PRINCIPLES AND TECHNIQUES OF TEMPOROMANDIBULAR JOINT RADIOGRAPHY AND ARTHROGRAPHY

Temporomandibular Joint Radiography:

The reference texts describe several forms of conventional radiography. The transcranial and infracranial approaches are the two types of lateral conventional radiography.

a) Transcranial method.

Though many variations of this technique exist, the post auricular approach of Lindblom gives the most reliable projection through the longitudinal axis of the joint. The central beam projects downward 25^{o}, anteriorly 20^{o} across the cranium and passes just above the petrous ridge on the film side and through the tempormandibular joint. Films are taken in both centric occlusion and on wide opening. [1]

It provides a reliable evaluation of the temporomandibular joints' bony relations. Weinberg [2] defends the use of temporomandibular joint radiography as a valid diagnostic tool. He states that the lateral transcranial is not distorted and according to Weinberg [3] their duplicability is to within $\pm$ 0.2 mm in the diagnostic area. Weinberg also defends his radiographic technique and says improved contrast and clarity means that more bone pathology is observable.

This type of radiograph displays only the lateral aspects of the condylar head and articular fossa in profile [4]. Weinberg [5] contends the image is actually a cross section of the lateral third of the condyle and fossa. However, Williams [6] employed this radiographic method to confirm diagnosis of internal derangements such as displaced discs in his practice.

The transcranial method cannot detect articular surface changes ie. arthritis. Even the new stereoscopic methods which give a greater depth perception are deficient in this aspect. [7] The current opinion is that the lateral tomographic method is better for the evaluation of condylar position and joint relations. Indeed, Greene, Laskin [8] admonish Weinberg [9] for using the 'distorted' transcranial approach instead of the corrected tomographic technique.

b) Infracranial Method (also known as the transpharyngeal or Parma technique).

This is thought of as the method of choice in lateral conventional radiography. Rasmussen [10] found in his study employing transpharyngeal radiography, that the irradiated area and the radiation dose are at the level of routine dental radiography.

This method is quick and provides valuable diagnostic information Indeed it has been used to monitor diagnosis [11] and treatment [12] of temporomandibular joint problems.

The transpharyngeal radiograph is taken with the central beam on the opposite side to the temporomandibular joint being viewed. The beam is directed cranially 5^{o}, posteriorly 10^{o}, through the mandibular notch below the base of the skull and through the oropharynx. The beam passes through the 'window' between the condyle, coronoid process and zygomatic arch. The Parma modification further reduces superimpositions.

The radiograph is performed on wide opening which -

i. Moves the condyle of interest away from the dense superimposing base of the skull into a soft tissue region which increases radiographic contrast.

ii. Increases the size of the 'window'.

However, Rasmussen [10] found that protrusion of the condyle in front of the eminence may hamper the picture of the condyle. He recommends the opening be limited to 35 mm by a mouth gag.

The transpharyngeal radiograph reveals structural changes in the condyle [13, 14] but little or no information about the glenoid fossa. The lateral pole is the most clearly depicted part of the condyle [10] but the consensus of opinion is that it only shows gross visualisation of the joint, i.e. it detects fractures, gross alteration of the condyle (because it is only an oblique view). Anterior condylar irregularities will be missed. Furthermore, the use of intensifying screens, despite decreasing radiographic dose, decreases diagnostic accuracy [10]. Its value in detecting temporo-mandibular joint degenerative changes is discussed by Toller[15] who in his 1969 article initiated its use in assessment of osteoarthritis of the mandibular condyle.

Other radiographic methods are discussed as follows:

a) Transorbital (Zimmer).

This is very similar to the transmaxillary approach [16]. Its benefits include its simplicity, lack of serious superimpositions and it being a 'true' frontal projection. These findings are supported by Updegrave [17].

The tube head is situated in front of the patient and the central beam passes through the ipsilateral orbit toward the temporomandibular joint of interest. The radiograph is taken on wide opening so the condyle is out of the articular fossa and situated on the crest of the articular eminence. If the patient cannot open wide, superimpositions will result. The radiograph observes the convex articular surface of the condyle and the broad ridge of the articular eminence. It may be used to compliment a transcranial projection (especially where the presence and extent of arthritic changes have to be defined)[18].

