IMPULSIVE AND STATIC LOADING OF THE PORCINE TEMPOROMANDIBULAR JOINT DISC

BY

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ABSTRACT

At present no validated mechanism explains the connection of trauma with the delayed onset of craniomandibular disorders (CMD). The hypothesis advanced in this study is that trauma can cause surface damage to the temporomandibular joint (TMJ) disc. This damage compromises the disc's lubrication function and initiates a fatigue-like process which can ultimately result in the premature failure of the disc. Damage to the lubrication mechanism will cause an increase in the friction associated with joint movement, giving rise to an increase in the rate of wear of the surface of the disc, and enlargement of the damaged area. As long as the rate of increase of damage outstrips repair, this surface abrasion effect will cause the premature failure of the disc. For any of this to be possible it is first necessary to demonstrate that a traumatic blow to mandible can cause damage to the surface of the disc. The *in vitro* study that was undertaken used porcine TMJ discs and subjected them to a variety of impulsive and static loads. Prior to and following the applied trauma the friction associated with the start of motion was recorded. The surface of the discs were subsequently examined with a scanning electron microscope. Damage to the surface of the porcine disc consisted of cracking principally in an antero-posterior direction. For both the static and impulsive loads, a threshold of approximately 150 N needed to be surpassed before there was evidence of surface damage. A conservative theoretical estimate of human condylar neck strength is 485 N. Given the similarity of the human and porcine TMJ discs, it is confidently predicted that a traumatic blow will cause surface disruption of the human disc prior to condylar neck fracture. The area of cracking on the disc surface was found to be the most reliable indicator of surface damage. An increase in static friction was related to the area of cracking of the disc surface. These results provide support for the hypothesis connecting non-fracture producing trauma to subsequent CMD experience.

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LIST OF ABBREVIATIONS USED

CMD	craniomandibular disorders
GAG's	glycosaminoglycans
H&E	haematoxylin and eosin
kD	kilodalton
kV	kilovolt
LM	light microscope
MPa	megapascal
mV	millivolt
nm	nanometre
r^2	squared value of the correlation coefficient
SEM	scanning electron microscope
SI	Systéme International
TMJ	temporomandibular joint
μ_s	coefficient of static friction

1 INTRODUCTION TO THE PROBLEM

The human temporomandibular joint (TMJ) is a somewhat unusual articulation. In an evolutionary sense it is a new acquisition by the order of mammals; the primitive jaw joint of the higher reptiles being modified to form part of the mammalian middle ear. Human foetal development mirrors this phylogeny with the initial development of a reptilian type joint, the development of which is soon redirected to the formation of two of the three auditory ossicles. The definitive TMJ subsequently forms. As a result of this circuitous developmental route it is not surprising that the TMJ appears late in development in comparison with other joints.

Rather than being covered with hyaline cartilage as most synovial joint surfaces are, the joint surfaces of the TMJ are covered with a fibrous connective tissue. Some early investigators ascribed inferior mechanical properties to this type of fibrous connective tissue covering and used this assumption to argue the TMJ would not be loaded during function. It is now generally accepted that the TMJ is a loaded joint (Hylander, 1975; Smith *et al.*, 1986), and experiences stresses at least as great as the hip joint (Nickel, 1992). It has been claimed that fibrous connective tissue is able to better resist shear stresses (compared to hyaline cartilage) and could explain this tissues appearance in the TMJ (Moss, 1966). It seems more likely that the fibrous connective tissue is a direct result of the unusual embryology of this joint rather than a functional adaptation (Mohl, 1983).

A further unusual feature is that of a disc of fibrous connective tissue that divides the TMJ into two normally separate compartments[1]. Little is known of the biology and biomechanics of the disc. The central, thin, loaded portion of the disc is avascular, poorly innervated, and relatively acellular. The low cellularity and avascularity limit the disc's reparative ability[2]. Acknowledging the disc's low cell turnover and limited repair potential, is a necessary precursor to any consideration of the effects of damage to the disc.

1.1 Synovial Joint Lubrication

One mechanism which would contribute to the maintenance of disc integrity is the minimisation of articular surface wear. This occurs by reduction of the friction associated with joint motion to inconsequential levels. Synovial joints are known to have extremely slippery surfaces (The degree of slipperiness being at least an order of magnitude better than anything man-made)(McCutchen, 1962). However, of greater import, and apparently crucial to joint function is the extremely low friction occurring at commencement of movement. Coefficient of static friction and coefficient of stiction are measures used to quantify the frictional resistance to commencement of movement of an object. These terms will be used throughout the

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[1] The reader is referred to Appendix A for a review of the anatomy and function of the TMJ.

[2] For details of TMJ disc repair and maintenance, see Appendix B.

text and are defined in the following footnote[3]. The coefficient of static friction (μ_s) of synovial joints is between 0.003 and 0.007. As a comparison the slipperiest of man-made materials (polytetrafluoroethylene[4]) has a μ_s of 0.04 when rubbed against itself.

This feature of low coefficient of static friction is quite the opposite of all but the most complex of man-made bearings. In simply lubricated bearings, high friction at the commencement of motion is due to direct bearing surface to surface contact. It is only when a threshold operating speed is attained that an entrained film of lubricant maintains separation of the bearing surfaces, resulting in decreased friction and wear. The high friction level at the commencement of motion is largely responsible for any damage of the surfaces and the majority of resultant wear. If such large start-up friction was seen in synovial joints, degenerative joint disease would become a major problem in the paediatric population (McCutchen, 1983).[5]

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[3] If a body is resting on a flat surface, the weight of the object (W) will represent the force of the object acting on the flat surface. A second force (P) parallel to the flat surface acts on this body. Coefficient of static friction (μ_s) represents the maximum value of P with the body at rest, divided by W. [$\mu_s = P_{max}/W$]. Coefficient of stiction is nearly synonymous to coefficient of static friction. Coefficient of stiction is experimentally determined and represents the largest force recorded just prior to motion. As the incremental increases in force (P) tend to zero, coefficient of stiction will increase and tend to coefficient of static friction.

[4]Teflon (Du Pont Chemicals)

[5] Various hypotheses have been proposed to explain the tribology of synovial joints and are discussed in Appendix C.

Aiding in joint lubrication has been proposed as part of the functional role of intra-articular discs including the TMJ disc[6]. Using the porcine TMJ disc, Nickel (1992) reported an initial coefficient of stiction of approximately 0.005. This extraordinarily low value would seem to imply functional specialisation, and would support the contention that lubrication is an important functional role of the TMJ disc (Nickel,1992).

If the lubrication mechanism is disrupted, it is conceivable that joint function, ultimately may be affected. Decreased lubrication will give rise to increased friction within the joint. The long-term consequence of this is likely to be wear, resulting in disruption of the articular surface. This premature wear in conjunction with the development of articular pain represent the beginnings of osteoarthritic changes. With any change in joint form, there exists the likelihood that change in function, possibly dysfunction will also occur. Dysfunction of the TMJ falls within the umbrella classification of craniomandibular disorders (CMD). Therefore, the hypothetical connection of compromised joint lubrication and CMD is made. The purpose of the two subsequent sections is firstly, to report on the likely role of trauma in the aetiology of CMD and secondly, to discuss the potential effects of trauma on the TMJ disc.

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[6] An account of the proposed function of the TMJ disc has been included in appendix D.

1.2 Trauma and Craniomandibular Disorders

Craniomandibular disorders (CMD) are a group of clinical problems involving either or both; the masticatory muscles and the temporomandibular joint. These disorders are considered a major cause of non-dental pain in the oro-facial region and to be a subclassification of musculoskeletal disorders (Bell, 1989)[7]. Many factors appear to be related to CMD, although it is unknown whether each of these is aetiological, consequential or both (Clark, 1991). McNeill (1990) lists the apparently important factors as predisposing, initiating or perpetuating to acknowledge this void in current understanding of these conditions.

The role of trauma in the aetiology and progression of CMD is frequently mentioned in the literature. Zarb and Speck (1979) state "The common denominator in virtually all cases of dysfunction[8] is trauma". Supporting this notion are studies which indicate that a history of head and neck injury are a frequent finding in patients with CMD (Greene *et al.*, 1969; Carlsson *et al.*, 1979; Pullinger *et al.*, 1985; Helkimo and Westling, 1987; Pullinger and Seligman, 1991). The concurrent reporting of signs and symptoms of CMD and a positive history of trauma vary between 22% (Greene *et al.*, 1969) and 79% (Pullinger and Seligman, 1991). Pullinger *et al.* (1985) demonstrated significantly increased trauma experience in a CMD patient population when compared to an asymptomatic non-patient group. All

[7] The interested reader is referred to McNeill (1990) for further information and citations on this topic.

[8] Craniomandibular disorders (CMD)

this information would indicate a positive trauma history correlated to signs and symptoms of CMD.

Speculation connecting the traumatic event to subsequent CMD rely upon incomplete resolution of the effects of trauma (joint laxity, hyper-mobility, or mechanical derangement), seemingly inappropriate repair (fibrillar organisation subsequent to synovitis) or a precipitating event to escalate an unstable asymptomatic condition into a clinical entity (Tallents, 1991).

In many cases, trauma is cited as the precipitating event for CMD (Harkins and Marteney, 1985). This would indicate a short time period between the traumatic episode and the onset of symptoms. The immediate mechanical derangement hypotheses (capsular stretching, disc derangement) and the precipitating event hypothesis would be favoured by proponents of this mechanism.

In an unknown percentage of cases, the insidious onset of symptoms appears to follow a variable period of latency (Kreutziger and Mahan, 1975; Pullinger and Monteiro, 1988). Of the hypotheses advanced to date, only the progressive adhesion hypothesis (progressive fibrillar organisation subsequent to synovitis) could explain this phenomenon (Sanders and Buoncristiani, 1989; Jones and van Sickels, 1991). It is worthwhile noting that any association between trauma and CMD involving a period of latency, would be under-reported as trauma history may not be accurately recalled by the patient (Hohmann *et al.*, 1983; Pullinger and Seligman, 1991).

1.3 Potential Effects of Trauma on the Temporomandibular Joint Disc

The characteristics of latency and insidious progression, are also characteristics of a fatigue failure mechanism. Therefore it is suggested that fatigue failure may be another mechanism by which trauma may cause CMD. One way that this may occur is by decreasing the effectiveness of the lubrication of the joint. By compromising the lubrication mechanism of the joint surfaces, increased wear is likely to occur, resulting in surface abrasion and roughness. Since the repair and remodelling characteristics of the disc are considered to be poor, surface renewal is not expected. Thus the process would appear to be slowly progressive with the potential failure of the surface due to a fatigue-like mechanism.

The only hypotheses which explain the low stiction (weeping lubrication and boosted lubrication[9]) rely on an intact surface layer. If trauma resulted in microscopic damage to the articular surfaces, it would seem plausible that localised derangement of the lubrication mechanism would occur. Coupling a compromised lubrication status with the limited repair potential of the disc, it is not difficult to extrapolate and suggest that the disc surface would progressively abrade and roughen.

The possibility of disruption to the deeper layers of the disc, without damage to the surface as a result of traumatic loading exists. This type of damage would be difficult to detect and could change the physical properties of the disc with regard to

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[9] See review of joint lubrication in appendix C.

load bearing and lubrication. No information is available on the location of damage as a result of trauma to the disc.

For either of these suggestions to be possible, the bones associated with the joint would need to be strong enough to transmit a force sufficient to damage to the joint surface. The weakest part of the mandible is the condylar neck (Hylander, 1975). If the neck of the mandibular condyle fails at loads below that required to damage the TMJ disc, then the disc would be protected, and its function unaffected. The neck of the mandibular condyle would then be acting as a mechanical fuse.

Theoretical figures on the strength of the condylar neck are available. Hylander (1975) calculated the minimum strength of the condylar neck of the human mandible in both shear and bending. He reported the mandibular condyle could resist a minimum of 1,700 N when applied as a shearing force. In bending, the minimal force which could produce failure would be a force of 485 N applied at the condyle (Hylander,1975). No figures are available for the strength of the TMJ disc.

1.4 Statement of the Problem

If it is possible to damage the TMJ disc at loads below the fracture strength of the condylar neck, a method exists by which trauma could cause long-term fatigue type failure of the TMJ disc and the opposing articular surfaces. Therefore, the principal purpose of this project was to determine whether the disc could be damaged using traumatic loading at a level below that required to fracture the condylar neck. If this was possible, then a number of other important areas of interest arise.

The nature and location of the damage due to traumatic loading of the disc is unknown. The consequences of damage location have implications in the lubrication mechanism of the joint. The behaviour of the disc under different loading conditions is unknown and worthy of study. The relative importance of the duration, peak force, rate of change of force and energy dissipated during a traumatic incident are unknown. These areas form the basis for this study.

2 MATERIALS AND METHODS

2.1 Porcine TMJ Disc

It is obviously more meaningful to draw conclusions about the function of the human TMJ disc from experiments using this structure from humans. However, the impracticality of obtaining a supply of fresh, healthy, human TMJ discs led this experimenter to use an animal model.

The pig was selected as the most appropriate model for a number of reasons. Similarity between human and porcine joint function has been demonstrated (Herring, 1976). As well, Ström *et al.* (1986) have shown likeness in the structure and chemistry of the connective tissue of the TMJ in these two animals. Nickel (1992) has reported a similarity in size and the general morphology; the porcine disc having a thickened posterior and anterior band of fibrous connective tissue and a central thinner area akin the human disc. Fontenot (1985) using tissue fixed in formalin, has reported a similarity for average thickness and the mechanical properties of human and porcine TMJ discs, *viz.* average relaxation time, average relaxed modulus of elasticity.

Pig discs were collected from a local abattoir. Approximately twenty minutes after exsanguination, a point was reached on the production line at which the eviscerated carcasses were decapitated. The pig's head was taken to a nearby table to dissect the disc from the TMJ.

Once the disc was separated from the surrounding tissue (according to the procedure of Nickel (1992)), it was placed in a warmed[10] physiologic saline[11] solution. The discs were then transported to the laboratory for subsequent experimentation. Testing was carried out as soon as practicable following removal of the disc from the pig, generally within 1.5 hours. During this time the discs were maintained in the same solution (see above) at the previously mentioned temperature range.

2.2 Loading Apparatus

Equipment was designed and constructed to load the porcine TMJ disc with either a static or impulsive load.

2.2.1 Impulsive Loading

An impulsive load was delivered to the specimen by a hinged hammer which could be dropped from different elevations (see fig. 2.1). When dropped, the hammer would collide with an acrylic indenter resting on the surface of the specimen. Lateral, but not vertical movement of the indenter was prevented by a supporting arm. The specimen rested on a flat piece of steel which had been milled to fit snugly against the top surface of a large block of steel (10 x 10 x 20 cm). The purpose of this large block was to concentrate the energy dissipation of the impulse in the

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[10] $37 \pm 3°$ Centigrade

[11] Krebs-Ringer, bicarbonate buffered (pH 7 - 7.2) salt solution (Sigma Chemical Company, St. Louis, Mo.

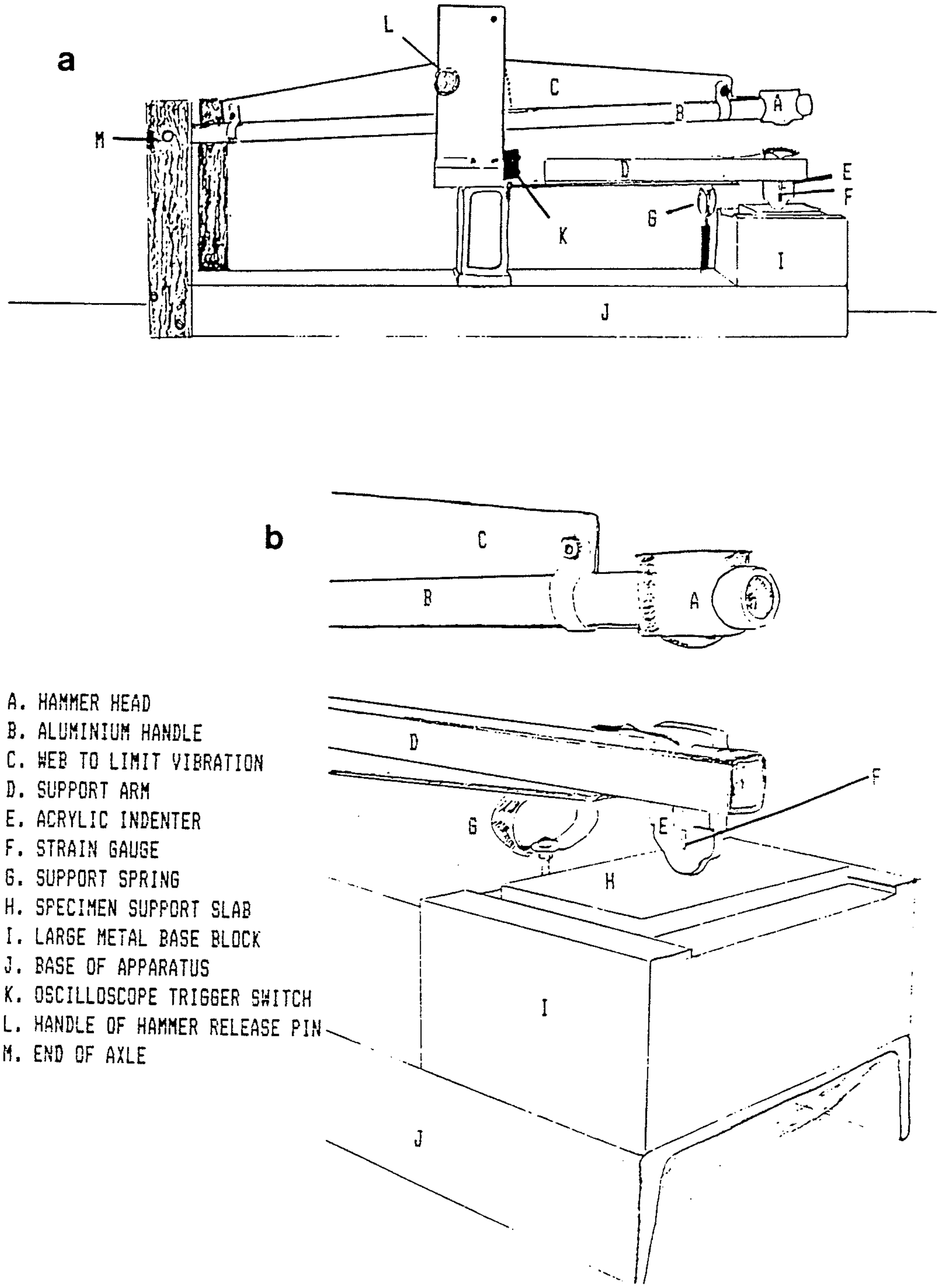

Figure 2.1 Impulse loading apparatus. a) side view, b) oblique view of hammer head and indenter.

specimen. The mathematical relationship between the height through which the hammer is dropped and the magnitude of the impulse is given in the equation below. This demonstrates that increasing the height through which the hammer drops causes an increase in the magnitude of the impulse.

m = mass of hammer head, g = gravitational constant, h = height of hammer, F = force associated with impulse, t = time

$$Impulse = \int_{t_1}^{t_2} F\,dt = m\sqrt{2g(h_2 - h_1)}$$

The indenter when viewed from the side was semi-circular in outline, having a radius of 8 mm. When viewed from a horizontally orthogonal position, the inferior surface of the indenter was also curved but with a larger radius (100 mm). The indenter was 6 mm thick. The dimensions of the indenter were based on the anatomical limitation of the thickened periphery of the disc and the importance of loading the disc at the tip of the indenter. If other parts of the indenter contacted the disc then a proportion of the load would be distributed through these extraneous contacts. This would lead to errors in determining the characteristics of the impulsive load.

