2

An open lesion (left) and a closed lesion (right).

Fig. 3

Fig. 4

Figure 3 – The depth of the penetrating injuries. Figure 4 – Classification of experimentally produced cartilage and bone lesions: type IA, B, splitting of uncalcified cartilage; type IIA, B, splitting at the subchondral plate; type IIIA, B, subchondral fracture; and type IVA, B, intra-articular fracture.

Fig. 5

Graph to show the distribution, by velocity and by energy, of articular lesions caused by experimentally applied impact loads.

Table I. Results of the impact shear force experiment

Type of lesion	Number	Energy (Nm)		Speed (m/sec)	
		Mean	SD	Mean	SD
Intact	35	26.65	13.06	3.50	0.85
IA	7	38.19	10.72	4.81	0.85
IIA	8	51.48	8.10	4.30	0.70
IIIA	4	60.60	8.31	4.48	0.73
IVA	9	59.87	16.64	4.51	0.72
Open lesions	28	51.50	15.58	4.52	0.73
IB	1	38.30	–	3.51	–
IIB	7	40.79	9.85	3.37	0.34
IIIB	0	–	–	–	–
IVB	0	–	–	–	–
Closed lesions	8	40.48	9.2	3.39	0.34
Total	71	38.01	17.97	3.89	0.91

Type III. Subchondral fracture involving the cancellous bone at some distance from the subchondral plate.

Type IV. Intra-articular fracture in which the fracture line extends to the metaphysis.

Since each of these four lesions could be open (A) or closed (B), there were eight types of injury.

The impact shear force experiment. We performed this experiment on 71 specimens in 28 of which cracks in the articular surface were produced. Of the remainder, eight had closed lesions. The impact speed and energy were

obtained from calculation of the weight of the loading box and the height from which it fell. Table I gives the type of lesion, the mean impact energy and the mean impact speed. The correlation of impact energy and impact speed is shown in Figure 5.

The distribution of cartilage and bone lesions was as follows:

- 1) At energy levels below 20 Nm no lesions were observed in 13 cases.
- 2) At energy levels ranging from 20 to 40 Nm, 11 specimens out of 24 showed cartilage and bone lesions, of which seven were open and four closed. Five of the open lesions were type IA with splitting of the uncalcified cartilage. Of the four closed lesions, one was type IB and the other three were type IIB.
- 3) At energy levels between 40 and 60 Nm, nine specimens out of 25 were intact; there were 12 open and four closed lesions.
- 4) At an impact energy of 60 Nm or greater, all the lesions were open and none was type IA.
- 5) Type IA lesions ($n = 7$) occurred at a significantly higher speed than did closed lesions ($n = 8$) ($p < 0.01$), but there was no significant difference between them in terms of the energy level.
- 6) Type IA lesions ($n = 7$) were observed at a significantly lower energy level than other open lesions ($n = 21$) ($p < 0.01$), but there was no significant difference between them in terms of speed.
- 7) Closed lesions ($n = 8$) occurred at significantly lower

Fig. 6

Graph to show the distribution by force and by displacement, of articular lesions caused by experimentally applied quasi-static loads.

energy levels and at lower speeds than did open lesions, if type IA was excluded ($n = 21$) ($p < 0.01$).

In summary, type IA lesions were seen at high-impact speeds and low-energy levels. Closed lesions occurred more frequently from a low-speed low-energy force. Open lesions, which damaged both the cartilage and subchondral bone, occurred at higher energy levels.

Since closed lesions were observed more often from low-impact speeds, we performed further experiments to confirm this finding, using an Instron testing machine to apply the force at low constant loading speeds.

Quasi-static shear force experiment. We performed this experiment on 49 specimens at a cross-head speed of 2.4 mm/min. The cross-head was stopped at various distances of displacement. When a crack was noted on the surface, the cross-head was immediately stopped. If this occurred after the peak load, the latter was recorded as the maximum force. In 11 specimens the force produced a crack on the cartilage surface and in ten, closed lesions occurred. The type of cartilage and bone lesion, the mean displacement of the cross-head and the mean maximum force are given in Table II. The correlation for each specimen of maximum force and displacement of the cross-head is shown in Figure 6. The distribution of lesions was as follows:

1) Cartilage and bone injuries developed when displacement of the cross-head exceeded 6.4 mm or the load exceeded 1370 N.