This projection is not as definitive as section tomography, but is simpler and more widely available. Its use is widespread. Agerberg, Lundberg [19] combined its use with arthrography and intercuspal transcranial projections to detect changes in the temporomandibular joint after surgical treatment.

b) Townesprojection - is valuable in viewing fractures but the superior part of the condyle is ill defined.

c) Waters projection - this may project the articular surface bilaterally without much superimposition. However, some areas are unclear due to superimpositions, i.e. inferior part of the condyle and the medial joint surfaces. Tomography or transorbital views are more reliable.

d) Submentovertex - this view is used for detection of temporomandibular joint carcinoma and to individually position the patients' heads for tomographic procedures.

e) Panoramic radiography - this is actually a tomographic view of the condyles. Updegrave [17] states that is may contribute helpful information of the temporomandibular joint. It visualises the entire dento-alveolar region (ascending rami, condylar and coronoid processes). It aids in the diagnosis of traumatic injury and according to Goaż and White is good for detecting gross osseous changes (tumours, extensive erosions or displaced fractures). However, the relation of the condyle to the mandibular fossa is distorted because the mouth is slightly open [20]. However, the detail is too unsharp to provide enough evidence to replace other methods [4].

According to Ogus & Toller, the existence of such a large number of projections indicates that none give an entirely satisfactory picture. Updegrave [17] however, believes that a lot of valuable information which could be gleaned from these radiographs is missed by the clinician due to poor interpretative skills. He states that comprehensive interpretation of temporomandibular joint radiographs depends on:

i. knowledge of the anatomy and histology of all the components of the joint and related structures.
ii. knowledge of the physiology of the functional occlusion system
iii. knowledge of the radiographic appearance of the normal joint in both static and functional positions.
iv. awareness of assymetry and of the broad functional range of normal joints.
v. a recognition of the radiographic limitations and the need to correlate the history and clinical observations.

vi. an effort not to tailor the radiographic evidence to fit the clinical symptoms.

Ogus & Toller established the following guidelines for optimal radiographic interpretation of the following functional components;-

The condyle - transpharyngeal, transorbital, rotated postero-anterior view.

The joint space - lateral transcranial (Lindblom technique).

Joint movement - transcranial in open and closed positions.

Tomography, however, is widely considered to be a more reliable method of radiographic evaluation. Tomography is described as a special cross sectional view used for demonstrating a selected plane or layer of a body structure. Linear tomography enables the operator to radiograph precise 4-6 mm sections through each temporomandibular joint. Tomography involves a complex hypocycloid or spiral motion and is the superior method for demonstrating changes on the articular surfaces and structural alterations within the condyle. It is highly informative on the bony relations. It obscures overlying structures by the motion involved.

There are two tomographic methods utilised:

a) lateral - this aspect used an individualised submentovertex radiograph employing a head positioner (cephlostat). The patient is seated upright to obtain the natural postural position of the bony relations. Views are taken in centric occlusion and on wide opening. Ma Xu-Chen et al [21] employed this technique.

b) frontal - in this projection, the patient again is upright with the head rotated and only one joint at a time is exposed. However, the view is often only taken in the closed position unless the articular eminence and condylar surfaces are to be viewed. The frontal view in the closed position defines the centric bony relations of the joint and condylar neck. Unlike the lateral view, it provides information about the medial and lateral poles and the length of the superior surface of the condylar head.

There is general agreement [22, 23, 24] that tomography provides the most definite radiographic information of the temporomandibular joint. It reveals detail ordinarily obscured by the superimposition of structures and it may observe many pathological changes of the temporomandibular joint.

In a comparison between tomography and several other radiographic techniques, Klein et al [25] found tomographs provided evidence of condyle and glenoid fossa irregularities that were undetectable by the usual radiographs.

Temporomandibular Joint Arthrography

Goaz & White describe this technique as a diagnostic tool for providing evidence of internal disk derangement or disk perforation, or both. It is performed by catheterising the upper and lower joint spaces under fluoroscopic guidance [26] and injection 0.5-1.0 ml. of water soluble radiographic contrast media into first the lower and then the upper joint compartments. Sequential radiographs with the jaws closed and in graded stages of opening are taken. In the successful normal temporomandibular joint arthrogram the temporomandibular joint disk is a radiolucent void between two opaque pools of iodine containing contrast medium. It supplies information on the soft tissue status of the temporomandibular joints, especially the position of the disk and its posterior attachment [26]. Arthrography provides artificial subject contrast by using materials with a high coefficient of absorption placed in or around the structure of interest, i.e. allowing us to visualise structures which would otherwise be radiographically invisible.

The basic arthrographic procedure involves initially sterilising the area with an agent like Savlon, 40 minutes before the operation pre-medication is prescribed (e.g. 20-40 mg diazepam). A local anaesthetic is administered around the area of the joint (e.g. 4 mls 1% lignocaine). There is some conjecture as to where it should act. Blaschke et al [26] advocates anaesthesia of the pre-auricular soft tissue while Toller [27] advocates an auriculo-temporal nerve block.