A foil strain gauge was luted to each side of the indenter. Using the circuit diagram in figure 2.2, the strain gauges were connected to an oscilloscope. Micro-deformation of the surface of the indenter results in a measurable electrical resistance

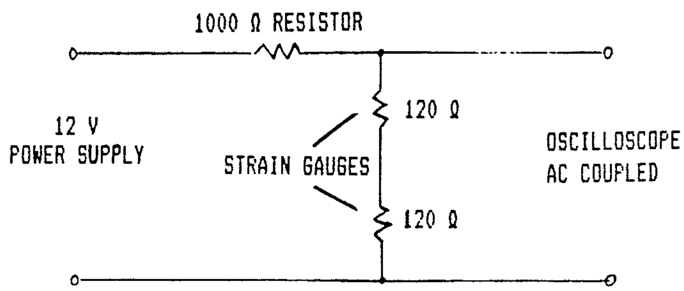

Figure 2.2 Strain gauge circuit diagram

change in the strain gauge. The indenter could thus be calibrated by imposing a known force and measuring the corresponding change in the electrical resistance. Hence, the oscilloscope recorded the changing magnitude of force acting on the indenter during the impulsive load.

The brass hammer was connected to an aluminium handle. At the opposite end of the handle, a cylinder of mild steel was snugly fitted to form an axle. The axle passed through a pair of plain bearings, one at each end of the axle. The construction of the bearings, together with the length of the handle, reduced frictional losses to inconsequential proportions. The long hammer handle also ensured that the hammer had negligible horizontal motion at impact. In addition, it was desired that the mass of the handle be small so that the mass of the hammer head was always dominant. To counteract the effect of vertical vibration in the handle, a thin aluminium web was attached to the upper surface of the handle.

The hammer could be held at various heights above the specimen by a supporting peg. When the peg was removed the hammer would fall toward the specimen. Shortly before contacting the indenter, the handle of the hammer would activate an electrical switch, triggering the oscilloscope trace which recorded the characteristics of the impulsive load delivered to the specimen.

2.2.1.1 Impulse Parameters

Three parameters are useful to describe an impulse (see fig 2.3). The area under the force-time curve is the impulse magnitude (the middle expression of the equation given above) for which the SI units are Newton·seconds. The peak force is the maximum force value achieved by the force-time function. However knowing these two features alone does not provide any information about the rate of change of force. The latter feature can be described using an indicator of kurtosis or

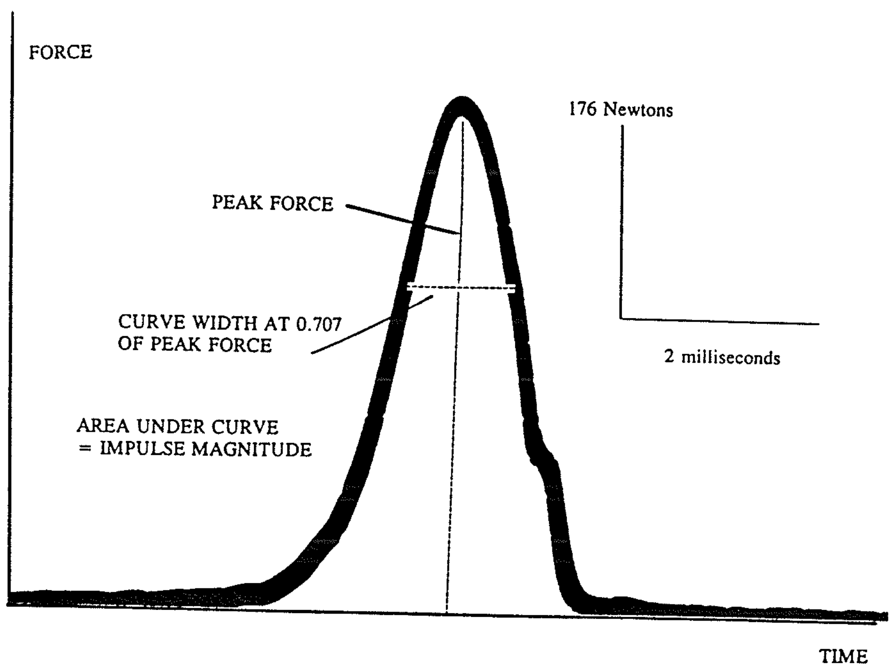

Figure 2.3 Sample impulse plotted on force/time axes

peakedness. The peakedness of the impulse was quantified using the quotient of the plotted height of the peak force and the curve width at 0.707 of the peak force[12].

2.2.2 Static Loading

The same equipment was used, though in a modified manner, to load the disc with a fixed force. The hammer was lowered onto the indenter with the disc in position. Appropriate weights were gently positioned on the hammer (depending on the load, a few seconds were required to position the individual weights). The total static load was left in position for 3 seconds. After this stage they were removed and the hammer lifted off the indenter.

2.3 Experimental Procedure

At the time of the experiment, the pig disc was removed from the warmed saline bath and placed superior surface upward on the platform of the apparatus. The indenter was then gently lowered onto the surface of the disc. Stable contact between indenter and disc was assessed using mild finger pressure on the indenter. The contact was judged to be stable if the disc did not move upon application of gentle pressure. This was important to achieve as movement of the disc at the time of

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[12] 0.707 is the inverse of the square root of 2, and represents a point on a resonance curve at which power drops to 50% of peak power. In this case it represented a somewhat arbitrary, but consistent, choice by which to quantify the shape of the curves. The peakedness value was calculated by making measurements from enlargements of tracings on acetate that were made directly from the oscilloscope screen. The oscilloscope settings were kept constant throughout the experiment. Thus a low flat curve would have a low peakedness value and conversely a sharp pointed curve would have a high peakedness value.

impact would give rise to uncertainty in both trauma location and magnitude. Once the disc was assessed as being in a stable position, the hammer was dropped from a pre-determined height, impulsively loading the disc.

2.3.1 Histological Evaluation of Damage Location

Since the effects of impact and static loading upon the disc are unknown, the aim of this part of the study was to quantify two unknowns. Firstly was damage due to the loading, superficial, deep within the disc or a combination of both. Secondly was a difference in damage pattern noted when statically and impulsively loaded specimens were compared.

The background to the methodology employed in this series of experiments is presented in appendix F. It involves the histological observation that collagen stains differently with a number of chemicals following strain. It was anticipated this phenomenon could be used as a "tension probe" for collagen in the test specimen.

The porcine discs were collected and stored using the method described in section 2.1. Four discs served as control specimens. Twelve discs were impact damaged with an acrylic indenter applied to the condylar surface of the disc at a range of loads up to 850 N peak force. India ink marks were then tattooed onto the disc anterior and posterior to the site of impact to allow preparation of the appropriate part of the disc for sectioning. The discs were fixed in 10% neutral buffered formalin for at least 24 hours. Prior to processing and embedding in paraffin, tissue blocks were prepared. Sectioning was in either an antero-posterior or medio-lateral direction.

Depending on the ultimate plane of section, the damage location was equally divided, resulting in two tissue blocks per damage location. The part of the tissue block representing the region closest to the location of damage was marked and positioned so that it would be the first area sectioned.

Sections were cut at 6 μm on a rotary microtome, mounted on glass slides and stained with either haematoxylin and eosin (H&E), Masson's trichrome (including variations thereof) or Mallory's trichrome. At this preliminary stage, sufficient sections were cut to test if the methodology was appropriate to the aims of the study. If it had proved to be scientifically rigorous, sections would have been cut serially so that a map of the damaged area could be constructed. It was proposed that a computer image analysis system could be used to quantify the distribution of colour and intensity of stain intensity. For this to be valid, and comparable between sections, the section thickness had to be identical as the transmission of light decreases with increasing thickness of section.

Significant sectioning difficulty was encountered and was ascribed to the highly fibrous nature of the disc. This was particularly evident in the long medio-lateral sections which were cut from some of the control discs. Sectioning was made simpler by cooling the blocks for 1 hour in the freezer compartment of a standard domestic refrigerator whilst soaking them in a glycerine/alcohol aqueous solution[13]. However, warming of the blocks to room temperature during sectioning resulted in

[13]10% glycerine, 54% alcohol

variation in section thickness. This could be easily assessed by observing the difference in translucence of the wax surrounding the tissue section.

The H&E sections were used as screening slides for preliminary examination. At least two of these slides were made of each damaged area and each control disc. The resulting slides demonstrated sections cut in both antero-posterior and medio-lateral planes.

In addition to Masson's trichrome, a number of variations of this staining method were subsequently employed, the purpose of which was to improve consistency and intensity of staining of the "traumatised" collagen. These variations included modifications of Masson's trichrome and Mallory's trichrome, varying the length of time the unstained sections were left on the slide prior to staining, presoaking the tissue in different mordants (1% acetic acid or Bouins solution) and varying the temperature (room temperature or 56° Centigrade) and time for pre-soaking (1 hour or overnight).

2.3.2 Evaluation of Damage Location

The effects of trauma on the surface of the disc are unknown. The obvious starting point is to determine whether it is possible to damage the surface of the disc. If trauma effects were visible on the surface, methodology needed to be developed by which this could be quantified. This method could then be applied to a range of both static and impulsive loads, to investigate whether "amount" and type of trauma are related to "amount" of surface damage.

Using the equipment previously described, the pig disc was experimentally traumatised. Following the experimental trauma, the disc was allowed to soak in physiological buffered saline solution for at least 20 minutes to allow for lost fluid to be re-imbibed. The trauma location was marked using 4 india ink points, tattooed at the corners of an imaginary square surrounding the loaded area.

The scanning electron microscope (SEM) was chosen as the most appropriate tool to image the surface of the disc and therefore, in preparation for viewing in the SEM, the pig material was placed in 10% neutral buffered formalin for at least 24 hours. The tissue was subsequently trimmed to a suitable size and processed through graded alcohols, critically point dried[14] using liquid CO_2 and mounted on aluminium stubs using silver paint[15]. A desiccator under vacuum in the presence of anhydrous calcium sulphate (Drierite[16]) was used to protect the specimens from water contamination and allow evaporation of the solvent of the silver paint (minimum 24 hours duration). The specimens were then sputter coated[17] with gold palladium for 2.5 minutes at 10 mA. This resulted in a coating of approximately 12.5 nm in

[14] Bomar SPC-1500 critical point dryer; The Bomar Company, Tacoma, Washington, USA

[15] High purity silver paint, St. Laurent (Montreal), Quebec

[16] Mallinckdrodt Specialty Chemicals Canada Inc., Mississauga, Ontario

[17] Hummer V; Technics

thickness. Examination under a scanning electron microscope[18] using a variety of accelerating voltages between 15 and 25 kV was undertaken.

The discs tended to warp when dried in preparation for viewing in the SEM. The direction of warpage generally involved the anterior and posterior thickened portions of the disc coming closer together. This created a more concave specimen surface, with the centre of the damage in the central portion of this concavity. The greatest angle of curvature measured was never greater than 15° in each direction. In setting the specimens for viewing in the SEM, care was taken to position the specimen such that the plane of the central portion of the damaged area was as close to perpendicular to the electron beam as possible.

If the curvature of the surface was under-estimated by as much as 25% then a maximum angulation of 20° would be present at the extremity of the specimen. When photographed, the cracking in the maximally angulated peripheral portion of the specimen would be under-represented due to the surface angulation. However using trigonometry, the maximal under-representation would range from 0% in the central area to 5% at the periphery. The damage tended to be more centrally located, and thus on a less angulated surface. Therefore the maximum error due to image distortion was considered to be no greater than 2.5%. This was considered to be inconsequential to the overall experimental results.

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[18]Jeol JSM-35C; Jeol Ltd., Tokyo, Japan

The specimens were photographed using either Ilford HP5 negative roll film or a direct positive Polaroid HP52 film. Enlargements (8 x 10 inches) which included the magnification reference bar, were made from the negatives.

In order to quantify the surface damage, both area and length of cracking of the surface of the disc were measured. This was achieved by using a digitising tablet linked to a computer that was running Sigmascan[19] software. The system was calibrated in millimetres using a standard square (10 cm x 10 cm). The photographs were taped to the surface of a digitising tablet. Using the electronic pointer, the outline of the individual cracks or the length of the cracks could be traced on the digitising tablet. The computer was then able to calculate a value which represented the summated area or summated length of individual cracks represented on the photograph. This was scaled to disc surface measurements using the bar scale on the photo-electron micrographs.

2.3.3 Evaluation of Stiction Change

To investigate the effect of trauma on the lubrication properties of the disc, it was necessary to be able to compare the "lubrication status" of the disc prior to and following the induced experimental trauma. The remarkably low friction upon commencement of movement is the feature of synovial joints which separates them from most mechanical bearings. This particular feature of lubrication (termed

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[19] Jandel Scientific, Corte Madera, California

coefficient of stiction[20]) seemed the most relevant parameter to observe and was measured using a previously constructed piece of apparatus (Nickel, 1992). A precis of design considerations and specifications of this apparatus[21] is included in appendix G.

This set of experiments include the same methodology as the traumatic loading experiments, but included the extra steps of measuring the coefficient of stiction prior to and after the experimentally applied trauma. This is described in the following paragraphs.

Prior to experimentally applied trauma, the stiction value of the surface of the disc was recorded with respect to time. This was done by placing the disc on the support base of the stiction measuring instrument. The pendulum was gently lowered onto the surface of the disc. Once the mass of the pendulum was supported by the disc, a clock was started to record elapsed time, and a tape recorder device recorded the deflections of the pendulum. At no stage throughout the experiment is the mass of the pendulum lifted from the disc. Once a stable position on the surface of the disc is located by the pendulum and the equipment set to zero, the experimental recordings were begun. The pendulum was then deflected maximally. By gently lowering the

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[20]Coefficient of stiction and coefficient of static friction are nearly synonymous. The stiction coefficient is calculated using the force just prior to movement. The coefficient of static friction is calculated using an infinitesimally larger force at which motion commences.

[21] Throughout these experiments, the mass of the pendulum used in the friction experiments was constant and resulted in a force of 4 N being continuously applied to the disc.

support on one side of the pendulum, a point was reached at which the pendulum lost contact with the support. This indicated that the weight of the pendulum was supported by the friction between the indenter and disc surface. This deflection was recorded and converted into a coefficient of stiction measurement. The deflection was repeated in opposite directions until the friction levels stabilise or 10 minutes of experimental time had elapsed.

The disc was then returned to the warmed physiological saline for at least 30 minutes and allowed to re-imbibe fluid lost during the initial stiction evaluation and return the disc to its original state.

Trauma in the form of a static or impulsive load was then applied to the disc. Again, the disc was returned to the warmed physiologic saline solution for at least 30 minutes. The time period of 30 minutes was selected based on a report by Nickel (1992). He has demonstrated the reproducibility of repeated stiction recordings provided a period of 30 minutes is allowed for re-soaking of the disc.

Following this, another series of stiction recordings were made with respect to time. The disc was then returned to the warmed saline for another 30 minutes. The area surrounding the traumatised portion of the disc was tattooed with India ink. The discs were placed in 10% neutral buffered formalin, in preparation for scanning electron microscopic examination.

The result of the pre- and post-trauma stiction recordings is a plot of stiction with respect to time. A curve of best fit was made for each series of points. The stiction levels at 200 seconds were used to produce a ratio of post-trauma to pre-trauma stiction levels (The equation for this is presented below).

$$Stiction\ Ratio = \frac{post\text{-}impulse\ stiction\ level\ at\ 200\ seconds}{pre\text{-}impulse\ stiction\ level\ at\ 200\ seconds}$$

The criteria for selection of this time was a compromise between accuracy confidence in the best-fit curve and attempting to utilise a portion of the curve during which the greatest changes in stiction were taking place. In most cases, following 200 seconds of elapsed experimental time, 2 stiction measurements had normally been made. On a plot of stiction values against time, the best-fit curve was manually drawn. If sufficient variation in recorded values existed, the curve which helped support the null hypothesis (H_0 = No difference exists in lubrication, before or after trauma) was selected. In the case of pre-trauma stiction this would mean selection of a curve with higher values whereas, the opposite was the case for the post-trauma stiction curve (Based on results of Nickel (1992)). This would tend to under-estimate any increase in friction.

3 RESULTS

The results of the experiments described in the previous chapter fall into three categories. The first are those results dealing with light microscopic examination of the experimentally traumatised discs to determine the location of any resulting changes to the disc. These results are apparently independent of imposed trauma and as such will be considered first. The second category deals with the relationship of the impulse parameters when impacting onto the porcine TMJ disc. The third group of results deal with surface damage of the disc, and include findings involving the loading characteristics of the disc with the imposed trauma, stictional changes and results from SEM examination of the discs.

3.1 Light Microscopy

3.1.1 Masson's Trichrome Stained Sections

Normally collagen stains green with Masson's trichrome stain. However, following strain, collagen has been reported to change its stain affinity (see appendix F). The red stain (Ponceau acid fuchsin), expected to highlight the strained collagen area in the traumatised tissue was not seen in any of the sections from the experimental discs including one series of sections from a disc which was obviously grossly traumatised. Some of the undamaged control specimens demonstrated an area of internal redness against a green counter-stained (light green) background. This amount of variation indicates that the reliability of this method as a tension probe for collagen is non-existent. Variation in staining technique produced equally inconclusive results. An unexpected staining pattern resulted when a delay occurred between mounting the tissue on the slide and the staining procedure. A number of sections were mounted on slides but remained unstained for approximately two months. These sections displayed considerable differences when compared to the slides which were stained within two days of being mounted. The tissue of the former group of slides took up the red stain in preference to the green counter-stain. This is the reverse of the staining pattern for the latter group. No attempt at quantitative assessment has been made due to the clear lack of relation between amount of trauma and staining characteristics.

3.1.2 Haematoxylin and Eosin Stained Sections

Two unexpected findings were noted in these sections. Blood vessels were found throughout the pig disc and tended to be distributed near the surface of the disc (see figure 3.1). Though more prominent in the peripheral areas of the disc, blood vessels were detected in the central thin load-bearing area. The presence of fat cells was also noted (see figure 3.2). The numbers detected were small and were principally distributed toward the periphery of the disc, and were frequently found near blood vessels. Neither of these features appears to have been described previously in porcine TMJ discs.

In the medio-lateral sections, the collagen fibres of the disc appeared to be cut perpendicular to their long axis. A wavy collagen fibre arrangement was present in

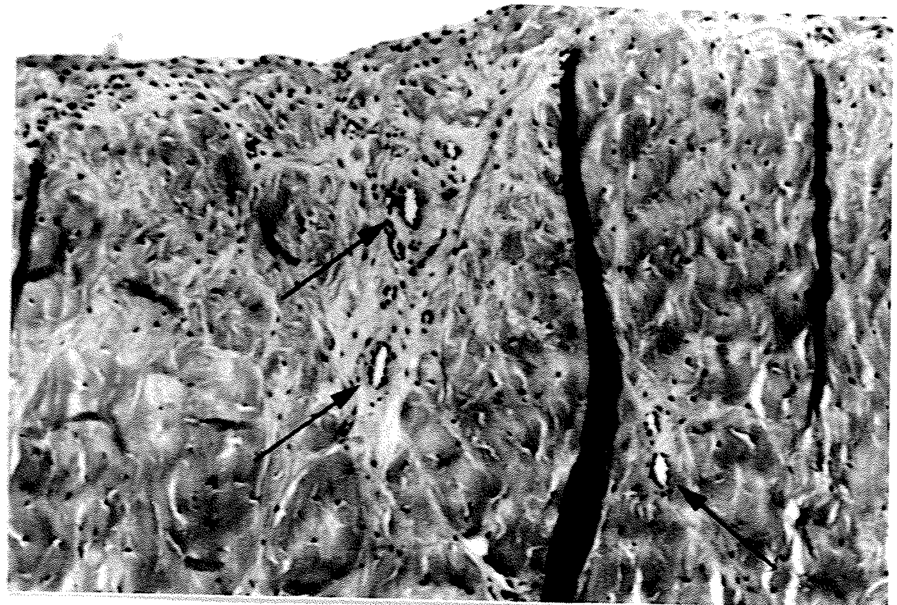

Figure 3.1 Photomicrograph of the medio-lateral section of the pig TMJ disc demonstrating blood vessels (see arrows) (H&E, magnification x200).