2) Open lesions ($n = 11$) were produced by a mean force

of 2275.27 N and a mean displacement of 12.60 mm; closed lesions by a mean force of 1902.1 N and a mean displacement of 11.20 mm. For the intact group the mean force was 1739.14 N and the mean displacement 6.52 mm. There was no significant difference between open and closed lesions in either the applied force or the displacement which produced them. There was, however, an apparent difference between the intact group and those with open lesions both in applied force and displacement ($p < 0.05$, $p < 0.01$) and between the intact group and the closed group in displacement ($p < 0.01$).

3) There were some morphological differences in the lesions which resulted from this experiment compared with those in the impact experiment. No type IA lesions were observed, but closed subchondral fractures (type IIIB), which were not seen in the impact experiment, were easily produced. Fractures caused by impact forces resulted in simple thin fracture lines, but those caused by quasi-static forces had wider, irregular fracture lines accompanied by compression of the bone trabeculae.

We conclude that a low force applied at a constant speed results first in splitting of the deeper layers of the cartilage and subchondral tissues and then involves the surface of the cartilage. Damage does not extend from the cartilage surface to the deeper layers.

DISCUSSION

The articular end of a bone is multilayered consisting, from the surface to the interior, of uncalcified cartilage, calcified cartilage, subchondral bone plate and subchondral cancellous bone. It is resistant to compression forces but is vulnerable to sideways forces because the considerable differences in the mechanical properties of the layers allow large distortions and make them prone to splitting.

In the present study we were able to produce characteristic lesions by changing the speed and energy of the shear force.

Since bone and cartilage are visco-elastic, their mechanical behaviour varies with the speed of application of the force. Thus, at high impact speeds, visco-elastic cartilage tends to stiffen, and if further force is applied, it becomes prone to injury and a type IA lesion develops from the articular surface. At lower impact speeds, cartilage is more flexible and can be distorted to a greater extent before it splits. In this case, the underlying subchondral bone is more likely to be damaged, giving rise to a closed lesion.

At low speed, with the force applied by the Instron machine, there were no type IA lesions demonstrating that at low-impact speeds damage occurs away from the articular surface.

Both high- and low-speed injuries finally develop into similar open lesions, from excessive loading.

We attempted to correlate these results with clinical

Fig. 7

Fig. 8a

Fig. 8b

Figure 7 – Open splitting of uncalcified cartilage (type IA) which does not involve calcified tissue. Figure 8a – Closed splitting of uncalcified cartilage (type IB). Figure 8b – Histological section ($\times 130$ haematoxylin and eosin).

Fig. 9a

Fig. 9b

Osteochondral fractures of two types (a) open lesion with splitting at the subchondral plate (type IIA) and (b) open subchondral fracture (type IIIA).

findings. Type IA injuries, open splitting of uncalcified cartilage, are often seen in the traumatised knee, but the crack does not reach as far as the subchondral bone (Fig. 7).

The type IB lesion (Fig. 8) was seen in only one case out of the 120 specimens tested in our experiments, but some of the specimens classified as type IIB and IIIB also had extensive splitting of uncalcified cartilage and thus overlapped with type IB lesions. Pommer (1915), Sokoloff (1956), Meachim and Bentley (1978) reported that

Fig. 10

Fig. 11

Figure 10 – Closed splitting at the subchondral plate (type IIB): the split line runs above, through and deep to the subchondral plate. Figure 11 – Closed subchondral fracture (type IIIB). A combination of intact articular surface and separation of a subchondral cancellous bone fragment.

histological studies of animal and human cartilage often showed horizontal splits at the uncalcified-calcified cartilage interface. Meachim and Bentley (1978) and Minns, Birnie and Abernethy (1979) have described how the considerable differences in the mechanical properties of uncalcified and calcified cartilage could cause horizontal microfractures from shear damage. The closed splitting in the deeper area of uncalcified cartilage produced by our experiment (type IB) seemed to be somewhat different from the horizontal splitting of

Table II. Results of the quasi-static shear force experiment

Type of lesion	Number	Force (N)		Displacement of cross-head (mm)	
		Mean	SD	Mean	SD
Intact	28	1739.14	523.06	6.52	2.78
IA	0	—	—	—	—
IIA	1	1470.00	—	11.00	—
IIIA	5	2475.40	628.67	13.10	4.80
IVA	5	2236.20	499.76	12.42	3.58
Open lesions	11	2275.27	586.20	12.60	3.84
IB	0	—	—	—	—
IIB	5	1764.40	359.88	12.38	2.45
IIIB	4	1737.50	266.93	10.28	2.75
IVB	1	3234.00	—	9.00	—
Closed lesions	10	1902.10	548.24	11.20	2.63
Total	49	1892.75	573.72	8.84	4.03

cartilage that develops on the tide-mark in mature cartilage. But it may be that our experimentally produced closed splitting is a precursor of the basal degeneration described by Goodfellow, Hungerford and Woods (1976).