The temporomandibular joint is divided into two compartments by the articular disk. Separate injections of 0.3-0.5 mls. of a contrast agent (Renografin-60, Hypaque-60) is achieved using an angiocatheter.[26], which is more successful than needles.

The skin site is chosen by information gained by the previous local anaesthesia injection [27]. Toller used a method of palpating the anatomical region with a needle head until the lower joint cavity was found. The approach can be via the side of the face or through the external auditory canal. Toller employed transcranial radiographs as soon as possible after injection with the jaw closed, then at wide opening, though tomographic techniques are now used. The posterior recess of the lower joint space should opacify. The injected contrast media should be confined to a distinct space and not dispersed indistinctly into the extra articular soft tissues. When the mouth is closed, most of the contrast media will transfer into the anterior recess [26]. If any contrast media fills the upper joint space as well, a perforation of the disk or the posterior attachment is

detected.

For the upper joint space, Toller [27] used the same puncture site but the patient opens the mouth wide and the needle penetration is much deeper. Similar palpation and radiographic methods were employed. Wide opening brings the disk far anteriorly [26]. The upper joint space can accommodate slightly more contrast medium.

Needle insertion is done with direct fluoroscopic control to avoid the mechanical trauma of repeated insertion and to the neurovascular bundle behind the condyle. It is preferable to inject contrast medium into the lower compartment first for when the condylar head of the mandible moves, the medium is more easily pressed into the upper cavity in the case of perforation than it would be downward if injected into the upper joint cavity first [19].

The arthrographic facility must have the capability for the tomographic technique. Currently, the injection and distribution of dye is observed by image intensifying fluoroscopy while the procedure is videotaped. At intervals of condylar opening, spot tomographic films are obtained for detailed study.

Katzberg et al [28] employed a multidirectional tomographic unit where lateral tomograms were obtained with the mouth closed, just before the click and on maximum opening. Antero-posterior multidirectional tomograms were used in some patients to assess more fully the type of meniscal displacement. Computed tomographic-assisted images were then obtained in a selected few patients.

Westesson [20] states in the most recent arthrographic advance that 'single contrast arthrography has gained increasing acceptance as a diagnostic tool for the differentiation of temporomandibular joint disorders. However double contrast arthrography of other joints is considered superior and a procedure for double contrast arthrography of the temporomandibular joint has been developed'.

Nevertheless, despite this recent advance when arthrography and tomography is combined, the soft tissue of the joint can be outlined and studied dynamically when viewed on a fluoroscopic screen or when recorded on videotape. Fluoroscopy does, however, produce some distortion of the temporomandibular joint image when it must be performed with an inclined transcranial angulation of the x-ray beam and partly for this reason, tomograms of the opacified joint are always recommended [26].

Static tomograms or transcranial views can then be taken to permanently record certain information about functional jaw relationships [29].

In cases that involve a clicking joint, one of several open jaw projections are made just as the patient thinks a click is imminent. Bronstein [29] observed that arthrography allows diagnosis of various soft tissue derangements of the temporomandibular joint; anterior and posterior meniscal displacement, meniscal displacement with or without reduction, meniscal or posterior attachment perforations, adhesions and possibly synovial proliferations.

Temporomandibular Joint Arthroscopy

Hellsing [30] described this method of direct visual examination of the temporomandibular joint. Ohnishi [31] was first to report arthroscopy on the human temporomandibular joint in 1980 and described its diagnostic advantages and non-noxiousness.

Arthroscopyis used in conjunction with a local anaesthetic and permits good control of jaw movement. It is safer in that its action does not irritate the joint. Discomfort during and after the operation is minor.

The arthroscope can observe the relationship of joint components during function. It yields additional information for temporomandibular joint diagnosis which cannot be achieved by clinical or radiographic examination. Fibrillation of the cartilage and colour changes in the inflamed tissue can clearly be seen. It can also perform biopsy and other surgical procedures under visual control [30].

Temporomandibular joint arthroscopy adds many potential diagnostic and therapeutic advantages and seems to be valuable together with clinical and roentgenographic examinations [32].

ADVANTAGES AND DISADVANTAGES OF TEMPOROMANDIBULAR JOINT RADIOGRAPHY AND ARTHROGRAPHY

Temporomandibular Joint Radiography

Ogus & Toller observe that the existence of such a large number of conventional radiographic projections indicates that none give an entirely satisfactory picture. We can only hope to gain some information of joint structure, form and condylar function [17]. However, one advantage of conventional radiography over tomography is that it is performed in less time.

The individual radiographic methods have various advantages and disadvantages.