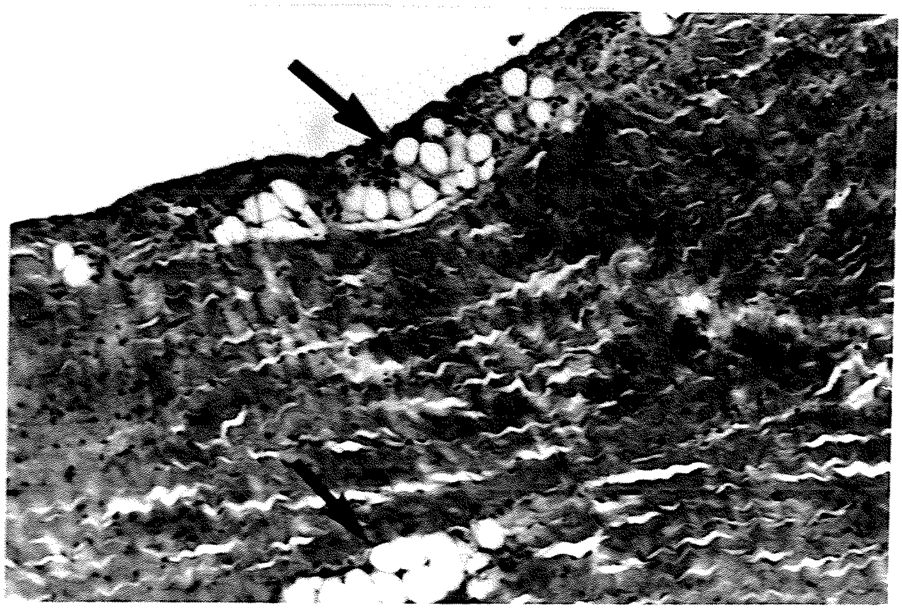

Figure 3.2 Photomicrograph of the antero-posterior section of the pig TMJ disc. The arrows indicate clusters of fat cells. Note the wavy fibre arrangement parallel to the plane of section. (H&E, magnification x200)

the antero-posterior sections. These two observations indicated that the principal fibre orientation was antero-posterior. This agrees with a number of other studies that are mentioned later in the text (see Table D.1 in appendix D).

3.2 Characteristics of Imposed Trauma

3.2.1 Impulsive Loads

The interplay of the three parameters describing an impulsive load (*viz.* peak force, impulse magnitude and peakedness) depend upon the material which is distributing the impulse energy. The variety of impulsive loads in this study was generated by maintaining the mass of the hammer and increasing the height through which it was dropped. The data relating these impulsive load parameters, for this group of porcine TMJ discs, has been plotted in figure 3.3.

As can be seen from the plots, a positive relationship exists between all three parameters. The best relationship is between impulse magnitude and peak force, followed by peak-force and peakedness. A weaker positive relationship exists between impulse magnitude and peakedness.

The positive slope of the peak-force/peakedness and impulse magnitude/peakedness plots demonstrates the changing shape of the impulse with increasing impulse size. This indicates that as the impulse magnitude increases both the peak force attained and the rate of change of force also increase. To uncouple these impulse parameters and investigate the relative importance of each to disc damage, it is necessary to change the relationship of one parameter to another. This

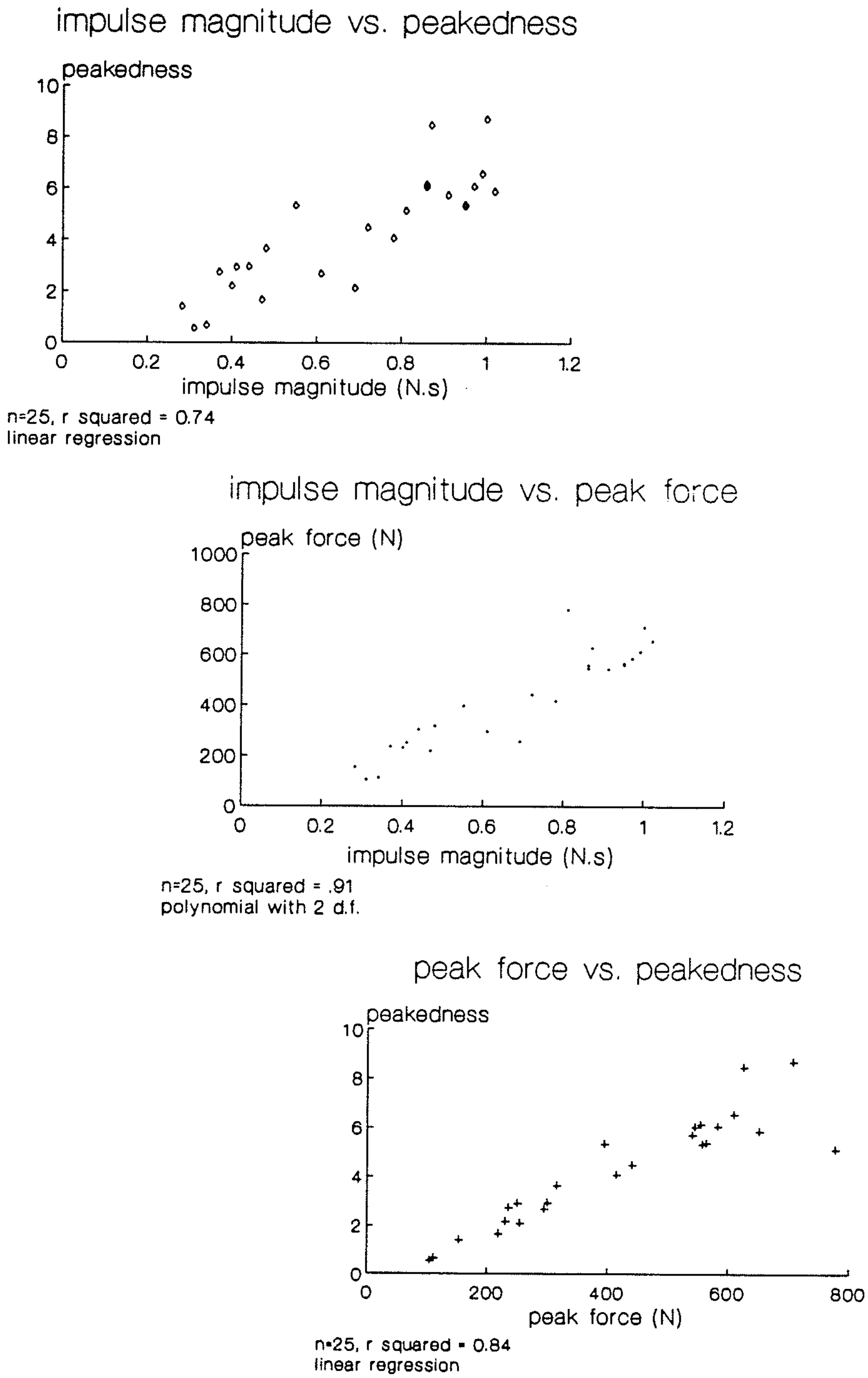

Figure 3.3 Plots relating impulse loading parameters, impulse magnitude, peak force, and peakedness. Note threshold for damage of the disc is peak force = 150 N, impulse magnitude = 0.3 N.s, peakedness = 0.5

will be pursued in more detail in the discussion.

Intuitively the minimum value that the Y intercept of impulse magnitude versus peakedness, and impulse magnitude versus peak force plots is zero. Therefore a second or higher order polynomial would better describe these relationships as linear regression yielded a negative Y intercept in both cases. The fitting of a single regression function to these data may not be appropriate as the disc may have an explainable change from non-linear to linear behaviour with increasing peakedness. On this basis the regression lines have been omitted from the plots. Possible explanations for a change from non-linear to linear behaviour will be discussed later (see discussion section).

3.3 Scanning Electron Microscopy (SEM)

The surface of the undamaged disc at low magnification (x20) revealed a smooth unremarkable surface. At higher magnification (x2000) characteristic undulations were noted (see figure 3.4), which are consistent with previous reports (Jagger, 1980). In some of the specimens, isolated thin tags of tissue arising from the surface of the disc were present (see figure 3.5). This feature was present in both experimental and control specimens. Therefore, its presence was considered unrelated to the experimental trauma.

3.3.1 Quantification of Surface Disruption

The imposed trauma resulted in cracking of the surface of the disc. The "amount" of cracking increased with the "amount" of trauma. In addition, the

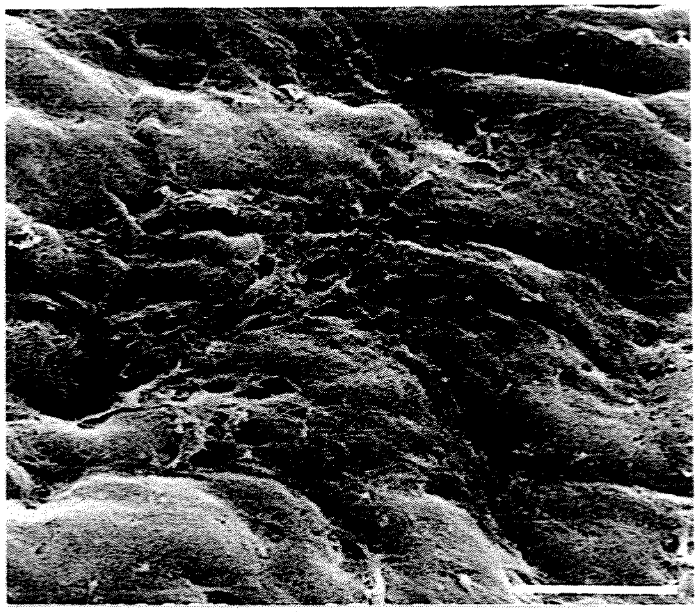

Figure 3.4 SEM photograph of porcine TMJ disc surface, demonstrating undulating surface. Bar scale represents 10μm.

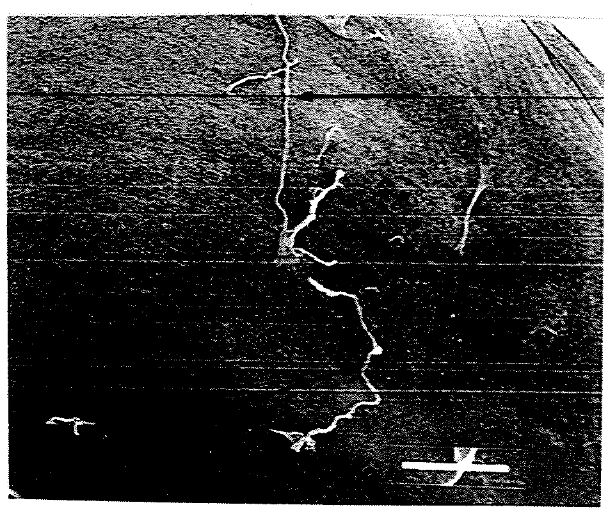

Figure 3.5 SEM photograph of porcine TMJ disc surface, demonstrating isolated tissue tag (indicated with arrow). These appeared on both experimental and control discs. Scale bar represents 1 mm.

orientation of the long axis of the cracking was always in an antero-posterior direction.

Table 3.1

r^2 Values for Relationships Between Damage and Loading Parameters

	Impulse load			Static load
	Peak force	Impulse magnitude	Peakedness	
Area of cracking	0.52	0.61	0.44	0.69
Total crack length	0.46	0.4	0.4	0.62
Average crack width	0.13	0.27	0.21	0.29

To assess the amount of surface disruption, the area, length and mean width of cracking were recorded. The highest correlation always occurred between the loading parameter (*e.g.* static load, peakedness, etc) and area of cracking (see Table 3.1). On this basis, area of cracking was selected as the best descriptor of surface damage.

Two figures demonstrating a range of surface damage have been included, one related to static loading (see figure 3.6); the other related to impulsive loading (see figure 3.7).

The subsequent sections present results relating the "amount" of trauma and its relationship to "amount" of damage. The data presented demonstrates notable

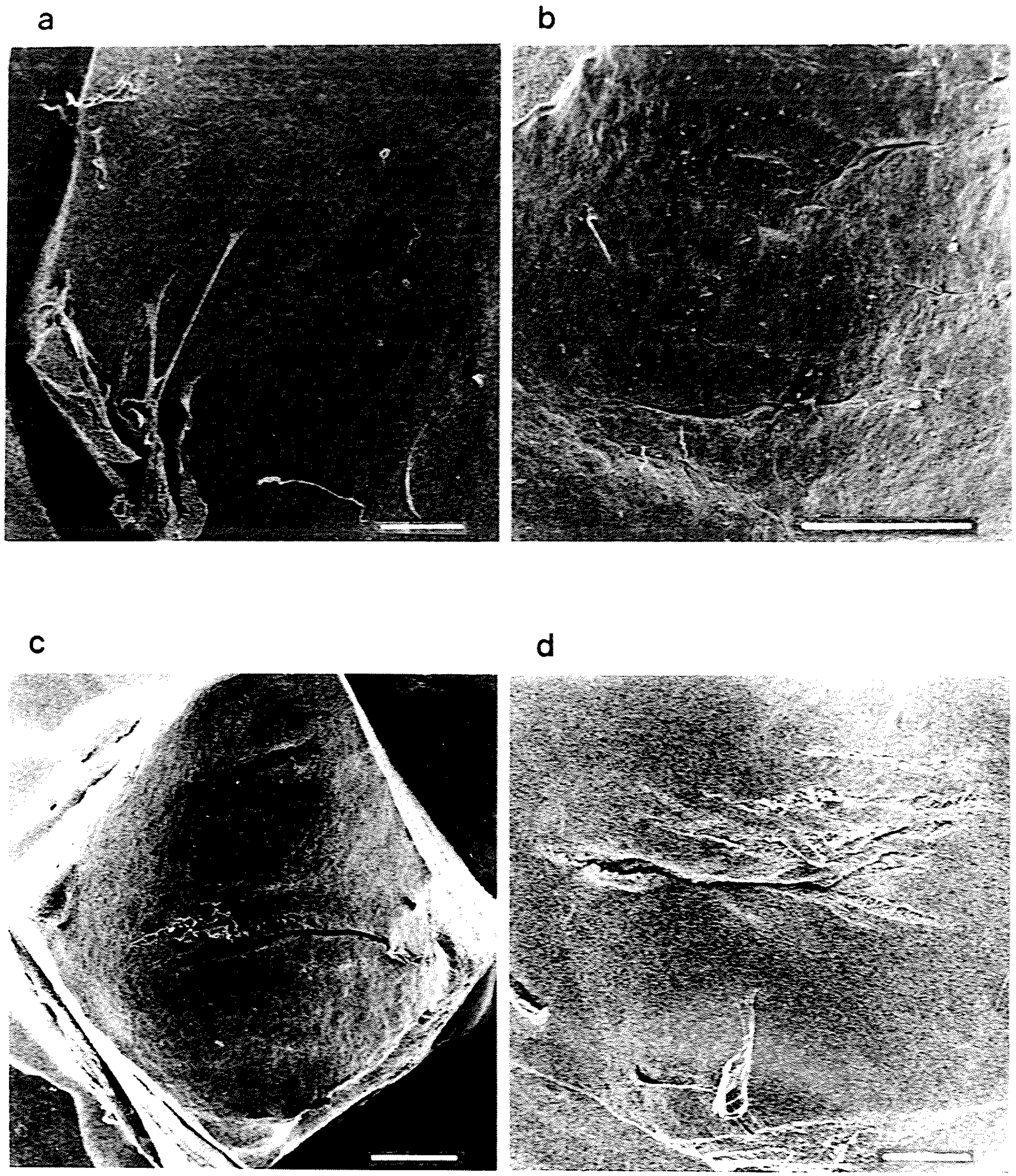

Figure 3.6 SEM photographs of porcine TMJ discs damaged with static loads demonstrating a range of surface cracking. a). normal surface, b). 0.08 mm^2, c). 0.5 mm^2, d). 2.9 mm^2. [Scale bar = 1 mm.]

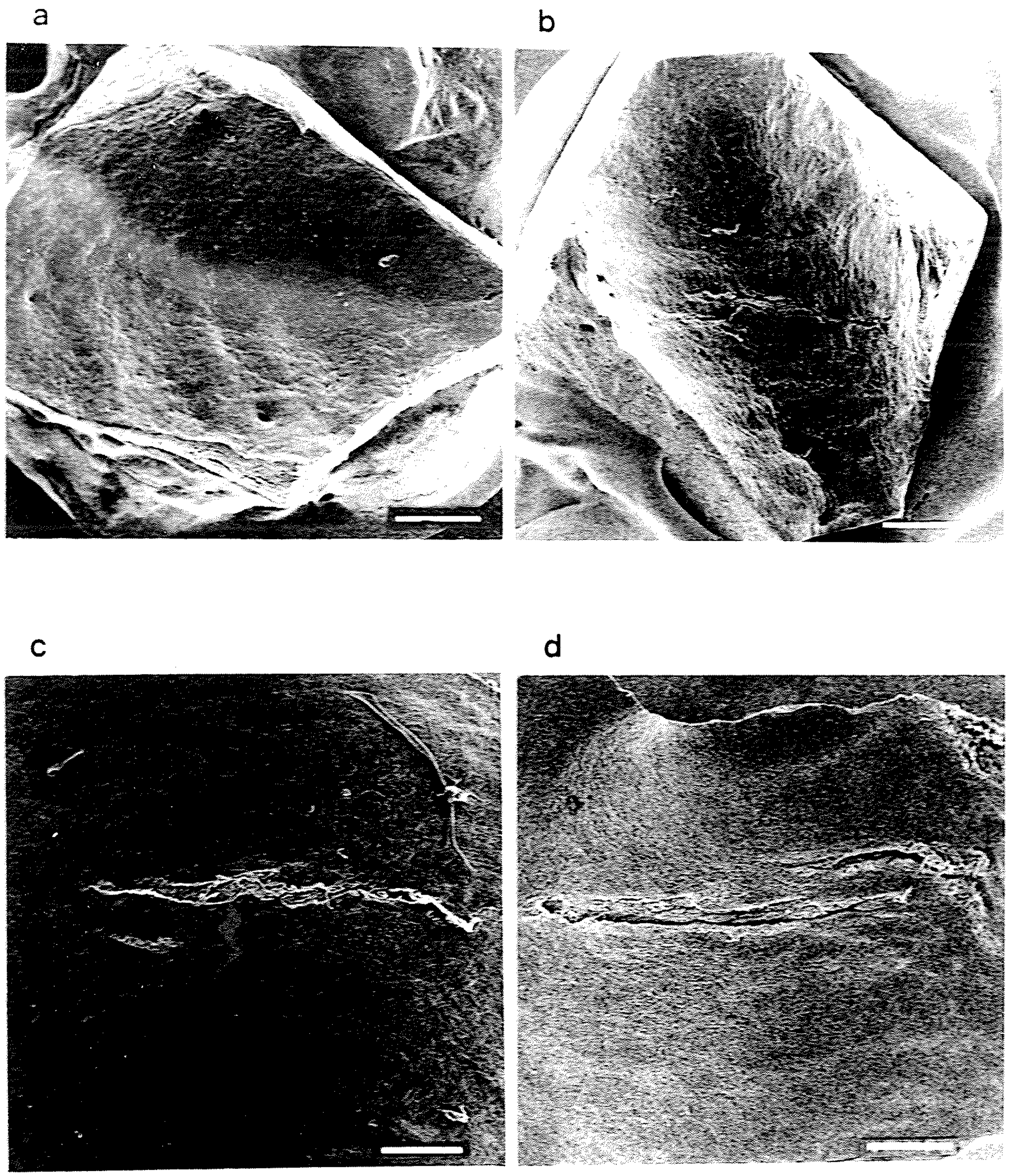

Figure 3.7 SEM photographs of porcine TMJ discs, impulsively damaged, demonstrating a range of surface cracking. a). normal surface, b). 0.31 mm^2, c). 1.37 mm^2, d). 2.47 mm^2. [Scale bar = 1 mm.]

scatter. This is an expected outcome as the cracking is critically dependant on the surface condition of the sample at the time of loading. Minute changes in the surface condition will yield different amounts of cracking. This is discussed further in the next chapter.