Other type IIA and type IIIA lesions are known clinically as osteochondral fractures (Fig. 9). The type IIA lesions have an eggshell-like fragment of bone detached immediately below the subchondral plate and type IIIA lesions may often involve detachment of a considerable piece of the cancellous bone.

Clinically, the closed lesions, type IIB (Fig. 10) and type IIIB (Fig. 11), are probably treated as simple contusion of the articular surfaces, because they are not detected by radiography at the time of injury. In dislocation of the patella or twisting of the knee, there may be little or no change visible on the articular surface, but flake-like shadows in the subchondral region may be observed later on the radiograph. These shadows may result from type IIB lesions.

Our experimental findings may also shed light on the pathogenesis of osteochondritis dissecans. In its early stages the superficial articular cartilage is intact but as the condition progresses, cartilage cracks appear and the cartilage and bone eventually become loose. These processes suggest that the crack develops first in the subchondral area and only later appears at the surface.

Experimentally induced closed subchondral fractures, type IIIB (Fig. 11), combine intact cartilage and

separation of an area of subchondral cancellous bone, which could well be a precursor of osteochondritis dissecans.

In type IV lesions, the fracture lines which extended to the metaphysis and outwards from the articular cavity were similar to the common intra-articular fractures of clinical experience.

In summary, our study indicates that a variety of cartilage and bone lesions including osteochondral fracture, chondromalacia patellae and osteochondritis dissecans may all result from shear injuries.

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REFERENCES

- Aichroth P. Osteochondral fractures and their relationship to osteochondritis dissecans of the knee: an experimental study in animals. *J Bone Joint Surg [Br]* 1971; 53-B:448-54.
- Freeman MAR, Kempson GE. Mechanical properties of articular cartilage. In: *Adult articular cartilage*. London: Pitman Medical, 1973:171-227.
- Goodfellow J, Hungerford DS, Woods C. Patello-femoral joint mechanics and pathology. 2. Chondromalacia patellae. *J Bone Joint Surg [Br]* 1976; 58-B:291-9.
- Hughston JC. Subluxation of the patella. *J Bone Joint Surg [Am]* 1968; 50-A:1003-26.
- Insall J, Falvo KA, Wise DW. Chondromalacia patellae: a prospective study. *J Bone Joint Surg [Am]* 1976; 58-A:1-8.
- Kennedy JC, Grainger RW, McGraw RW. Osteochondral fractures of the femoral condyles. *J Bone Joint Surg [Br]* 1966; 48-B:436-40.
- Matthewson MH, Dandy DJ. Osteochondral fractures of the lateral femoral condyle: a result of indirect violence to the knee. *J Bone Joint Surg [Br]* 1978; 60-B:199-202.
- Meachim G, Bentley G. Horizontal splitting in patellar articular cartilage. *Arthritis Rheum* 1978; 21:669-74.
- Milgram JE. Tangential osteochondral fracture of the patella. *J Bone Joint Surg* 1943; 25:271-80.
- Milgram JW. Radiological and pathological manifestations of osteochondritis dissecans of the distal femur: a study of 50 cases. *Radiology* 1978; 126:305-11.
- Minns RJ, Birnie AJ, Abernethy PJ. A stress analysis of the patella, and how it relates to patellar articular cartilage lesions. *J Biomech* 1979; 12:699-711.
- Pommer G. Zur Kenntnis der Ausheilungsbefunde bei Arthritis deformans, besonders im Bereiche ihrer Knorpelursuren, nebst einem Beitrag zur Kenntnis der lakunären Knorpelresorption. *Virchow Archiv* 1915; 219:261-78.
- Rosenberg NJ. Osteochondral fractures of the lateral femoral condyle. *J Bone Joint Surg [Am]* 1964; 46-A:1013-26.
- Sokoloff L. Natural history of degenerative joint disease in small laboratory animals: pathologic anatomy of degenerative joint disease in mice. *Arch Pathol* 1956; 62:118-28.