The transcranial projection defines the joints' bony relations well, is easy and quick [2, 7] and according to Bronstein [29] it may provide some information on muscular basal dysfunction if the anterior, superior and posterior joint spaces are measured and compared. Weinberg [33, 34, 35] claims a high degree of correlation using this technique with his temporomandibular joint pain dysfunction patients.

However, according to Kinnie [24], there are many factors complicating transcranial projections a) the anatomical differences between patients b) distortions caused by the geometric projections of the technique c) the difficulty in reorientating the patient in the same positions for serial radiographs. Additionally, this method displays only the lateral aspects of the condylar head and articular fossa and erosions are not easy to detect [27].

Carlsson et al [36] compared transcranial radiographs with gross anatomical findings and found the radiographs inadequate in diagnosis. Indeed other investigators have found the oblique transcranial unreliable [37,38]. The lateral tomogram is better for the evaluation of condylar position and joint relations.

The transpharyngeal radiograph is quick and easy to perform, gives gross visualisation of the joint and supplies valuable diagnostic information. It detects fractures and gross alterations (because the condyle is only seen obliquely in this view).

However, this view provides little diagnostic information about the glenoid fossa and bony alterations on the anterior condylar surface will be missed. Also, there is often distortion and enlargement of the condyle[10]. Rasmussen [13], in using transpharyngeal radiographs to detect temporo-mandibular arthrophy found it to be useful in revealing structural changes in the condyle (a finding supported by Toller [14]) but because the early changes may take place in unmineralised tissue, its radiographic use was

limited. He also stated that this radiographic examination may give a judgement as to whether the structural changes in the temporomandibular joint were active and if on-going, the speed of the developing temporomandibular arthropathy. In a developed osteoarthritic lesion, these radiographs could detect erosion, flattening, changes in the articular surfaces, subarticular pseudocysts and marginal proliferation of the temporomandibular joint. His radiographic findings confirmed the pattern of temporomandibular osteoarthrosis found at autopsy (i.e. lateral location, varying degrees of destruction and disk perforation [36, 39, 40].

This radiographic method is contraindicated in patients who cannot open wide (at least to 35 mm [10]) as the superior aspect of the condyle is superimposed by the articular eminence in these patients.

The transmaxillary projection is a simple, true frontal view which is without any serious superimpositions (unless the patient cannot open wide). Its value is in observing the convex articular surface of the condyle and the broad ridge of the articular eminence. It is a good complement to a transcranial projection. However, as with the previous projections it is not as definitive as section tomography, but is simpler and more widely available.

Hansson, Petersson [23] compared the diagnostic yield of the transpharyngeal, transmaxillary and oblique lateral transcranial projections of the temporomandibular joint. Most structural changes were detected in the transcranial and transmaxillary view. The transpharyngeal view could not reveal the involvement of the fossa and articular eminence. The transcranial view revealed most areas of sclerosis and flattenings while the transmaxillary found far more erosions. They suggest using more than one projection despite the superiority of the transcranial view. However, they concede tomography is the most reliable method of temporomandibular joint examination.

The diagnostic information gained from a panoramic radiograph is limited to a change in the condylar position or joint space, joint hypermobility or gross bony abnormality.

Tomography provides the most definitive radiographic information of the temporomandibular joint [22, 23, 24, 25]. It reveals detail ordinarily obscured by the superimposition of structures in the radiograph and thus may observe many pathological changes in the temporomandibular joint (articular surface, structural alterations in the condyle and bony relations). It is more reliable than conventional radiography which reproduces a very restricted part of the joint which is not always representative of the structure of the joint components and their spatial

relationships while tomography reproduces a greater portion of the joint. The amount of information in a tomographic series is greater; the selectivity in multidirectional tomography is high, making it possible to record even small structural changes [22]. However, Updegrave [17] makes the point that perhaps it is mot the radiographs which are deficient in information but our own ineptitude to detect valuable information which they reveal.

The disadvantages of tomography according to Ogus & Toller are some loss of detail due to blurring of overlying structures, the complexity and cost and the amount of radiation exposure.

The lateral tomogram gives good lateral views of the cortical margins of the temporomandibular joint and the position of the condyle within the mandibular fossa and its range of translatory motion upon opening.

The frontal tomogram supplies information of the medial and lateral poles and the length of the superior surface of the condylar head (views which cannot be seen by the lateral tomogram).

Bronstein [29] gives an overall assessment as to the value of tomography. Tomography can further define the bony contour of the condyle, fossa and eminence and more closely deliniates osseous structural relationships in certain joint positions. No information can be gained about the meniscal position or its integrity except for variations in the dimensions of joint spaces. However, when arthrography is combined with tomography, the soft tissue of the joint can be outlined and studied dynamically.