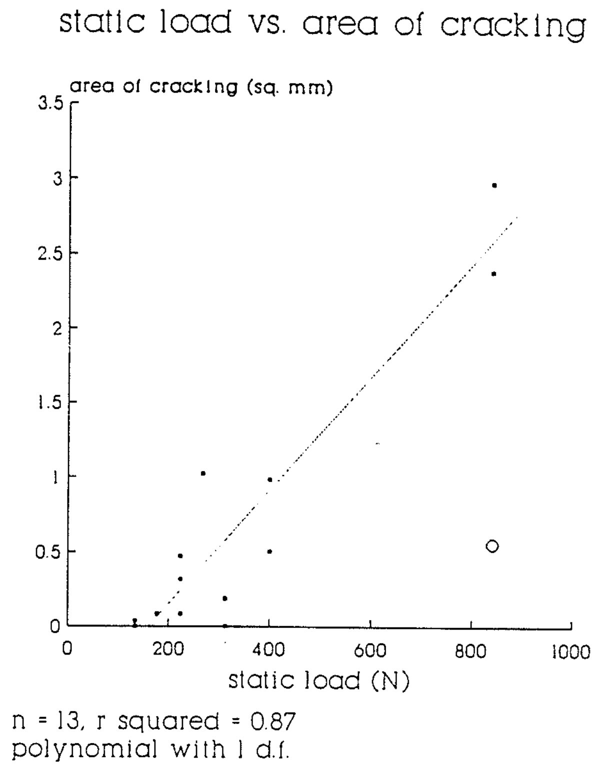

Figure 3.8 Plot relating static loading to resultant area of cracking

3.3.2 Static Loading

Increasing the magnitude of static load is shown to be significantly related to area of cracking (see fig 3.8). The lack of data for force values between 400 and 800 N leaves ground for conjecture as to the nature of the relationship between static load and area of cracking. However it would be difficult to conceive a mechanism by which increasing static load could cause a decreasing amount of surface disruption. The positive X axis intercept indicates a threshold force value of about 150 N below which cracking did not occur. The point represented by a ○ on this plot (fig. 3.8) was considered to be an "outlier" (Wonnacott and Wonnacott, 1977) and as such was left out of the regression analysis. It is believed this result is explainable due to errors in damage location prior to specimen preparation for the SEM. This resulted in some of the damaged area being discarded during specimen preparation leaving a smaller area of damage to be measured.

3.3.3 Impulsive Loading

Plots relating impulse magnitude, peak force and peakedness to area of cracking are included in figure 3.9. These plots all demonstrate a positive relationship between area of cracking and impulse parameters. Worthy of note is the positive value for the X intercept in each case (peak force 150 N, impulse magnitude 0.3 N.s, and a peakedness value of 1). This indicates that a threshold value exists, below which cracking does not occur.

3.3.4 Disc Thickness Related To Surface Damage

For the impulsively loaded specimens, a range of impulse magnitudes have been grouped and plotted, thickness against area of cracking (See figure 3.10). Insufficient data exists to make definitive statements about absolute relations. However, the general trend seems to be increasing disc thickness is related to decreased surface damage within the study groups. One group contains only one point (0.94-1.1 N.s) whilst the remaining group (0.55 - 0.64 N.s) demonstrates increasing area of cracking over a small increase in thickness range.

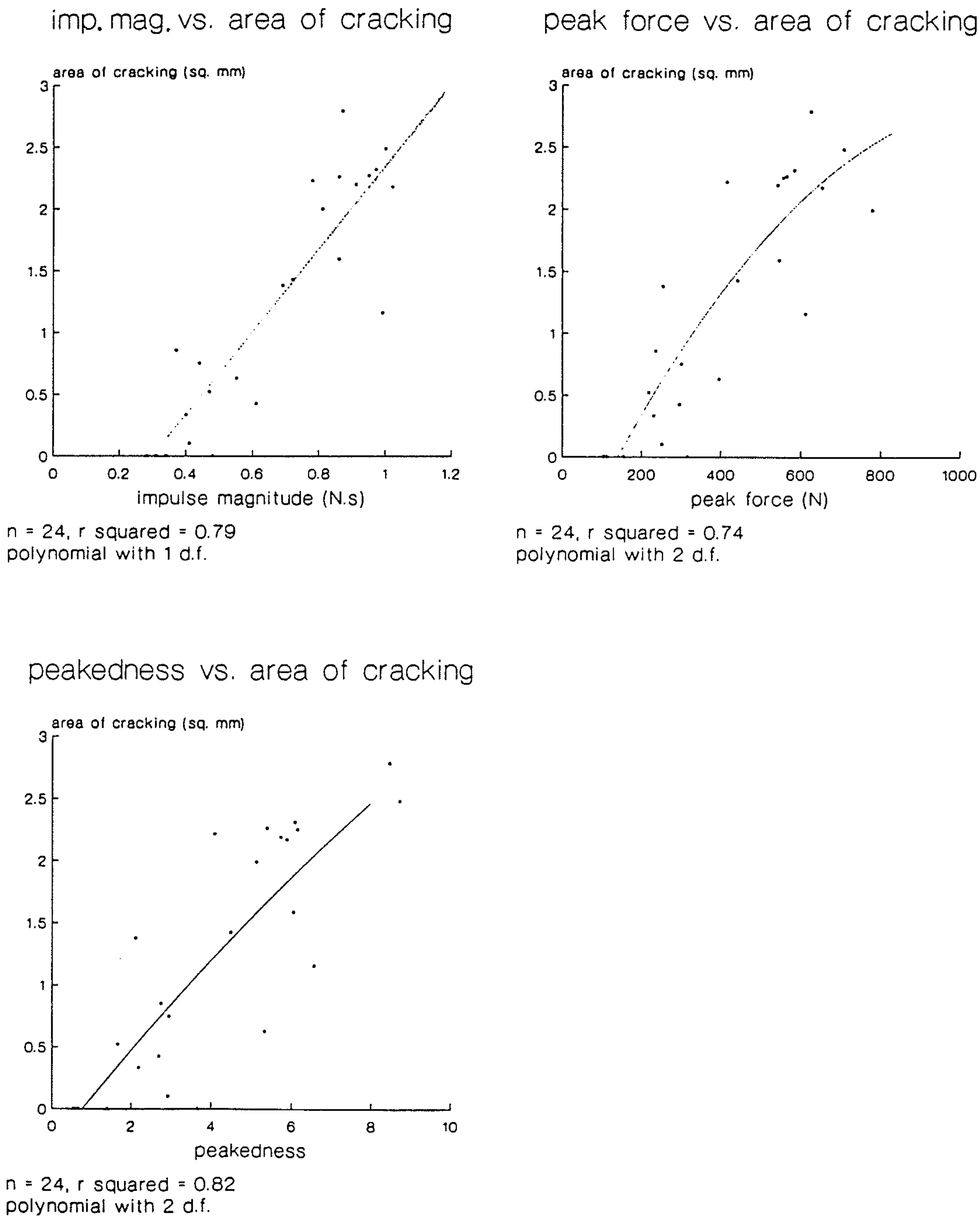

Figure 3.9 Plots of impulsive parameters related to area of cracking.

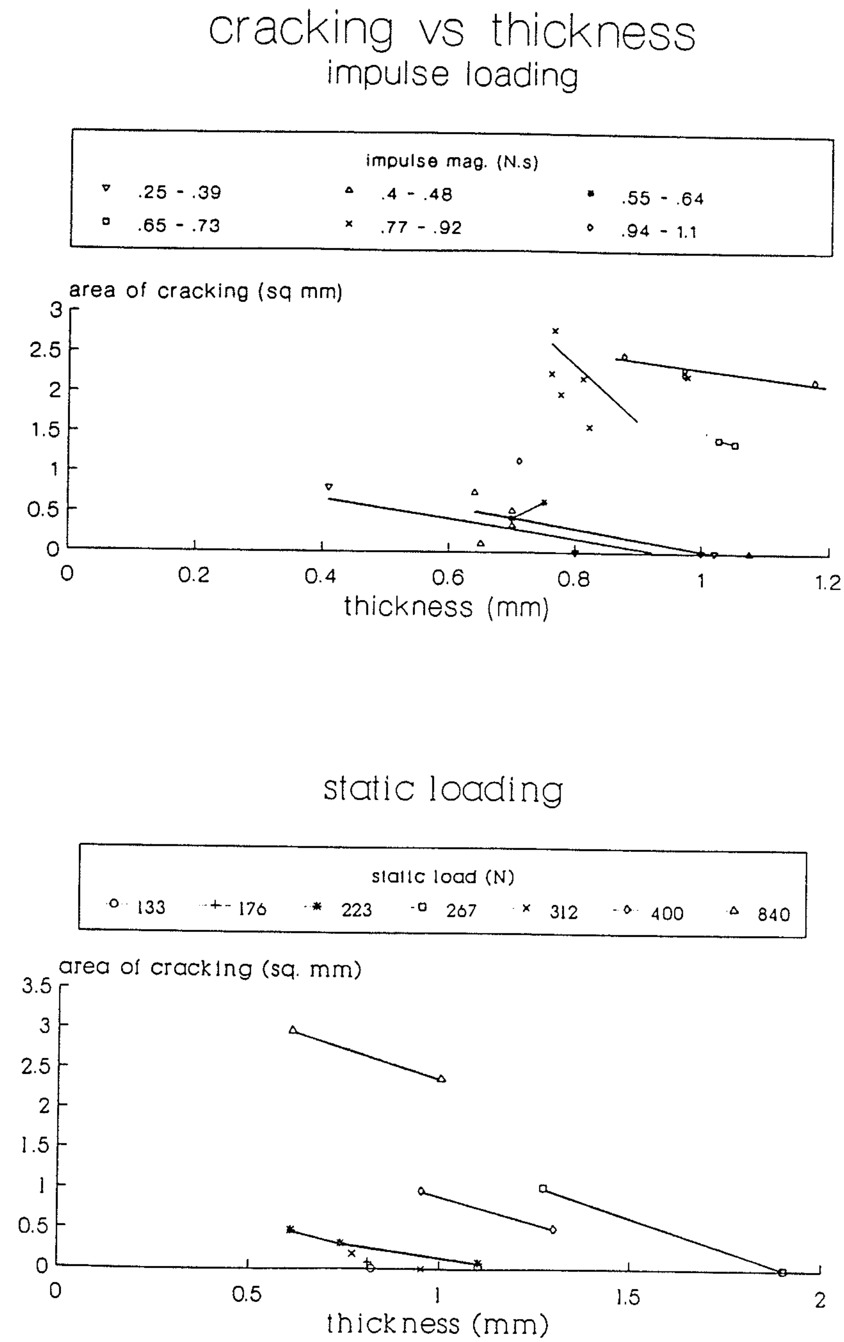

Figure 3.10 Plots relating disc thickness to area of cracking for a). grouped impulsive loads, b). grouped static loads

The statically loaded specimens have been grouped according to actual static load and thickness has been plotted against area of cracking. All groups demonstrate increasing disc thickness is associated with a decreased area of cracking.

3.4 Surface Damage Related to Stiction Changes

3.4.1 Static and Impulsive Loading

Area of cracking has been plotted against stiction ratio[22] (see figure 3.11). Although this data demonstrates scatter, some information can be extracted from these plots. At zero area of cracking in both the static and impulsively loaded specimens, the data points are clustered about a stiction ratio of 1 which indicates no change in stiction as would be expected.

The impulse loading plot (figure 3.11) demonstrates a general increase in stiction with increase in area of cracking. The points marked as x's are considered to be the result of incorrect positioning of the stiction measuring pendulum following the experimental trauma. If these data points are not considered, the linear regression function has an r^2 value of 0.76. Thus a strong argument can be advanced to provide support for the connection between increasing area of cracking and increasing stiction ratio. The same discussion applies to the plot relating area of cracking with stiction ratio for statically loaded specimens ($r^2 = 0.71$).

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[22] Stiction ratio represents the quotient of μ_s at 200 seconds post-trauma and μ_s at 200 seconds pre-trauma

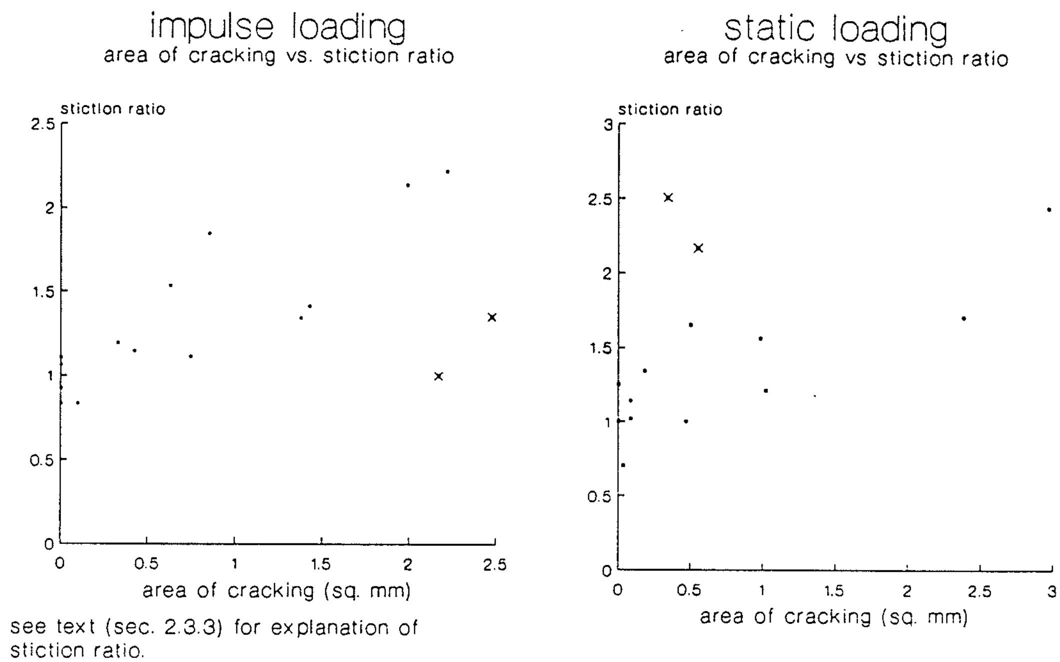


Figure 3.11 Plots of stiction ratio related to area of cracking.

4 DISCUSSION

The focus of the present investigation was to determine whether it was possible to damage the TMJ disc at impulsive loads below that required to fracture the neck of the mandibular condyle. Underlying this is the hypothesis that trauma to the disc may initiate damage that leads to progressive deterioration of function, ending in failure of the disc. Such trauma would be well below the level required to produce immediate frank failure of the disc.

Two of the purported major functions of the disc in the TMJ are those of load distribution between, and lubrication of, the articular surfaces. If either of these functions is compromised, progressive damage caused by normal function is likely to ensue. In the case of load distribution, trauma may cause internal damage which produces stress concentration in a particular region of the disc. Such concentration of stress may cause further damage under normal joint loads and a fatigue-like process is initiated. Loss of the disc's lubrication function in one area may similarly initiate a fatigue-like process; loss of lubrication being associated with increased area of surface damage. The increased area of surface wear then resulting in further loss of lubrication function. This effect may become cumulative during normal function, leading to final rupture of the disc. The above hypotheses primarily imply damage to the load bearing region of the disc; the deep region (load distribution) or the outer region (lubrication).

The experimental results showed that in all data describing the area of surface damage (see fig 3.8 and 3.9), there was a threshold value below which damage did not occur. For the statically loaded specimens, this is approximately 150 N peak force. For the impulsively loaded specimens the first damage was noted to be at an impulse magnitude of 0.3 N.s, a peak force of 150 N, and a peakedness value of 0.5. A comparison of these figures with functional loads encountered in the joint is given later in this section.

Both the static and impulsive loads have *in vivo* parallels. The impulsive load is equivalent to a traumatic blow, the static loading being somewhat equated with the type of loading expected during para-functional clenching (long duration). Given the difference in loading pattern, it is surprising to find the threshold for damage is the same for both the statically and impulsively loaded specimens. In the case of static loading in these experiments, the load has been applied for at least two orders of magnitude longer than the impulsive loads. This extra time would permit more fluid movement through and out of the disc, and allow the indenter to embed further into the disc. The shorter duration impulsive load would be expected to have a higher "splitting" component and a lower "crushing/squeezing" component, due to differences in stiffness associated with rate of loading.

The threshold value (150 N) for the initiation of surface damage of the disc is well below the value of 480 N that Hylander (1975) published in relation to bending strength of the human condylar neck. It must be remembered that the experimental material in this study is porcine disc. However, the comparison between human and porcine material is felt to be justified. Firstly, Hylander (1975) set out to deliberately under-estimate the strength of the condylar neck. He deliberately chose a gracile mandible, used the minimum of a range of tensile strengths for cortical bone, and then reduced his result by 25%. Therefore, the bending strength of the human mandibular condylar neck is likely to be much higher than the reported 480 N. Secondly, the neck of the porcine mandible is a more robust structure than the comparable portion of the human mandible and thus would be expected to be stronger. The results of this study show that the porcine disc will be damaged before fracture of the pig's mandible occurs. Lastly, the similarity of the human and porcine masticatory apparatus has already been mentioned, as with the similarity of size, and thickness of the disc of the TMJ of both animals. On this basis the human disc would be expected to behave in a similar manner to the porcine disc. Therefore, based on the presented evidence, cracking of the surface of the TMJ disc can result from a non-fracture producing blow to the mandible. The actual ratio of the bending strength of the condylar neck of the human mandible to the damage threshold of the disc is likely to be higher than the 3:1 reported for the porcine mandible and disc. This indicates that the human disc is more prone to traumatic damage. This is the most important finding of this research. The implications of this have already been alluded to and will be discussed further throughout this section.