Ogus & Toller have concluded that the value of radiography in the management of common disorders of the temporomandibular joint is limited and that little information can be added to that found through a clinical examination. However, it does have value in confirming diagnosis i.e. degenerative change, meniscal displacement, and to rule out traumatic or rare lesions of the joint. Radiography has undoubted value as a research tool and radiation exposure should be avoided. The traditional radiographic examination is not significantly informative, since the early pathologic changes usually occur in the disk and capsule. Therefore, an arthrograph is required. [19]

Temporomandibular Joint Arthrography

Goaz & White describes arthrography as most helpful diagnostically in those cases in which little or no bony damage is evident on prearthrography tomograms and in which clinical evidence (clicking, popping, limitation of

opening, locking) suggests a diagnosis of disk derangement.. Arthrography may also differentiate disk derangement from other non-bony joint problems (e.g. capsulitis, myofascitis, myofacial pain dysfunction syndrome). Where the diagnosis of damage to the intra articular soft tissue is indicated, arthrography is an essential aid. Dolwick et al [41] and Toller [27] contend that the role of arthrography should be restricted to clinical research as it has limitations in clinical diagnosis.

However, Bronstein [29] states that arthrography 'has greatly improved the diagnosis for temporomandibular joint dysfunctional disorders. The ability to view the inner workings of the joint has provided a workable base on which to build a rational and reasonable treatment plan. It is now possible to correlate many of the patients' clinical symptoms with the radiographic picture and develop meaningful guidelines on which to base treatment'. Furthermore, arthrography allows diagnosis of various soft tissue derangements of the temporomandibular joint ; anterior and posterior meniscal displacement, meniscal or posterior attachment perforations, adhesions and possibly synovial proliferations [29]. This statement on the value of arthrographic detection of pathological changes in the disk and soft tissue is supported by Ma Xu-Chen et al [42]. 'Neither plain radiographs nor tomographs are comparable to arthrographs in this respect'.

Currently, Westesson [20] has applied double contrast arthrography to temporomandibular joint dysfunction and claims it is easily used, has little discomfort, is effective for studying the intra-articular anatomy and function of the joint, is valuable in the differential diagnosis of unclear facial and head pain, enables good depiction of the disk, its attachment to the capsule and the articulating surface and additionally is good for seeing anterior disk displacement.

However, arthrography does engender certain criticisms. The procedure is technically difficult (especially the catheterisation of the two joint spaces even when the fluoroscopic aid is at hand). Both Agerberg [19] and Toller [27] see this as the main reason why it has not been widely adopted until recently. Hellsing et al [30] says the procedure is complicated, difficult and time consuming.

There is also a risk of introducing the contrast media into the brain through the roof of the glenoid fossa which will lead to convulsions and possibly death. It is contraindicated in the presence of infection in or near the joint, when patients have a history of hypersensitivity to the contrast agent or related compounds (infrequently shock, local allergic

reactions and inflammation may result), in patients with impaired renal function and those with infective arthritis.

There is a train of thought that the insertion of a needle into a small joint cavity followed by the injection of non physiologic fluids can further irritate an already damaged joint. Additionally, this increased volume of fluid and sensation of fullness may cause difficulty in establishing tooth contacts post operatively. There are varying periods for normality to return. Blaschke [26] defends this 'there have been few occasions in which the diagnostic benefit to the patient did not heavily outweigh the transient discomfort.'

Another disadvantage is that there is the risk of infection if an aseptic technique is not used, but this is apparent in all surgical procedures.

Perhaps the most contentious issue is that arthrography requires great skill and experience in its interpretation [27] and some authors see that performing this procedure without a full understanding is a contraindication in itself. However, Agerberg [19] contends that the difficulties in interpretation may be exaggerated. He found that all his arthrographic diagnoses were confirmed by surgical findings.

ARTHROGRAPHY - CLINICAL USES, INDICATIONS, CONTRAINDICATIONS

Clinical Uses

(according to Toller [27])

1. To determine the presence or absence of either joint cavity
2. To determine the extent of joint cavities
   a. Volume, loose or tight capsule
   b. Filling defects at synovial edges
   c. Capsular distortions due to periarticular adhesions
   d. Intra-articular adhesions or obliterations of cavities.
3. Whether movement occurs in each cavity and its extent
4. To determine the integrity of meniscus and the extent of its movement.
5. Palpation with the injecting needle gives some information
6. The rate of absorption of contrast medium gives an indication of periarticular fibrosis.