The amount of trauma delivered to the disc is defined by a number of parameters. For a static load this was the magnitude of the load. Impulse loading was defined in terms of impulse magnitude, peak force and the peakedness of the impulse. However, the surface of the disc does not respond to the force of the impulse but rather to the stress generated by that force, *i.e.* the impulsive force distributed over the area of the indenter in contact with the disc. The indenter used for all the experiments was the same, however it is expected that the area of contact between the disc and indenter increased as the magnitude of the loading increased. This is because the indenter embeds further into the disc as the amount of load increases. Therefore, only estimates of the resultant stress during the loading can be made.

The general and local stresses encountered in the human TMJ have been investigated by Nickel (1992) and are reported to be similar to other synovial joints. He demonstrated an increase of average peak local stress with age to a value of approximately 4 megapascals (MPa) at 25 years. These figures were calculated from theoretical maximum bite forces and as such represent an extreme of normal function. From the results of this study, the minimum peak force to produce surface damage is approximately 150 N. At this peak force, the surface contact of the indenter with the disc is estimated to be 24 mm^2. This represents a peak stress in the vicinity of 6.25 MPa, a value at least 50% greater than the average theoretical peak local stresses reported by Nickel (1992). This supports the concept that normal function of the human masticatory system produces a range of stresses that are below the damage threshold of the disc. The finding that a threshold of loading must be surpassed before surface damage starts to occur, would seem obvious in retrospect. It would not make any sense that normal functional activities of the masticatory system would initiate damage to the disc.

The area of cracking has been demonstrated in these experiments to increase with amount of trauma. The rate of change of area of cracking with increasing amounts of loading has been plotted (see figure 3.8 and 3.9). The observed scatter of the data is expected due to the nature of the experiments. In common with fatigue experiments, the surface condition of the specimen is critically important to the "amount" of trauma required to produce failure, or in this case to initiate cracking. In an attempt to minimise the influence of this phenomenon in material testing, engineers create a standardised notch in the part of the specimen where they wish failure to occur. This notch acts as a focus of stress concentration and tends to reduce the scatter of the results. No such method seemed appropriate to use in these experiments, as an unaltered surface was a necessary starting point. Therefore, the scatter was deemed an inevitable part of the experimental results.

Increased disc thickness (in both the statically and impulsively loaded specimens) was related to a decreased area of cracking of the surface of the disc. It is reasonable to assume that the increased thickness of the disc allowed the load to be distributed to areas of the disc other than the surface. The effect of trauma on the layers deep to the surface of the disc is unknown (This area will be discussed later in this section). Kopp (1976) considered that thinning of the disc was associated with loss of glycosaminoglycans (GAG's) and represented one of the early signs of degenerative joint disease. Nickel (1992) has demonstrated that thinner discs have inferior load distribution properties. The thinner disc as well as being the potential site of osteoarthritic change is more susceptible to traumatic damage. This predisposes such discs to an increased likelihood of damage.

The repair potential of the TMJ disc is very limited (see appendix B). However, the disc may be able to repair minute amounts of damage. If the rate of remodelling is greater than the rate of advancement of the fatigue-like failure, then resolution of the damage is expected, with no long-term sequelae. However, if the rate of repair of the disc surface is less than the rate of progress of damage, then progressive destruction must ensue. Damage may progress by a number of fatigue-like processes. These could include increased wear due to decreased lubrication, crack extension due to stress concentration in crack tips, crack extension due to fluid flow and metabolic effects secondary to a breech in the surface of the disc. The second method is related to the load distribution properties of the disc. The latter two methods are speculative and will be mentioned in passing. Changes in surface lubrication have been demonstrated in this study and further discussion will be directed in this area.

The demonstration of a relationship between surface damage of the disc and diminished lubrication function is an important finding of this work. The results show that coefficient of stiction rises as area of surface damage increases. This increase in coefficient of stiction is a demonstration of decreased lubrication function. Thus as the area of surface disruption increases, the ability of the surface to lubricate itself decreases. This finding agrees with results reported by Nickel (1992). He related increased coefficient of stiction to impulse magnitudes greater than or equal to 0.6 N.s. This study has shown a positive relationship between impulse magnitude and area of cracking, and, area of cracking and increase in stiction. The threshold impulse magnitude for surface damage is 0.3 N.s. This is significantly lower than the figure reported by Nickel, but this can be explained in terms of his limited sample.

The experiments involving the stiction measuring apparatus were technically demanding due to the highly sensitive nature of the measurements. Accurate repositioning of the pendulum on the damaged area of the disc following trauma was challenging. Although the shape of the surface of the disc after trauma is unknown, it is feasible that extra fluid is imbibed by the damaged disc, resulting in a swollen lump on the surface of the disc. Inducing the indenter of the pendulum to rest on a swollen, extremely slippery portion of disc would be difficult. If the pendulum slides off the damaged area during the experiment, the stiction recordings will be meaningless as they relate to friction levels on an undamaged area of the disc. The greatest change in stiction occur in the early seconds following the application of the load. Therefore it is extremely important to obtain readings as close as practical to the start of the experiment. By 200 seconds most of the changes in stiction will have already occurred. Due to the extremely low levels of friction involved, the experimental recordings are highly sensitive to vibration. During the time of some of the stiction experiments outdoor construction (pile-driving) certainly complicated data collection.

However, it is impossible with this apparatus to over-estimate the coefficient of stiction (the pendulum has to rest in a deflected position - it will never rest in a position indicating a higher stiction level than is present as the laws of static equilibrium will not be satisfied). On a positive note, all of these experimental difficulties mean that any changes reported in the plots represent under-estimates of the actual stiction changes. Therefore, given the nature of the experiment, the actual relationship between area of cracking and stiction change is likely to be much stronger and more positive than the reported results were able to demonstrate.

As a result of these findings more extensive cracking gives rise to an increased rate of wear due to increased stiction. The immediate effects of a damaged lubrication mechanism will be unnoticed. The friction levels encountered will still be low even by engineering standards, so that joint motion will not be impaired. Pain is not expected due to the paucity of innervation of the articular disc. Normal function of the TMJ will involve repeated movement across the damaged area. At this stage, minute amount of scuffing and thus wear of the surface are likely to occur.

If this occurs, then a progressive increase in the area of the surface damage is anticipated as more surface material is abraded which cannot be replaced. This leads to a larger area of surface damage and hence the lubrication status would be further compromised. Thus, the increasing level of destruction creates a self-perpetuating cycle. It is proposed that this will lead to the premature failure of the disc.

Crack extension and enlargement of the damaged area are another possible fatigue-like mechanism. The importance of an intact surface for either of the lubrication mechanisms[23] proposed for porous-permeable surfaces is mentioned in appendix C. With either lubrication hypothesis operating, the surface cracks would appear to allow the uncontrolled, and rapid loss of fluid from the disc. When the disc is loaded the matrix of the disc is at a higher pressure than the neighbouring crack. This would cause fluid flow into the crack from the internal portion of the disc. It is feasible that in such a situation, crack extension may slowly occur due to the long-term wear produced by high pressure, low volume fluid flow out of the crack tip.

Maroudas (1979), commented on the surface of cartilage acting as a selective filter. This feature may also act to protect the deeper layers of the disc from degradation by preventing the entry of potentially damage producing molecules. Mast cell tryptase, plasmin, interleukin 1 and tumour necrosis factor have been shown to activate latent metalloproteinases such as collagenase (Tyler, 1991). A breech in the surface of the disc in the form of a crack would certainly by-pass the potentially

[23] either boosted (Walker *et al.*, 1968) or weeping (Lewis and McCutchen, 1959)

protective surface layer and allow the initiation of an enzymatic degradation of the collagen matrix.

As the level of trauma increases, it is unknown whether disruption first occurs in the deeper region or at the surface of the disc. A histological method, which has been promoted as a tension probe for strained collagen (Craik and McNeil, 1966) was chosen to investigate this problem. Unfortunately, no results pertaining to this question could be reported even following numerous attempts, as progress was hampered by a lack of a scientifically rigorous method. Assistance from individuals experienced in histological staining, produced the same results, and as such the lack of reliability of this method was not considered to be operator dependant. The noted variation in staining (see section 3.1.1) and thus lack of reliability of Masson's trichrome staining as a tension probe for collagen agrees with a report by Lanir *et al.* (1984). An alternative method of investigation could involve freeze-fracture and subsequent SEM examination. Time constraints did not allow development, testing and utilisation of this method.

The coincidental finding of both blood vessels and fat cells (see section 3.1.2) in the porcine disc may be important. Presence of fat cells in the porcine disc is of significant interest as this phenomenon has not been previously reported in the human disc and may represent a distinct difference between the two animals. However care must be used in interpreting this finding as the damaged sites were close to the periphery of the disc and the presence of fat cells in a non-load bearing area of the disc could be considered normal. Nickel (1992) reported macroscopic signs of degenerative joint disease in some of the porcine TMJ discs from his sample. Perhaps, the presence of blood vessels and fat are an indication of chronic degenerative changes occurring within the disc. No literature pertaining to the normal histology of the porcine TMJ disc is available. The only report which deals with fatty change in the human TMJ disc deals with the sub-synovial layers and is not directly applicable (Helmy *et al.*, 1990).

The cracking of the surface of the disc was noted to be principally in an anteroposterior direction. If the preferred orientation of cracking existed in a direction other than antero-posterior, secondary cracking in the preferred direction would be expected along the principal crack. No such observation was made. Based on this information it would appear that the porcine disc surface is stronger in an antero-posterior, rather than medio-lateral direction and histologically one would expect to see principally antero-posterior fibre arrangement. The histological findings of the present study support this finding. A number of other studies support this premise[24].

It is unknown whether a maximum amount of damage is reached under these experimental conditions and further elucidation of the nature of the relationship between amount of trauma and resultant damage is important. The variety of

[24]See - in the dog (Gillbe, 1973; Shengyi and Yinghua, 1991), the human (Strauss *et al.*, 1960; Thilander, 1964a; Knox, 1967; Thilander *et al.*, 1976; Scapino, 1983; Isaacsson and Isberg, 1985; Wong *et al.*, 1985; DuBrul, 1988), the rabbit (Mills *et al.*, 1988), the rat and sheep (Gillbe, 1973).

impulses considered in this study arose from dropping a fixed mass through a variable distance. As the impulse magnitude increased so did the peak-force and the peakedness of the impulse. These relationships have been presented in the form of plots (see figure 3.3). The minimum value that peakedness may assume is zero (a flat line), and likewise for impulse magnitude and peak force in this experimental arrangement. Therefore extrapolating from the data to include a non-negative intercept with the Y axis, indicates that the relationships are non-linear.

This can be explained by a change in the apparent stiffness of the test material as the impulsive load increases. A number of mechanisms seem to offer such an explanation. One is the increasing area of contact between the indenter and the disc as the level of loading increases. This results in lower than expected stress, as stress is a function of the area over which the load is distributed. The second mechanism proposes that fluid flow within the disc accounts for a decreasing relative contribution to the absorption of impulsive energy as the level of trauma increases. The third mechanism involves an increase in the modulus of elasticity with increasing strain. These mechanisms are discussed in the following paragraphs.

Given that the indenter has a curved surface, an increase in the area of contact between the indenter and the disc will occur as the impulsive loading increases due to the indenter embedding further into the disc. This feature results in the impulse load being distributed over a larger area. Thus, the disc surface will appear to have increased stiffness. Until the area of contact of the indenter with the disc stabilises with increasing impulsive loads, the apparent stiffness will keep increasing.

The second method to increase apparent stiffness is by increasing the rate of change of stress with respect to time. This affects the amount of impulsive energy used to move fluid through the disc. If one considers an extremely slowly applied impulse, then almost all of the energy will be utilised in producing fluid movement through the disc. In contrast, an impulse that produces a rapid change in stress will result in most of the impulsive energy being distributed in the elastic and plastic deformation of the collagen matrix, with insufficient time to dissipate fluid. Thus in the latter case, the test material will have an increased apparent stiffness.

Tanne *et al.* (1991) using dog's TMJ discs, have demonstrated that the modulus of elasticity increases non-linearly with increases in strain. The reason for this is unknown but appears to be related to the fibre orientation of the disc. In this case, the increase in stiffness is observed due to an increase in modulus of elasticity.

The inter-relation between the impulse parameters has already been mentioned. This coupling of the impulse parameters has made it impossible to determine the relative contribution of the individual impulse characteristics to the manifestations of surface damage. However it is possible to uncouple these impulse parameters by changing the mass of the hammer, the height through which it drops, and the inter-position of different materials between hammer and indenter (latex strips etc.). These changes would allow the production of a rank order of relative importance of the impulse parameters.

It is most significant that the time delay before the onset of signs and symptoms involved with the fatigue-like mechanism proposed in this study coincides with many anecdotal reports of CMD patients with a history of trauma. Kreutziger and Mahan (1975) report that after trauma, an unexplained period of quiescence occurs which is followed by the insidious onset of signs and symptoms of CMD. The only other hypothesis to date which could explain this delay is by progressive fibrillar organisation and adhesion between the joint surfaces (Sanders and Buoncristiani, 1989). No experimental evidence is available to validate such an hypothesis. The fatigue mechanism presented in this thesis is the only hypothesis which has some of its premises validated. It appears quite feasible that a fatigue mechanism, such as presented in this study could form part of the progressive adhesion hypothesis.

The finding that the disc can be damaged at loads well below the threshold for mandibular fracture, the reliance of lubrication on an intact surface layer and the apparently poor repair potential of the TMJ disc, when coupled together form a strong argument to connect initial trauma to subsequent CMD, by a fatigue-like mechanism. The demonstration, that a threshold of trauma required before damage to the TMJ disc occurs, and that the threshold appears well below the bending strength of the weakest part of the mandible has not been previously reported. Such information represents a significant advance in our knowledge of the aetiology of CMD.

5 CONCLUSIONS

The underlying purpose of this study was to investigate the response of the TMJ disc to varying amounts of trauma. The most important finding was that a definite threshold existed below which surface damage did not occur. For the particular conditions used, this threshold was 150 N peak force for both impulse and static loading. The damage is manifested as cracking of the surface of the disc predominantly in an antero-posterior direction. This suggests a principal fibre orientation within the disc in an antero-posterior direction as well. This is supported by the results of the histological portion of this study.

The quantity of force required to initiate surface damage of the disc is significantly lower than the bending strength of the human condylar neck. Given the similarity of the human and porcine disc, this argues strongly, that a traumatic incident of insufficient magnitude to cause fracture of the neck of the condyle can cause damage to the surface of the disc. The results of this study have linked surface damage of the disc with diminished lubrication function. A fatigue-like mechanism has been presented to connect the disrupted lubrication with the long-term premature rupture of the disc. This involves a self-perpetuating cycle of increased wear giving rise to further compromised lubrication function.

The calculated threshold stress at which surface damage begins to occur to the disc is above the peak local stresses achieved during normal function of the masticatory apparatus. As would be expected, this would indicate that normal function occurs in the range of stresses unlikely to cause damage to the TMJ disc.

6 SUGGESTIONS FOR FUTURE WORK

A connection has been made in this study between damage to the TMJ disc and the compromised lubrication function. The relative contribution of the articular surfaces of the condylar and temporal components to joint lubrication is unknown. If these surfaces can provide adequate lubrication then the overall effect of compromised lubrication function of the disc will be less important. Thus experiments to determine the contribution by the condylar and temporal articular coverings to joint lubrication are needed.

As the level of trauma increases, it is unknown which area of the disc is first damaged (the superficial or deep region). This information is useful to determine physical properties of the disc and secondly to determine whether the load distribution or lubrication function is first affected. A histological technique was used in this study but proved unreliable. It is suggested that a freeze fracture technique with subsequent examination using an SEM be attempted to answer this question.

In the impulsive loading experiments, the variety of impulses was produced by dropping a fixed mass through varying distances. This has resulted in the impulse parameters being coupled (*i.e.* peak force is related to impulse magnitude and peakedness etc.). These parameters can be uncoupled by changing the shape of the impulse curve. Practical ways that this can be achieved are by increasing the mass of the hammer and interposing latex strips between the hammer and the acrylic indenter. This uncoupling process will allow the determination of the relative importance of the individual impulsive parameters to the resultant damage.

The finding that the threshold for both impulsive and static loads is similar, is interesting in terms of the different method of loading. Future investigation could be directed toward the effect of variation in the time period of static loading relative to the resultant disc damage. This has practical implications in determining the effects of para-functional clenching on the TMJ disc.

Some discussion has been directed toward the fibre orientation of the porcine disc. Confirmation of a principally antero-posterior fibre orientation could be made by changing the orientation of the indenter by 90° and observing the resultant crack direction.

The present study has used an *in vitro* model and has proposed that increased articular surface wear occurs, by an increase in friction over a long period of time. Further support for this hypothesis could be gained through *in vivo* experiments. At the present time the difficulties in controlling the experimental variables prohibits a meaningful experiment of this type. Perhaps this will be possible in the long-term.

Up to this point, only the adult TMJ has been considered. Given the smaller size of a child's mandible, the load required to fracture the condylar neck would be much lower. Therefore the possibility still exists that in a child, the weaker condylar neck may be able to act as a mechanical fuse and protect the disc from damage. This is worthy of further investigation.

Appendix A

THE ANATOMY AND FUNCTION OF THE TMJ

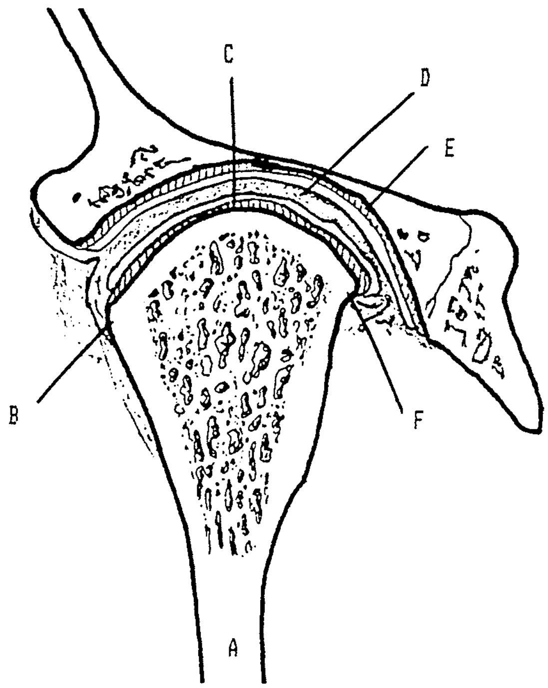
The purpose of this section is to provide some background information on the general joint structure[25] and function.

A.1 Anatomy of the TMJ

The temporomandibular joint is a complex bilateral synovial joint which allows movement of the mandible relative to the cranium (squamous portion of the temporal bones). Since the mandible is one bone, movement of one TMJ is not possible without concomitant movement of its contralateral namesake. To emphasize the inter-dependent bilateral functioning of this structure, Sicher (1962) preferred the term craniomandibular articulation. However, this bilateral inter-dependence is not unique to the mandible. The paired joints of the vertebrae possess the same type of bilateral function. Even so, the range and type of movement of the TMJ is much larger than the vertebral joints.

The articular portion of the mandible is the upper and anterior part of an ovoid condylar process, which is connected to the ramus by a thin condylar neck. In the frontal plane the condyle is generally mildly convex and approximately 15-20 mm in

[25] The anatomy of the TMJ is the subject of many excellent reviews: see Bauer, 1932; Moffett, 1962; Öberg *et al.*, 1971; Wright *et al.*, 1974; Thilander *et al.*, 1976; Öberg *et al.*, 1979; DuBrul, 1980; Mohl, 1983; Meyenburg *et al.*, 1986; Mjör, 1986; Mohl, 1988; Hylander, 1992.



A. CONDYLAR NECK; B. LATERAL CONDYLAR POLE; C. CONDYLAR CREST; D. ARTICULAR DISC; E. ARTICULAR SURFACE OF TEMPORAL BONE; F. MEDIAL CONDYLAR POLE

Figure A.1 Frontal section through TMJ. Lateral is left. Modified from Agur,(1991)

width medio-laterally (see figure A.1). A range of condylar shapes has been reported from osteological material (Yale *et al.*, 1966). This investigator found the most frequent superior outline of the condyle to be convex (58%), the next most frequent outline being flat (25%). In about 11% of cases a gabled superior surface was seen (Yale *et al.*, 1966). Thus, the articular convexity in the frontal plane can generally be divided into a medial and lateral slope, with variation in prominence of the intervening crest. The rounded medial pole of this structure projects strongly from the plane of the ramus. In contrast, the blunted lateral pole projects only weakly.

The long axis of the condyle is approximately perpendicular to the plane of the ramus. When viewed from above, the long axis of each condyle forms an angle of between 5° and 20° with an imaginary line connecting the midpoint of each condyle (Hylander, 1992; Mjör, 1986; DuBrul, 1980) (See figure A.2).

In para-sagittal section, the condyle is strongly convex and appears to be bent anteriorly (See figure A.3). The antero-posterior dimension of the condyle is about 8 to 10 mm in the adult.

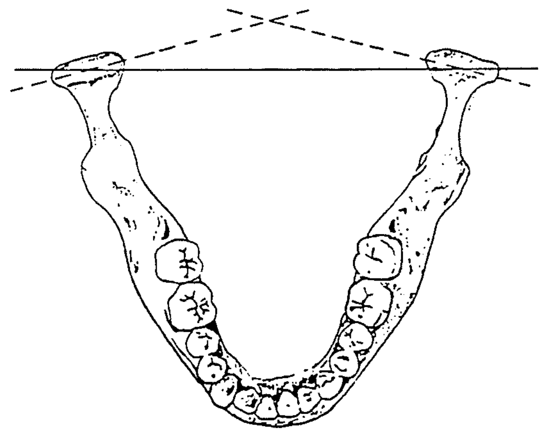
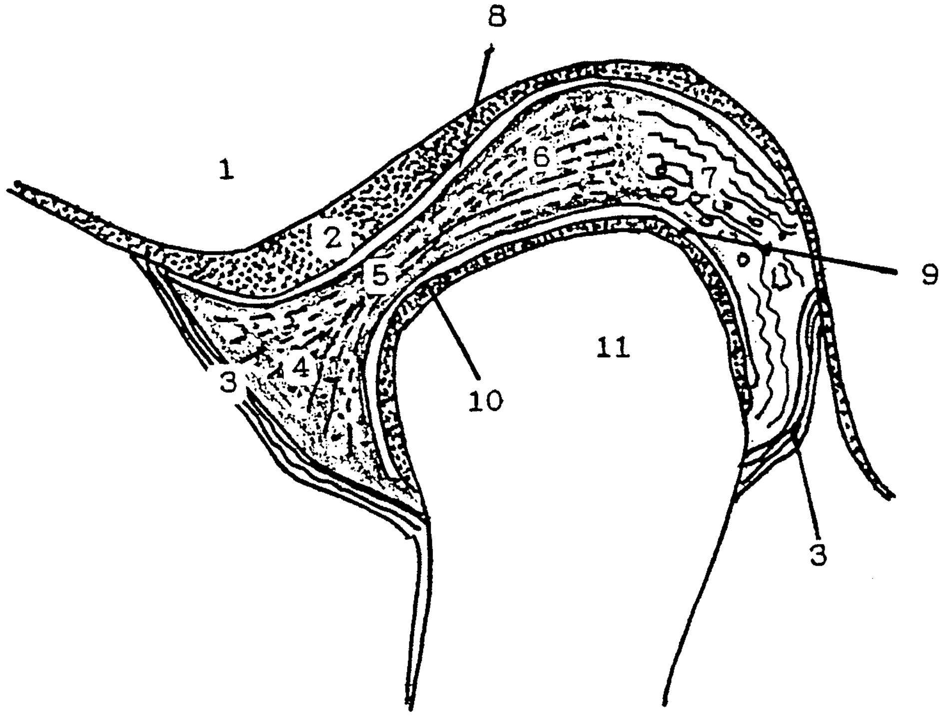


Figure A.2 Occlusal view of disarticulated mandible. The dashed lines represent the long axes of the condyles. Note the posterior convergence of these axes

The cranial portion of this articulation is bounded by the attachment of the articular capsule to the squamous portion of the temporal bone. The concave mandibular (glenoid) fossa is bounded posteriorly by the squamotympanic fissure which represents the limit of the articulation in this direction (DuBrul, 1980). The anterior portion of the fossa is formed by the articular eminence. This is a saddle shaped transverse bar of dense bone and represents laterally, the posterior root of the zygomatic arch (Hylander, 1992). In sagittal section the articular eminence is strongly convex, whilst in frontal section it is mildly concave. A wide range of variation in the radius of this concavity (5-15 mm) has been reported by DuBrul (1988). The thin bone covering the roof of the glenoid fossa is in stark contrast to the heavily braced posterior slope of the articular eminence. These facts strongly suggest that the articular eminence is the loaded portion of this joint rather than the roof of the fossa. The gently sloped region



1. articular eminence of the temporal bone, **2.** articular soft tissue of the temporal component of the joint, **3.** joint capsule, **4.** anterior band of the disc, **5.** load-bearing central region of the disc, **6.** posterior band of the disc, **7.** retrodiscal tissue, **8.** superior joint space, **9.** inferior joint space, **10.** articular soft tissue of the mandibular condyle, **11.** mandibular condyle

Figure A.3 Sagittal section of human TMJ with anterior to the left. Note magnification of joint components is inconsistent throughout diagram. Modified from Knox, 1967.

anterior to the articular eminence is termed the pre-glenoid plane and it represents the anterior extension of the articular surface.

The capsule of the joint is composed of relatively thin and loose fibrous connective tissue. The cranial side of the joint is attached posteriorly to the tip and anterior margin of the post-glenoid process and continues medially along the raised border of the squamotympanic and petrosquamosal fissures. This continues along the medial surface of the joint, attaching to the spheno-squamosal fissure. Anteriorly the capsule is attached well anterior to the crest of the eminence, but antero-medially the delineation of the capsule is indistinct due to attachment of fibres from the superior head of the lateral pterygoid muscle to the capsule and disc[26]. The lateral connection of the capsule runs from the articular tubercle on the root of the zygomatic process, along the lateral edge of the eminence, glenoid fossa and post-glenoid process.

The capsule is attached to the mandible slightly inferior to the medial and lateral condylar poles. However the anterior and posterior attachments are further inferior along the neck of the condyle. DuBrul (1980) makes the point that many fractures involving the condylar neck are intra-capsular due to the sizeable extension of the joint capsule anteriorly and posteriorly.

The capsule of the TMJ is strengthened laterally by the temporomandibular ligament. The outer portion of this ligament arises from the lateral aspect of the articular tubercle. Ligamentous fascicles converge to run posteriorly and inferiorly

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[26] Harpman and Woollard (1938) demonstrated that the medial portion of the disc is formed from part of the tendon of the lateral pterygoid muscle. Controversy surrounds the acknowledged insertion sites of the superior head of the lateral pterygoid muscle into the disc. Three opinions exist. a) All of the superior head is attached to the disc (Honee, 1972). **b)** Part of the superior head is attached to the disc (Rees, 1954; Choukas and Sicher, 1960; Dixon, 1962; Williams and Warwick, p 440, 1980). **c)** The disc is separated from the lateral pterygoid muscle by loose fibrous tissue (Pinkert, 1974 [cited from Meyenberg *et al.*, 1986]).

to insert on the posterior surface of the mandible immediately below the lateral pole. The deeper portion of this ligament consists of a narrow band of fibres in a nearly horizontal direction. This ligament serves to limit the lateral and posterior movement of the condyle. No medial reinforcement is present. The limitation of medial movement is assumed to be the responsibility of the lateral temporomandibular ligament of the contralateral condyle. This hypothesis is supported by evidence from studies of border movements of the mandible (Posselt, 1952; Brill, 1959; McMillen, 1972, Hylander, 1979). Other extra-capsular ligaments are present but are of much less significance than the temporomandibular ligament.

Interposed between the mandibular and cranial components of this joint is a firm but flexible, fibrous disc, which cradles the head of the mandibular condyle and separates the normal joint completely into an upper and lower compartment. This oval shaped disc possesses thick anterior and posterior bands with a thin central load-bearing area. The concave inferior surface of the disc fits over the top of the condyle. Rosenbaum (1946) likened the intimate relation of the disc and condyle to a jockey's hat on its wearer's head. It is about 1-2 mm thick in the central load-bearing portion and about 3 mm thick at its posterior border and 2 mm at the anterior border (Choukas and Sicher, 1960; Hansson *et al.*, 1977; Baldioceda *et al.*, 1989).

In a normal joint, with the teeth contacting in centric occlusion, the thin central portion of the disc will be well related to both the anterior slope of the condyle and the posterior slope of the eminence. This will mean the thick posterior portion of the disc will be superior to the head of the condyle. The anterior band will be near the crest of the eminence. The medial and lateral portions of the disc are tethered to the medial and lateral poles of the condyles. This limits movement of the disc to principally a rotation about the head of the condyle in an antero-posterior direction. Movement of the disc in a medio-lateral direction has been reported and is small in extent.

Posteriorly the disc continues into a thick layer of loose vascularised connective tissue, termed the retro-discal pad. This structure is generously innervated and contains many large endothelial lined blood filled spaces which will engorge to fill the space created when the condyle-disc assembly move anteriorly during function. The loose tissue, the high degree of innervation and the large blood-filled channels argue strongly that the retro-discal pad is not a loaded portion of the joint. The retrodiscal pad is attached posteriorly to the joint capsule. Anteriorly the disc merges with the joint capsule. The disc is not directly attached to the temporal bone (Mohl, 1983).

The articular surfaces of the temporal and mandibular portions are composed of fibrous connective tissue. It has been argued that this represents a functional adaptation whereby fibrous connective tissue will resist the presumed predominant stresses (shear) of the TMJ, better than hyaline cartilage (which is the normal covering of synovial joint surfaces) (Moss, 1966). More common is the explanation that the unique embryology of this joint is responsible for the unusual articular lining[27]. The soft tissue thicknesses of the articular coverings varied with location (Hansson *et al.*, 1977). On the posterior slope of the articular eminence the soft tissue layer decreased from near 0.5 mm close to the crest of the eminence to approximately 0.1 mm at the superior concavity of the fossa. Little variation in average measurements was noted between medial and lateral portions. Variation in soft tissue thickness of the head of the condyle was also seen, with the thickest tissue over the superior aspect (0.5 mm) of the medial portion. The superior aspect of the lateral portion of the condyle is slightly thinner (0.4 mm). The soft tissue thickness decreases slightly progressing onto the anterior surface (0.4 mm) and more significantly on the posterior surface (0.2 mm) (Hansson,1977). These results should be interpreted with the understanding that quoted figures are averages with large standard deviations.

A.1.1 TMJ Disc

A.1.1.1 Micro-anatomy

A.1.1.1.1 Cellular Component. According to Öberg *et al.*(1966), the cells of the disc at age four years are mostly fibroblasts and distributed near the articulating surfaces. At this stage the disc is described as being fairly cellular (Öberg *et*

[27] For details of embryology of TMJ see the following. (Harpman and Woollard, 1938; Symons, 1952: Scott and Symons, 1961; Baume, 1962; Furstman, 1963; Moffett, 1966; Youdelis, 1966; Perry *et al.*, 1985; Wong *et al.*, 1985; Van der Linden *et al.*, 1987)

al.,1966) or highly cellular (Thilander *et al.*, 1976). In the thin central portion of the disc, Öberg *et al.* (1966) describe a small number of cystiform cells which they ultimately name chondroid. With increasing age, the cellularity of the disc decreases, however the proportion of cystiform cells to fibroblasts increased. The cystiform cells were now distributed over a larger area of the central portion of the disc. In adulthood the disc is relatively acellular. This investigation would appear to indicate that the normal gerio-physiologic change in the disc is conversion of the central area from a fibrous-like connective tissue to a more fibrocartilaginous material. This viewpoint is supported by Solberg (1986) and Kim (1985). Thilander (1964 a) interpreted studies which had demonstrated cartilage cells in the disc were looking at pathological material. In her investigation she reported the absence of cartilage cells. Öberg *et al.* (1966) argued that these results were due to inadequate staining technique and that the presence of glycosaminoglycans (GAG's) surrounding a cell, was used as evidence to support classification as a chondroid type of cell. Other cell types have now been shown to synthesize GAG's, one of which is the fibroblast. The term cartilage-like cells or chondrocyte-like has since made its appearance (Mills *et al.*, 1988; Milam *et al.*,1991) and is used to describe these cystiform cells.

Vascularisation of the disc also decreases with age. Agerberg *et al.* (1969) has demonstrated high vascularity in this structure toward the end of intra-uterine life with progressive decrease in vascular density with increasing age. By adulthood the whole of the central area is avascular. These results are in agreement with those of Öberg *et al.*(1965) and Öberg *et al.*(1966). Perhaps the appearance of the cystiform cells is related to the apparently compromised nature of the nutrition in the avascular portion of the disc.

The central thin portion of the TMJ disc is almost completely aneural. Thilander (1964b) studied a range of ages of human material. In the foetal material she found nerves in the anterior and posterior part of the disc, which did not traverse the disc. These nerves were associated with blood vessels and/or terminated as free nerve endings. By contrast in the adult material, nerves could only be located in the peripheral portion of the disc close to the capsular attachment. The nerve fibres in this area seemed to follow blood vessels. Thilander felt these nerves terminated in the adventitia of the blood vessels and no complicated nerve endings were seen. More recently Wink *et al.* (1992) has reported the presence of more complicated nerve endings similar to Golgi tendon organs, Ruffini endings and Pacinian corpuscles in the human disc. The highest concentration of the neural structures was found in the peripheral areas of the disc. The central load-bearing area was reported to be almost devoid of neural elements.

A.1.1.1.2 Extracellular Matrix. The major extracellular component of the TMJ disc is water and comprises some 70% of its total weight (Gage *et al.*, 1989). The other major components are an interlacing collagenous framework (75% dry weight), embedded in a proteoglycan matrix (20% dry weight).

The collagen is the same type as found in tendons, that being type I (Hirschmann and Shuttleworth, 1976; Kashima, 1988; Milam *et al.*, 1991, Mills *et al.*, 1988). Type III collagen has been described in small amounts in the porcine disc (Kashima, 1988) and in the rat disc *in vitro* (Carvalho, 1990). These findings are interesting when viewed in relation to the controversy regarding description of the disc as a fibrocartilaginous entity (Choukas and Sicher, 1960; Griffin and Sharpe, 1960; Öberg *et al.*, 1966; Granstrom and Linde, 1973; Hirschmann and Shuttleworth, 1976) or dense fibrous connective tissue (Isaacsson and Isberg, 1985; Taguchi *et al.*, 1980; Thilander, 1964a; Wright and Moffett, 1974).

However, the collagen type universal to cartilage is present in only trace amounts in the TMJ disc. This is made more puzzling by a report of *in vitro* Type II collagen synthesis in disc explants (Landesburg *et al.*, 1989). This may represent explant contamination with fibroblasts. Fujita *et al.* (1989) reports a small amount of positive immunohistochemical staining for type II collagen under experimental conditions. This information would seem to indicate that the cells of the disc are able to synthesize type II collagen but are blocked from or do not receive a message to do so, *in vivo*. It can be surmised that the coarser more cross linked fibres of type I collagen are more suited to disc function than the finer type II collagen normally seen in cartilage.

Many morphological studies of the TMJ disc have been undertaken to determine the orientation of its fibrous component (See Table A.1). Form/function

Table A.1 *Summary of Conclusions from TMJ Disc Fibre Orientation Studies*

Investigator	*Disc Type*	*Method*	*Fibre Orientation*
Choukas and Sicher (1960)	*Human*	*LM*	*Strong interwoven bundles of fibrous connective tissue.*
deBont et al. (1985)	*Human*	*LM and SEM*	*No principal orientation. Surface layer is principally antero-posterior with some tightly coiled fibrils. Deeper layer has coarser fibres with no apparent orientation.*
Dixon, (1962)	*Human*	*LM*	*Intricate delicately woven fibrous tissue.*
DuBrul (1988)	*Human*	*not reported*	*Anisotropic surface layer, deeper layer has antero-posterior orientation with antero-medial convergence toward lateral pterygoid insertion. Lesser diagonal fibres interposed amongst major fibres.*
Gillbe (1973)	*Sheep*	*LM*	*Antero-posterior centrally. Varying directions peripherally.*
	Dog	*LM*	*Antero-posterior*
	Rat	*LM*	*Antero-posterior (long axis of disc is AP)*
Griffin and Sharpe (1960)	*Human*	*LM*	*Fibres of the anterior band are antero-posteriorly oriented (same orientation as fibres of the lateral portion of disc). Central portion of disc fibres are not as oriented as anterior band.)*
Isaacson and Isberg (1985)	*Human*	*LM*	*Central portion has antero-posterior orientation. Posterior and anterior bands show various orientations.*
Jagger (1980)	*Human*	*SEM*	*Comments on surface ridging which varies between discs and within the disc according to location. Upper and lower surfaces appear similar. Less ridging in the central portion of the disc compared to posterior. Increased ridging was thought to be an early sign of degeneration (fibrillation)*

Table A.1 continued. Summary of Conclusions from TMJ Disc Fibre Orientation Studies

Investigator	Disc Type	Method	Fibre Orientation
Knox (1967)	Human	LM and Polarizing LM	Fine delicate collagen fibres running in an unoriented pattern throughout the disc. Superimposed on this fine meshwork coarser collagen fibrils with distinct orientation were present. On the superior and inferior surface, the principal orientation was antero-posterior. Between the surface layers the fibre orientation differed slightly. In the anterior portion some antero-medially directed fibres were interspersed amongst the A-P fibres. The thin central portion of the disc possessed fibres with principally an antero-posterior orientation with much interlacing with the fine collagen fibrils. On the surface of the posterior band the heavy fibres bent in a medial or lateral direction to blend into the fibres of the posterior band. The subsurface layers of the posterior band demonstrated thick fibre bundles running in an medio-lateral direction and attached to the medial and lateral poles of the condyles. Throughout the peripheral portion of the anterior band, central portion and posterior band, circumferential fibres were noted.
Mills et al. (1988)	Rabbit	LM	Antero-posterior in central portion (crimped pattern), interlaced with transverse fibres in anterior and posterior bands.
Rees (1954)	Human	LM	Densely plaited tissue - no direction cited.
Scapino (1983)	Human	LM	Central portion of disc has principally antero-posterior orientation. These fibres interlace with transverse fibres of anterior and posterior band. Author states the fibre orientation is more difficult to determine in medial and lateral portions.
Shengyi and Yinghua (1991)	Dog	LM and SEM	Fibrils of central portion are oriented antero-posteriorly with a windy and wavy nature (postulated as a shock absorption mechanism).