Indications

Arthrography is helpful diagnostically in those cases where there is clinical evidence of derangement of the disk and intra-articular soft tissue (clicking, popping, limitation of opening, locking). In fact, in several U.S. institutions arthrography is used routinely for suspected temporomandibular derangement. [26]

It is also indicated when further information of joint dysfunction is required in a joint which posed unusual diagnostic difficulty [27]. Additionally, in those cases in which little or no bony damage was evident on pre arthrographic tomograms.

Arthrography is indicated for clinical research studies and also in evaluating joints which have been treated surgically.

Contraindications

Arthrography is not advised

- Where infection exists in or near the joint
- Where the patient has a history of hypersensitivity to the contrast agents or related compounds.
- In patients with impaired renal function
- In patients with infective arthritis
- In psychologically or emotionally disturbed patients where the probability exists for pre or post operative discomfort

- Where the procedure may possibly aggravate the existing joint problem.
- Where no fluoroscopic guidance is available for angiocatheter placement.

ETIOLOGY OF TEMPOROMANDIBULAR JOINT DYSFUNCTION AND ITS MANAGEMENT

In order to understand the clinical diagnostic importance of arthrography, it is imperative to consider the nature of dysfunction of the temporomandibular joint.

Many authors engage in a deal of loquacious rhetoric and dogma in an effort to expound their own views, but invariably this only confuses an already perplexing and involved subject.

Several terms are used interchangeably to explain this multi-factorial problem.

- mandibular stress syndrome - a disorder generally confined to highly developed communities and associated with neurotic tension and emotional stress
- Myofascial pain-dysfunction syndrome - where the pain source is purely in the muscles.
- Temporomandibular Joint pain-dysfunction syndrome - where the temporo-mandibular joint itself is also involved.

Helkimo [43] in 1976, made several observations of this disorder. It occurs in advanced social systems and the symptoms of mandibular dysfunction are much more common than previously thought. However, patients do not seek treatment until there is pain or limitation of function.

Essentially, the controversial question as to the etiology of the functional disorders of the masticatory system and the role played by pathological changes in the temporomandibular joint can be divided into two theories.

a. the psychophysiologic and psychosomatic theory which suggests emotional and personality causes.
b. joint trauma.

However, there is no clear cut answer. It indeed seems to be a multicausal phenomena which is self perpetuating. Weinberg, in his series of articles [33, 34, 35] sums up many current dysfunctional theories and the etiology of pain in temporomandibular joint pain-dysfunction syndrome. By understanding the etiology and causes of this syndrome, the clinician has a chance to gain long relief prognosis, whereas if we only treat the pain symptoms, reoccurance is highly probable [33]. Weinberg cites five principle causes of temporomandibular joint pain.

I. NEURALGIC PAIN

Neuralgia is not associated with function, it comes on suddenly, it may have periods of remission and often has associated trigger points. It can be initiated by direct pressure of blood vessels or neoplasms on nerve roots or by contracture pain (pressure on nerves distributed within muscle during contraction). When muscles contract around the upper cervical and greater auricular nerves, they may simulate the pain of temporomandibular joint pain-dysfunction syndrome.

Condylar displacement can also produce pressure pain. Costen [44], an early worker in this field, thought that posteriorly displaced condyles caused pain by direct pressure on the auriculotemporal nerve. Sicher [45], points out that this is unlikely but pain could result from the sensitive soft tissue posterior to the condyle. Zimmerman [46] claims that the sensory nerve endings of the auriculotemporal nerve in the posterior aspect of the capsule can produce pain due to posterior condylar displacement rather than direct pressure on the nerve root itself. Superior condylar displacement can cause pain due to impingement of nerve fibres located in the periphery of the disk. Ogus & Toller confirmed this finding. Furthermore, Bronstein [29] believes that the pressure of the capsule on this neurovascular tissue, or pain from the posterior attachment may cause myospasm which itself is painful. Some authors [9, 2, 20] have correlated a high incidence of condylar retrusion in those patients with temporomandibular joint pain-dysfunction syndrome. This is confirmed by Farrar & McCarty [47] 'there is evidence that most patients with temporomandibular joint pain or dysfunction have suffered anterior displacement of the disk associated with posterior/superior displacement of the condyle'. The causes of abnormalities will be discussed later.

Nauralgic pain near the temporomandibular joint is caused by pressure on nerves by a neoplasm, a displaced spinal disk or a blood vessel. Peripherally, pressure on nerve fibres can be caused by muscle contracture and condylar displacement.

II. VASCULAR PAIN

The symptoms of migraine, temporal arteritis can be discerned from those of temporomandibular joint pain-dysfunction syndrome.

III. TEMPOROMANDIBULAR JOINT PAIN

This pain occurs within the temporomandibular joint itself and is caused by traumatic injury, microtrauma, infection, inflammation and condylar displacement [48]. It may lead to tearing of the medial and lateral ligament attachments of the disk to the condyle, thus causing disk displacement. Occasionally the trauma can initiate osteoarthritis.

Microtrauma is due to posterior condylar displacement or the loss of posterior teeth . This repetitive trauma sets up a pattern of pathologic joint remodelling which is often destructive. Blackwood [49] assessed this pathologic joint remodelling and maintains that the disk degenerates first, then bony changes in the condyle and eminentia develop. There may be a relationship between the loss of teeth, the thinning of the disk and subsequent bony changes. There are both similarities and dissimilarities between remodelling due to the functional requirement of the temporomandibular joint and due to pathologic osteoarthritic change. Perforations of the disk are often associated with progressive remodelling of that portion of the condyle adjacent to the perforation. Disk breakdown appears to be a stimulus for subsequent remodelling activity on the condyle and temporal bone at the sight of the perforation.

Work by Toller in 1976 suggests that repeated trauma to the joint (repetitive overloading) is the principle factor and underlying mechanism in temporomandibular joint pain-dysfunction syndrome. It is actually a degenerative syndrome which is influenced by many conditions. Toller supports his theory by mechanical and histological evidence and cites parafunction and increased mandibular movements for the gradual fatigue of the cartilage.

Infection of the joint space by a disease such as gonorrhea is radiographically evident by a widening of these spaces and can produce quite severe pain.

Osteoarthritis is a primarily non-inflammatory condition of the temporomandibular joint which can cause quite severe discomfort [50]. Ogus & Toller describe osteoarthritis as a breakdown of the joint which may occur when the tissues are subject to repetitive loading in excess of their functional capacity or when they are subject to normal loads but when their functional capacity is reduced. It occurs most frequently as a result of aging but there is an age overlap between temporomandibular joint pain-dysfunction syndrome and degenerative osteoarthritis. Both are similar in that they are rarely active bilaterally.

Radiographically, osteoarthritis shows widening of the joint space on the affected side. There is a mottled effect on the bone of the condyle. As the disease progresses, marked deformity in the anterior portion of the condyle occurs in the form of 'lipping'. Rasmussen [10] describes the radiographic sequence of joint degeneration in those patients with what he called temporomandibular arthropathy (a term used to describe both clinical and radiographic entities in temporomandibular joint disease). There exists a primary active phase, characterised predominately by erosions but radiographically apparent, flattening, sclerosis, concavities, osteophytes and lipping may be apparent, especially in advanced cases. Erosions occur on the lateral pole of the condyle, while osteophytes and lipping occur on the anterior condyle. The secondary or healing phase involves bone remodelling and a flattened, reduced condyle remains.

The disease process of osteoarthritis may be very rapid. Acute or chronic osteoarthritis can take place with or without condylar retrusion. It may or may not have severe symptoms. In acute osteoarthritis, muscle pain and trismus may stimulate myofascial pain-dysfunction syndrome, while the radiographic findings will reveal a rapid destruction of the condyle and eminentia. Crepitus and limitation of jaw movement due to splinting of the muscles occurs early, due to inflammatory irritation of the masseteric and posterior temporal nerves. Osteoarthritis is unilateral and effects the temporomandibular joint centrally and then moves peripherally [15]. Radiographically, Weinberg's sequence is much the same as Toller's; radiolucent spots and spur formation on the condyle followed by flattening of the condyle and articular eminence.

Carlsson et al [36, 50] disagrees with Weinberg [33] that osteoarthritis of the temporomandibular joint is inflammatory. He states it is characterised by both deterioration and abrasion of the articular soft tissue surface and by simultaneous remodelling in the underlying bone. He does confirm, though, the radiographic signs which have been previously discussed. However, he maintains that osteoarthritis may be revealed histochemically as a loss of glycosaminoglycoans from the articular soft tissue layers long before radiographic signs are visible. Moreover, the clinical signs of osteoarthritis are not specific and resemble those of others caused in functional distrubances of the masticatory system. Carlsson [50] suggests that it is probable that a pathological process in the temporomandibular joint is often a cause of pain-dysfunction of the masticatory system, but pathosis may be present when the radiograph does not reveal any structural changes and the clinical picture is dominated by muscle symptoms. Similarly, Kopp [51] concluded from his study, the

clinical signs and symptoms of temporomandibular joint osteoarthrosis did not differ from patients with mandibular dysfunction except for crepitation of the temporomandibular joint.