Table A.1 continued. Summary of Conclusions from TMJ Disc Fibre Orientation Studies

Investigator	Disc Type	Method	Fibre Orientation
Strauss et al. (1960)	Human	LM	Dense feltwork of fibres in which some fibres seem to predominate. Thin surface layer which consists of diagonal fibres (antero-medial to postero-lateral and perpendicular) crossing at irregular intervals. Deep to this surface layer, the fibres run sagitally and deviate medially toward the anterior border. Transverse fibres well developed in posterior band. Some vertical fibres in the posterior band.
Taguchi et al. (1980)	Monkey	LM	General orientation is antero-posterior. Central portion is wavy winding form. Marginal collagen is torn and irregular.
		SEM	Surface layer composed of close network of delicate fibrils entangled vertically and horizontally. Sagittal section revealed antero-posterior orientation. Collagen fibrils (.1 - .3 μm) clumped together to present a wavy windy structure. Different pattern of fibrils on condylar and temporal surfaces.
Thilander et al. (1976)	Human	LM	Central portion is very dense with antero-posterior orientation. With increasing age the structure of the anterior and posterior bands appears as a strong 3-D net-like pattern.
Thilander (1964a)	Human	LM	Collagen fibre orientation similar to adults by 3 months of age. Anterior and central portion of disc have antero-posterior orientation. Posterior has antero-posterior orientation on the surface but deeper layers are isotropic.
Wong et al. (1985)	Human	LM	Antero-posterior centrally with transverse orientation in anterior and posterior bands.
Wright and Moffet (1974)	Human	LM	Antero-posterior in central portion of neonatal joint. Adult joint not mentioned.

correlations appear to be the reason behind some of the more recent studies. Most of these studies have used light microscopy, some scanning electron microscopy. Knox (1967) described a fine mesh-work of randomly oriented fibres superimposed on a more oriented coarser fibre framework. The general consensus is that coarse fibre orientation is predominantly antero-posterior in the central thin portion of the disc, interlacing with both vertical and transversely oriented fibres in the thicker anterior and posterior bands of the disc. The medial and lateral margins are also thicker and contain continuations of the transverse fibres from the anterior and posterior bands, thus forming a circumferential fibre arrangement.

In the tabulated investigations, the surface layers are reported with antero-posterior orientation in all but one investigation (DuBrul, 1988). As can be seen from the table some authors have not mentioned fibre direction at all, simply describing the disc as densely plaited fibrous tissue (Dixon,1962) or fibrils with wavy, windy orientation (Shengyi *et al.*, 1991; Taguchi *et al.*, 1980). deBont *et al.* (1985) using SEM concluded that **no** difference in fibre orientation of the non-surface layers could be detected between sagittal and frontal sections. They interpreted this result as indicating isotropy of the non-surface layers. Aspden *et al.* (1985) used X-ray diffraction to determine collagen fibre orientation in the meniscus of the knee. This would seem to be the method of choice to apply to the TMJ disc, but is yet to be done. However, this study also reported results using polarised light microscopy that agreed with the X-Ray diffraction experiments.

The other major extracellular component of the TMJ disc are the proteoglycans. These protein-polysaccharides have very high molecular weight (1000 - 4000 kilo-Dalton (kD)) and vary considerably in their precise chemical content. The basic structure is that of a central protein core with covalently bound side-chains. The side chains are termed glycosaminoglycans.

In the TMJ disc several types of GAG's have been described. They are keratan sulphate, chondroitin-6-sulphate (rabbit disc [*in vitro*], (Mills *et al.*, 1988), human (Kopp, 1976), and monkey (Milam *et al.*, 1991)), dermatan-sulphate (human (Kopp, 1976), rabbit, rat, dog and monkey (Granstrom *et al.*, 1973)) and hyaluronate (rabbit, rat, dog and monkey (Granstrom *et al.*, 1973), monkey (Milam *et al.*, 1991)).

The distribution of GAG's throughout the disc is not uniform. Kopp (1976) concluded from an autopsy study that the highest concentration of GAG's was in the central portion of the disc. Since it is hypothesized that at least one of the functions of GAG's is to bear load and therefore necessitates higher concentration in areas of increased stress in the disc. It is reassuring to find this high concentration is in an area expected to be most loaded during function (Mohl, 1983). Mills *et al.* (1988) has demonstrated GAG synthesis *in vitro* for the rabbit disc. This group recorded localization of immunohistochemical stain for chondroitin-6-sulphate and keratan sulphate in the area surrounding cartilage-like cells in the anterior and posterior bands. That only two GAG's were investigated and the study was carried out *in vitro* make it difficult to add to the information provided by Kopp's study.

Since the roles of the individual GAG's is unclear, and the number and distribution in the TMJ disc incompletely mapped, it would seem more valuable to concentrate on the group properties of these macromolecular aggregates.

The flexible hydrophilic nature of the GAG chains and their high concentration of negatively charged groups leads to a high swelling pressure within the tissue (due to both ionic and osmotic conditions) whilst the fine macromolecular mesh ensures a low hydraulic permeability. These two properties, in conjunction with a high water content confer the ability to carry load (Maroudas, 1979). The fineness of the mesh-work of GAG chains within the collagen skeleton and the concentration of the negatively charged groups effectively constitutes a pore of between 2 - 10 nm. This feature allows the matrix a degree of selectivity in the penetration of various solutes (Maroudas, 1979). It has been hypothesised that the selectivity of this filter may play some role in the protection of the disc from degradation by preventing access of compounds which may initiate an enzymatic degeneration (Nickel,1992; personal communication). As an example such molecules may include activators for latent metalloproteinases such as mast cell trypsin, plasmin, interleukin 1 and tumour necrosis factor (Tyler, 1991).

The high swelling pressure is balanced by tension in the collagen fibres of the matrix (Maroudas, 1976; Tepic *et al.*, 1983). The large swelling pressure will thus serve to limit the amount of fluid loss during load and ensure rapid recovery when the load is removed.

A.1.2 Synovial Fluid

The volume of synovial fluid in the human TMJ is low. Toller (1961) estimated the volume of fluid to be approximately 0.05 millilitres. It is a dialysate of blood plasma with added mucin (hyaluronate), protein (9×10^{-3} g/l, Williams and Warwick, p 428, 1980) and a low concentration of cells (Toller, 1961; Swanson, 1979; Williams and Warwick, p 428, 1980). It is slightly alkaline and the pH decreases with exercise. Some of the protein of the synovial fluid is bound to the hyaluronate, the remainder appears to be free. The mucin and protein content of this fluid accounts for the observed thixotropic and viscoelastic properties, both of which vary significantly. This variation in viscosity appears unrelated to joint, joint size, exercise state or weight of the animal.

The function of synovial fluid includes the provision of a liquid environment, maintenance of a narrow pH range, nutritive source for articular cells, and a lubricant to increase joint efficiency and decrease erosion of joint surfaces (Williams and Warwick, p 428, 1980). The fluid functions in a non-newtonian manner, i.e. the viscosity of the fluid is inversely related to rate of application of the shearing force (Swanson, 1979). This has implications when the lubrication of the TMJ is considered in a later section.

A.2 Function of the TMJ

The range of motion in a joint is a direct adaptation to the characteristic activities in that animal form (Williams and Warwick, p 425, 1980). This would seem plausible, as refinement of joint function by limitation of range of motion and direction of movement, would be conducive to optimal muscle distribution around the joint and improvement in control of the articulation. Thus, it would be expected that the range of motion of the TMJ would reflect the bilateral functional requirements of the joint.

The tasks are undertaken by the stomatognathic system and include speech, mastication, incision, swallowing and provision of an orifice for respiration. During normal activity most of these functions require control of a mobile mandible. It would thus appear that the TMJ allows a large range of virtually friction free motion, compatible with normal function, of the mandible relative to the cranium. In conjunction with the associated musculature, an additional and important function is to stabilise and control the movements of the mandible.

Since the disc is tightly tethered to the medial and lateral pole of the condyle, the only physiologic movement which occurs is rotation of the disc relative to the mandibular condyle in an antero-posterior direction. Therefore rotation about a medio-lateral axis is the principal movement of the lower compartment of the joint. The disc is not tightly attached to the medial and lateral portions of the joint capsule, free sliding movements can occur between the upper surface of the disc and the articular surface of the temporal bone. Translation, principally in an antero-posterior direction therefore occurs in the upper joint compartment (Okeson, 1989).

Figure A.4 illustrates a normal opening and closing cycle. As can be seen in the

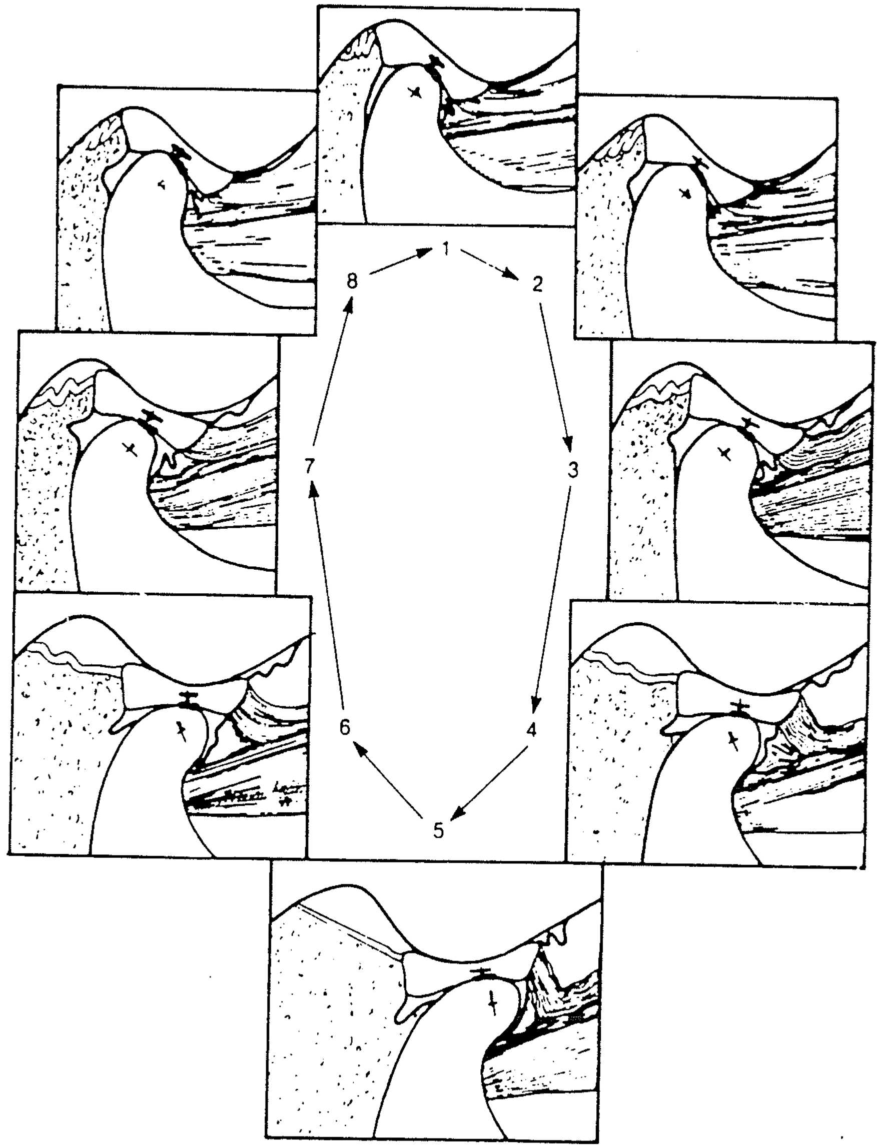

Figure A.4 Relationship between condyle, disc and fossa during normal opening and closing. Sagittal section with anterior to right. From Okeson (1989)

figure the predominant movement in the lower compartment is rotation of the condylar head relative to the disc. The principal movement in the upper compartment is translation of the disc relative to the articular eminence. The joint has been described as ginglymoarthrodial which describes its hinge-like and translatory capabilities. The bilateral inter-dependence of the TMJ must be remembered, with movement in one TMJ causing movement in the opposite TMJ.

Appendix B

REPAIR AND MAINTENANCE OF THE TMJ DISC

Good repair potential is generally coupled to availability of appropriate cell types and a good blood supply. Most of the TMJ disc has neither of these features, particularly the central portion of the adult disc. The passage of nutrients to the disc cells is only available via the synovial fluid and the ingress and egress of fluid associated with loading / unloading cycle of the disc. The vascular supply to the peripheral portion of the disc would supply nutrients to this peripheral area and possibly by some diffusion to the surrounding area. Presumably this would allow a higher metabolic rate required for tissue turnover and repair.

In order to investigate repair potential, Sprinz (1961), and Wallace *et al.* (1986) placed incisions in different positions in the TMJ disc of rabbits. These investigators found that incisions within the retrodiscal tissue or at the junction of the retrodiscal tissue and the disc healed normally. However incisions within the disc did not repair and resulted in hard and soft tissue degeneration. Misawa (1985) performed similar experiments in primates with the same results. This argues strongly in favour of the limited repair potential of the disc.

No information is directly reported on the rate of turnover of the extracellular matrix of the disc. Most reports ignore this issue or assume it to be low[28]. Indirect

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[28] As an example see Meikle (1992).

evidence from photographs from autoradiographic studies of the joint which fortuitously included the area normally occupied by the disc can be cautiously interpreted to support the hypothesis that minimal synthetic activity and tissue turnover is proceeding (Blackwood, 1966; Öberg *et al.*, 1969).

This information would indicate that should the disc be damaged, it would be extremely optimistic to expect it to repair and remodel rapidly.

Appendix C

A REVIEW OF THE TRIBOLOGY OF SYNOVIAL JOINTS

Animal synovial joints are remarkable for the extremely low coefficients of friction they exhibit during function. Many hypotheses have been presented to explain this phenomenon (See McCutchen,1983; McCutchen, 1978), however the precise method of lubrication of articular hyaline cartilage in synovial joints is still controversial.

MacConaill (1932), proposed the hydrodynamic theory of joint lubrication, based on qualitative assessment of low joint friction. The mechanism of separation of the opposing joint surfaces by an entrained film of synovial fluid was deemed to be responsible for the unmeasured but low operating friction. This mechanism is the most commonly exploited in engineering bearings and requires relatively high operating speed and rigid surfaces. The lubricant is forced into a progressively narrowing gap, which raises the fluid pressure within this space, and gives rise to separation of the bearing surfaces. This results in decreased friction and *pari passu*, decreased wear as the bearing surfaces, when in relative motion, are not in contact.

Jones (1934) reported that two types of lubrication were present in synovial joints, depending on rubbing speed. At high rubbing speed, he felt hydrodynamic lubrication predominated, however a characteristic of this mechanism is its failure at low speed, with the expectation that friction would rise. Jones, however found no such rise in the coefficient of friction[29] at low rates of relative motion. He hypothesised the joint surfaces were now in contact and that low friction was maintained by a second lubrication mechanism, that being boundary lubrication. Boundary lubrication depends on the nature of the contacting surfaces, *i.e.* the molecules adsorbed onto the surface will affect the friction that results when they are rubbed together.

Charnley (1959) undertook a set of experiments using human knee joints in a pendulum apparatus. Using low frequency oscillation, he found the coefficient of friction to be no more than 0.02. Of greater interest at that time was the relative constancy of the friction at differing speeds. Hydrodynamic lubrication is characterised by increasing friction at both higher and lower oscillation frequencies due to viscosity factors and loss of lubricating film respectively. He deduced the lubrication of the joint must be boundary lubrication, as the coefficient of friction changes little with speed. If correct, this would indicate that synovial joints are better than twice as slippery as the slipperiest man-made material (Teflon[30] against Teflon $\mu = 0.04$)

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[29]coefficient of friction is generally represented by μ. For a stationary block resting on a flat surface, the force acting upon the block due to gravity, normal to the flat surface can be represented by W. The maximum force which can be applied to the block parallel to the flat surface without the block moving can be represented by P_{max}. μ_s represents the coefficient of static friction. $\mu_s = P_{max}/W$

[30] Trade name for polytetrafluoroethylene (Du Pont)

Dintenfass (1963) allowed for deformation of the cartilage surface and viscosity effects of the lubricant to overcome difficulties with the hydrodynamic model. Thus evolved the elastohydrodynamic theory. By deforming the cartilage surfaces, the joint load could be spread over more congruent surfaces and thereby reducing the pressure gradient in the fluid film. However, neither the physical properties of synovial fluid or the high stresses encountered in the joints allow for the generation of sufficiently thick lubricating films (Hou *et al.*, 1992).

Other non-porous surface to non-porous surface lubricating hypotheses exist and the interested reader is referred to reviews of tribological theories by McCutchen (1978) and McCutchen (1983).

Hypotheses dealing with the deformable and porous-permeable nature of articular cartilage are polarised with respect the direction of fluid flow during the act of lubrication. Weeping lubrication proposed by Lewis and McCutchen (1959), utilises the principle of a hydrostatic bearing. In this type of bearing a pressurised fluid film is maintained by actively pumping the lubricant into the bearing space. These investigators suggested the weeping of fluid from cartilage under load could maintain such a pressurised fluid film between the joint surfaces. When the load is applied, the joint surfaces are normally separated by a squeeze film. The load increases the fluid pressure in the cartilage. The result is rapid radial flow of the liquid within the squeeze film and slower movement of the fluid out of the cartilage into the narrowing gap between the surfaces. As a result the previously separated surfaces begin to contact at their high spots. The reduction in film pressure as a result of the high spots starting to carry load causes the incomplete film to imbibe fluid from the cartilage. The collagenous skeletons approach each other, becoming more compressed, and as a consequence carry an increasing proportion of the load. Thus boundary lubrication forms an increasingly important part of the weeping lubrication as the cartilage becomes more "wrung out". McCutchen (1983) claims the signature of weeping lubrication is friction that is very low when the load is first applied and slowly rises to a maximum, equivalent to solid rubbing against solid.

The boundary lubricant thought to assist weeping lubrication was originally considered to be hyaluronic acid. This has since been shown to be incorrect. Radin *et al.* (1970) demonstrated the lubricating portion to be a protein or glycoprotein. Swann and coworkers (1981) narrowed this to a glycoprotein. More recently Hills and Butler (1984) suggested that previous investigators had misinterpreted their experimental results by overlooking the possibility that the lubricating fraction of synovial fluid could be a phospholipid, principally phosphatidyl choline.

Rather than the fluid flowing out of the cartilage under load, as is proposed with weeping lubrication, Walker *et al.* (1968) hypothesized the direction of net fluid flow is into the cartilage. This has been termed "boosted" lubrication. This group found difficulty in accepting McCutchen's contention that net fluid flow is out of the cartilage, since they claim the pressure gradient is in the opposite direction. Since cartilage is known for its small pore size (Maroudas, 1979), it is presumed to act as a molecular filter. The larger "lubricating" molecules of the synovial fluid will be trapped at the surface of the cartilage whilst the smaller molecules will pass into the cartilage. This leaves an increasingly enriched macro-molecular layer, to lubricate and protect the surface. The direction of fluid flow is explained using permeability factors.

A multiple of mechanisms may operate under different conditions (Williams and Warwick, p 429, 1980), although it would appear difficult to combine the last two hypotheses due to fluid flow in opposing directions.

C.1 Stiction

The low frictional forces during motion are certainly interesting, and methods by which the biological system manages to attain these are worthwhile elucidating. However, what makes synovial joints remarkable and worthy of continuing attention, is that unlike most ordinary bearings, the friction at commencement of motion is extremely low. This occurs even after a number of minutes of rest under load (McCutchen, 1983).

Of all the lubrication hypotheses, only weeping lubrication (Lewis and McCutchen, 1959) and boosted lubrication (Walker *et al.*, 1968) provide an explanation of this low start-up friction (stiction). The common element of both of these hypotheses is the requirement of an intact surface. In the case of weeping lubrication, congruency in the area of contact restricts the fluid outflow from the cartilage surface and allows pressurization of the "wept" fluid in the bearing space, and thus enable lubrication in a hydrostatic manner. The boosted lubrication hypothesis relies upon favourable permeability gradients which support movement of the mobile fraction of synovial fluid into the cartilage surface when load is applied.

If surface disruption were to be present in one of the loaded surfaces, fluid could be quickly dissipated out of the area of high stress and lubrication according to both of the porous-permeable theories would be compromised. If this cleft were to be in a load-bearing area of the articular surface when the joint was stationary, failure of both weeping and boosted lubrication would be expected. The higher start-up friction hypothesised with all other lubrication theories would be expected. Common-sense would indicate that failure of lubrication in non-biological systems results in surface abrasion, heat production, and wear of the bearing surfaces. The same is expected in synovial joints. Experimental evidence to support this contention is available. Radin and Paul (1971) oscillated joints at loads just below their maximal loading capabilities and reported minimal wear even after long experimental runs. When periodic impact loading was superimposed on this oscillation scheme, cartilage wear was readily discernible after a short time. An explanation of this observation is failure of the lubrication mechanism as a result of the periodic impact loading.

Appendix D

FUNCTION OF THE TMJ DISC

The functional role of the TMJ disc is unknown. Indeed its necessity for normal function of the joint is far from resolved. Data from Westesson *et al.* (1989) would seem to indicate some 10 - 15% of the population have normal jaw function in spite of arthrographically abnormal disc position. In addition, it would appear in some individuals without TMJ disc's, that subjectively normal function can occur in the long-term. In a series of 15 patients who had a unilateral discectomy an average of 29 years previously, Eriksson and Westesson (1985) found; all patients were free of pain, none had subjectively experienced dysfunction, and all but one could open their mouth more than 39 mm. Other reviews mention beneficial early results following discectomy, but report long term results as less favourable due to development of dysfunctional signs and symptoms (Agerberg and Lundberg, 1971; Zarb and Speck, 1979; Carlsson *et al.*, 1981). Poswillo (1979), stated that, due to the possible late complications of pain and limitation of movement, the role of meniscectomy (discectomy) in the treatment of painful, clicking TMJ's should be relegated to one of historical significance only. These polarised views illustrate the almost complete ignorance and lack of evidence of the role of the TMJ disc in joint function.

However, the TMJ disc is not alone. The function of all intra-articular fibro-cartilages of synovial joints is unknown. This stems from the difficulty of testing hypotheses relating to function (Williams and Warwick, p 424, 1980). Speculation based on deductions from phylogenetic and structural data, with mechanical analogies, has provided the basis for a plethora of proposed roles. These include lubrication, improvement of continuity between joint surfaces, shock absorption, load distribution, protection of the edges of the articular surfaces and facilitation of rolling movements (see Table D.1). These proposed roles may not be mutually exclusive.

The common feature of arthrodial joints with intra-articular discs is the functional requirement of translatory motion in conjunction with other movements (Williams and Warwick, p 424, 1980). The TMJ is certainly no exception in this regard. Phylogenetically, concatenation of multiple joints seems to have occurred in order to increase the range of motion of the limb, thereby reducing the range of motion required of the individual articulations (Williams and Warwick, p 424, 1980). It does not take a great leap of faith to view the individual joint compartments (superior and inferior) of the TMJ as separate joints in series, with the purpose of increasing the range of motion of the complete structure. Okeson (1989) supports this view by describing the disc as an nonossified bone with articulations at superior and inferior surfaces and thus infers its function to be at least in part, to increase the range of motion.

Table D.1 Proposed Functions of the TMJ Disc

Proposed Disc Function	Proponents
Increase range of motion by concatenation of joints	4,6,9,10,12,17
Stress distribution	3,4,5,6,11,15,17
Shock absorption	1,2,15,17
Improve congruity of joint surfaces	3,5,6,9,10,13,17
Restrict / control mandibular growth	8,14
Destabilisation of condyle	11
Stabilisation of condyle	2,9
Aid in lubrication	7,9,11,16,17
Facilitation of rolling movements	17
Limitation of translation	17
Protect edges of articular surfaces	17

1. Bauer (1932)
2. Boman (1950)
3. Gillbe (1975)
4. Hjortsjö (1953)
5. Knox (1967)
6. Lindblom (1960)
7. MacConaill (1932)
8. McDonald (1987)
9. Moffett (1962)
10. Okeson (1989)
11. Osborn (1985)
12. Parsons (1899)
13. Rees (1954)
14. Sprinz (1963)
15. Taguchi *et al.* (1980)
16. Toller (1961)
17. Williams and Warwick (1980)

Stress distribution has been proposed most frequently as the preferred function of the disc. Nickel (1992) appears to be the first to investigate the stress distribution properties of porcine TMJ disc. He ascertained, the disc did distribute load, and this function was highly dependant upon disc thickness (The thinner the disc, the poorer the load distribution). The relative ability to distribute load was made by comparing results with load testing of silicone rubber. The disc appears to be little better than silicone rubber. In his experiment, the disc was tested in a fully hydrated condition whilst the silicone was tested dry. This difference would create differing boundary conditions which favour the silicone rubber, as dry friction would limit spreading of the silicone test piece. This would seem to indicate that given the extremely low coefficients of friction inherent in normal joint activity, the disc can distribute stress at least as well and perhaps significantly better than silicone rubber under *in vivo* conditions.

Shock absorption is also mentioned as the function of the disc. The physical requirements one would envision of a good shock absorber would be the same as for load distribution, but with the added benefit of dissipating a rapidly increasing stress in a short period of time. No information on the stress distribution ability of the disc in relation to rate of change of stress is currently available.

No information about the ability of the disc to improve the congruency of the TMJ articular surfaces appears in the literature, although this role is frequently mentioned. Mohl (1991, personal communication) mentioned the long-term moulding of the disc to the shape of the apposite surfaces. Nickel (1992) proposes a mechanism by which areas of low stress on the surface of the disc may become "filled in" by stimulation of articular growth. These mechanisms are possible long-term consequences of incongruity.

From the literature supporting the proposed function of the disc as a mechanism to improve fit of the joint surfaces, one gets the gets the feeling that the disc has an immediate change in shape, so as to improve congruity when the jaw is moved from one position to another. It is well known that some of the fluid of hyaline cartilage can be expressed by loading this tissue (McCutchen, 1962). Functionally this would enable the tissue to change shape. Nickel (1992) writes of the same phenomena in the porcine disc. How quickly this loss of water occurs is related to the hydraulic permeability of the matrix, the internal pressure, and the applied stress (Maroudas, 1975). This would seem to provide a mechanism by which the disc can change shape during loading to facilitate increased congruency and thereby improve stability and load distribution.

As a result of animal experiments, in which the TMJ disc was removed, a compensatory shape change of the mandibular condyle took place (Sprinz, 1963; McDonald, 1987). This has led these investigators to postulate that a function of the disc is to restrain the growth of the mandibular condyle. Perhaps this is really a variation on the proposed function of stress distribution, whereby the presence of the disc provides the appropriate stress levels to inhibit growth of the condyle. Upon removal of the disc, the stress pattern would be changed. This may provide the explanation for the condylar overgrowth.

Osborn (1985) proposes that the purpose of the disc is to destabilise the condyle. Analysis of this paper would seem to indicate that the arguments advanced could be more appropriately placed under the banners of aiding in lubrication and thereby reducing friction, and secondly distributing load. Various mechanisms for the disc to aid in joint lubrication have been proposed. Generally, these hypotheses have been direct extrapolations of experimental work performed using hyaline cartilage from the articular surface of other joints. This topic is discussed in another appendix (see appendix C).

Stabilisation of the condyle, has been proposed by Moffett (1962) as an integral part of the function of the disc. No description of the suggested mechanism is provided.

The proposed functions of facilitation of rolling movements, limitation of translation, and protection of the edges of the articulation have not been attributed directly in relation to the TMJ disc and may be more applicable to other intra-articular discs. On this basis these functions have been included but will not be discussed further.

Appendix E

ANATOMY OF THE PIG TEMPOROMANDIBULAR JOINT

Although the skull of the pig is well described in veterinary anatomy texts, very little description has been afforded the temporomandibular joint (TMJ). The attention that the TMJ has received, has come from investigators interested in using the pig as an experimental model.

When comparing the oral apparatus of humans and pigs, considerable similarities have been observed in tooth form, mandibular shape, muscle form, and TMJ anatomy (Scheman, 1967). Herring (1976) concluded that these anatomical similarities were due to likeness in function; the masticatory systems of both animals being adapted for the processing of a wide variety of foodstuffs. Scheman (1967) stated that the domestic pig lacks only a well-defined glenoid fossa; in other respects, its temporomandibular articulation is closely analogous to the same joint in man. Based on the overall similarity of human and pig masticatory form and function, the selection of the pig as an experimental model to study the TMJ disc appears justified.

The pig TMJ consists of the same major components as the human joint. Scheman (1967) claims the relationship between the head of the condyle and the temporal bone is the same as that of the human. Presumably he means that the articular surface of the pig condyle is related to the disc which is in turn related to the articular surface of the temporal bone. The individual joint components are considered in the following paragraphs.

The pig mandible has very little condylar neck, the condylar process flaring immediately into the condyle. Similar to humans, the long axis of the condyles is directed medio-laterally with the medial pole of the condyle projecting more from the plane of the ramus than the lateral pole (Ström *et al.*, 1986). The medio-lateral dimension of the condyle is between 30 and 40 mm. This is somewhat larger than the human condyle. Ström and coworkers also report that the intersection of the long-axes of the condyles forms an angle of 140°. This is similar to reports from human material (Mjör,1986). The articular surface is on the antero-superior aspect of the condyle, the contour of which is slightly convex in both antero-posterior and medio-lateral directions. Both of these features are similar in the human.

The temporal component of the pig is almost flat, being somewhat convex antero-posteriorly and slightly concave medio-laterally. This lack of fossa and pronounced articular eminence is one of the most obvious differences between the human and pig TMJ. Similar to humans, a large range of motion of the mandible relative to the skull is thus permitted. On dried skulls, the movement of the mandibular condyle is limited medially by the elongated root of the auditory bulla (Herring and Scapino, 1973). The lateral limit of movement is the zygomatic arch which provides a bony stop to prevent excess condylar movement in this direction. There is no post-glenoid process, but in life, a tough fibro-cartilaginous portion of the capsule is attached to a protuberance behind the articular surface of the condyle

(Herring and Scapino, 1973). The fibrous capsule outlines an oval articular surface, the long axis of which has a medio-lateral orientation. The medial side of the temporal component is below the lateral aspect (Herring and Scapino, 1973).

The overall morphology of the pig's disc is very similar to the human. It is an oval dense fibrous tissue formed into a thickened anterior and posterior band (the posterior being thicker than the anterior) with a centrally positioned thinner biconcave area. The biconcave area is related to the articular portion of the condyle and is assumed to be the major load-bearing area of the disc. The lateral pterygoid muscle is inserted into the antero-medial portion of the disc (Ström *et al.*, 1986). Nickel (1992) reported a macroscopic antero-posterior fibre orientation in the pig disc. Similar fibre orientation is reported in human discs (see appendix D). No literature pertaining to the histology of the porcine disc is presently available.

Although not a direct part of the joint, there is a difference between the muscles of mastication in the pig and the human. The masticatory muscles of the human were similar to those of the pig except for the pig zygomatico-mandibular muscle. This small triangular muscle arose from the entire medial surface of the zygomatic arch and inserted into the uppermost part of the mandibular ramus including parts of the joint capsule (Ström *et al.*, 1986).

Appendix F

DETECTING STRAIN HISTORY IN COLLAGEN

A histochemical phenomenon first described by Craik and McNeil, (1965) seemed appropriate for use in this experiment. These investigators described a change in the pattern of collagen staining in skin stretching experiments. Masson's trichrome method (Masson, 1929) normally stains collagen green. Following strain, the collagen may relax to its normal architecture but retains the red stain in preference to the green counter-stain. These investigators subsequently reported differences in collagen staining following experimentally induced strain with Mallory's trichrome method and numerous variations of Mallory's trichrome method (Craik and McNeil, 1966). Other investigators have found similar differences using printer's ink (Lerchenthal, 1974), and a modified Whipf's polychrome technique (Wilson *et al.*, 1977). Forensic odontologists have used this phenomenon to identify bite-marks (Harvey *et al.*, 1976).

The mechanism by which the staining change occurs is unclear although a number of mechanisms have been proposed. A change in the availability of charged amino groups on the collagen as a result of strain has been proposed (Flint and Lyons, 1975; Flint *et al.*, 1975). If such a mechanico-chemical effect is responsible for the change in stain affinity, it is then possible that this could be used as a measure of prior tension in the tissue i.e a tension probe.

An alternative hypothesis is that the change is due to strain causing a change in the rate of penetration of the histochemical stain. This has been rejected by Flint for two reasons. Firstly the staining time of two minutes is much longer than that required for complete staining of dermal and tendinous collagen by both stain and counter-stain (normally 40-60 s). Secondly, he reported a difference in staining properties of collagen that had been strained but allowed to relax prior to staining, even though the "normal" wavy collagen architecture was present at the time of staining (Flint and Lyons, 1975; Flint *et al.*, 1975).

In an attempt to clarify this problem, Lanir *et al.*(1984) conducted a series of experiments on rat achilles tendon. This group reported considerable staining variation in even unstressed tendon. They concluded that the rate of penetration hypothesis (rather than the availability of charged groups hypothesis) was a much more plausible explanation of the observed phenomenon. Rate of penetration could be affected by more than one factor (permeability, concentration, pressure gradient, composition, thickness etc).

Appendix G

STICTION MEASURING APPARATUS

This is a precis of the description and specifications of the stiction measuring instrument from Nickel (1992, with permission).

The measuring apparatus was designed with the principal objective of testing the hypothesis of weeping lubrication. The apparatus was built using a pendulum design and allowed continuous loading of the test specimen during the entire experiment. Following a short period of stabilisation of the pendulum on the disc, frictional measurements could be made at any desired time throughout the experiment.

G.1 Description of the Pendulum

The pendulum was made of an aluminium cross-beam with weights attached to each end (see figure G.1). An acrylic indenter was centrally positioned on the inferior surface of the cross beam. The indenter was constructed from a flat plate of acrylic, the plane of this plate being the same as the plane of the pendulum. The inferior surface of the indenter was curved and rested upon the pig disc. The radius of the curvature of the indenter in the plane of the pendulum was 12 mm. The radius of the curvature when viewed from the side was 125 mm. Attached to the superior surface of the middle of the cross-beam was a polystyrene tower, the apex of which carried a flag. The function of the flag was to disrupt a beam of parallel light. The position of the flag and thus the angular deflection of the pendulum could be

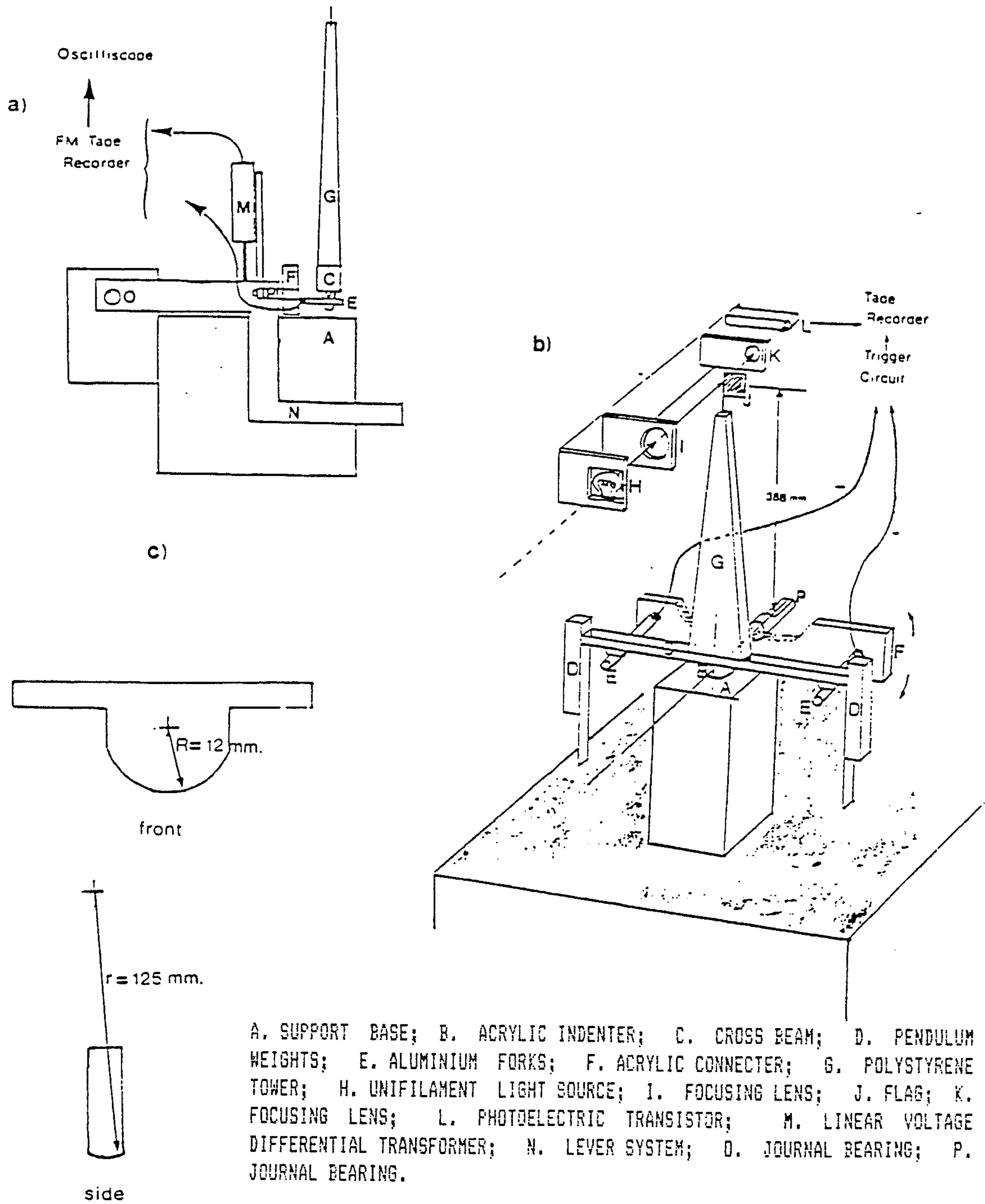

Figure G.1 Working components of stiction measuring device, including pendulum and photoelectric transistor: a) side view, b) oblique view without lever system, and c) indenter. [From Nickel,(1992) with permission] determined from the intensity of the residual light beam reaching a photoelectric transistor.

A handle was attached to a pair of metal supports by which the pendulum could be raised, lowered or angularly deflected. When an experiment was to begin the disc would be placed on sandpaper upon a metal block. The pendulum would be maximally deflected and gently lowered to the surface of the disc. As the metal supports were further lowered, the pendulum would break contact with initially one support, maintaining contact with the other support by rotation of the curved surface of the indenter against the surface of the disc. By making a series of very gentle stop/start movements during the lowering of the pendulum supports, a point would be reached at which the frictional force resisting rotation of the indenter against the disc surface was greater than the restoring force of the pendulum. At this time the contact of the pendulum with the remaining support would be broken. The deflection of the pendulum in this position would be recorded. The next reading would be made by deflecting the pendulum to the opposite side and repeating the procedure as described. The mass of the pendulum is continuously supported by the disc.

The interested reader is referred to Nickel (1992) pages 135-147 for further details.

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