In summary, Weinberg [33] states, osteoarthritic change can take place with or without a change in vertical dimension of occlusion and the patient may be asymptomatic. Its pathology may go undetected due to poor radiographic techniques and secondary muscle spasm which masks the primary joint inflammation. Osteoarthritis can be initiated by perforation of the disk, posterior condylar displacement, microtrauma and systemic involvement.

Another example of temporomandibular joint pain is the inflammatory condition of rheumatic arthritis. Synovial cells are converted to panus with a subsequent erosion of the articular surface. The condyle is affected first but unlike osteoarthritis is is usually bilateral and the pathological process erodes from the periphery to the centre of the joint [33]. Rheumatoid arthritis may be indistinguishable symptomically from temporomandibular joint pain-dysfunction syndrome [36].

Bell [48] describes arthralgic pain as dull, depressing and poorly localised with sharp periods of intensity (lancing pain) when the capsule is stretched or the articular disk impinged by forceful movements. There may be an associated secondary muscle spasm. This anatomical cause of myospasm (i.e. disk displacement) is proposed by Bronstein [29] and Ogus & Toller, and has become widely accepted.

In more recent studies [21] on temporomandibular joint pain-dysfunction syndrome, it has become accepted that the 'nature of the organic destructive phase of temporomandibular joint pain-dysfunction syndrome is essentially a secondary degenerative arthrosis owing to repeated intra articular microtraumas (e.g. bruxism) and that those cases showing transient pain-dysfunction syndrome are probably functional and reversible. However, the indolent and deferred cases with internal derangement of the temporomandibular joint actually belong to earlier manifestations of degenerative arthrosis as degenerative changes of the condyle and meniscus do occur'. In effect, Ma Xu-Chen et al [21] describes a three stage progression.

1. functional disturbance
2. internal derangement
3. organic destructive phase.

On a slightly differing tangent, Bronstein [29] maintains that the symptoms of internal derangement of the temporomandibular joint appear based on organic joint changes though the onset, development and persistence are attributed to a) continuous microtraumatic effects of myofascial pain-dysfunction syndrome b) acute mandibular trauma before the onset of chronic symptoms. In summary, be believes damage seen within joints results from the condyles being chronically directed upward and backward into the glenoid fossa by functional abnormalities. Weinberg [2, 33] similarly asserts that chronic posterior condylar displacement influences pathologic osteoarthritic remodelling. In an overview of the use of arthrography, Farrar McCarty [52] assert that 'no one can continue to argue against the validity of the phenomena of internal derangements of the temporomandibular joint'.

lV. MUSCLE PAIN

Bell [48] describes myogenic pain as deep, somatic, dull, depressing, with sharp lancing pain when muscles are stretched. Movement and function modifies the pain and it is indicated by stiffness, tenderness and positive treatment by massage. There is also evidence that pain in the muscle is related to its vascular supply.

Schwartz & Cobin [53] believe that pain in or near the temporomandibular joint is attributable to the functional incoordination or dysharmony of the mandibular muscles (as a result of psychological stress factors).

Weinberg [33] describes many causes of pain associated with muscle spasm in relation to the temporomandibular joint.

Franks [54] relates a high incidence of bruxism in temporomandibular joint pain-dysfunction syndrome. Christensen [55] showed that experimental clenching caused temporomandibular joint dysfunction pain. Sudden stretching of the musculature causes spasm and pain though muscle fatigue can produce pain also. There is mounting evidence that long standing microtraums due to condylar displacement causes inflammation within the joint which in turn creates muscle spasm and a splinting action of the muscles moving that joint [33]. (Weinberg [2] cites a 90% incidence of temporomandibular joint pain-dysfunction syndrome with condylar assymetry in the fossae). Posterior condylar displacement is also associated with muscle spasm and pain.

Muscle contraction on nerves and blood vessels creates ischaemic entrapment which impedes the removal of waste products and adds to the inflammatory response thus producing more pain. Additionally, Yemm [56] and others have attributed a higher muscle activity to stress in the form of an exaggerated response in the masseter and temporalis. Butler & Stallard [57] reported more frequent and prolonged tooth contact during stress in normal subjects. It is this increased muscle activity due to stress that confirmed Laskin's opinion that muscle spasm causes pain rather than the joint itself [58]. Also, infra red emission is hotter on the masseter of the affected side (Berry Yemm [59]).

Additionally, there are trigger points originating from sites of severe tenderness within the muscle spasm which refer pain, but there is conjecture about this origin. Protracted spasm may induce atrophic changes which suggests there are trigger sites which are stimulated by stretching or contracting, thus producing pain.

Because muscles work in group function for all oral mechanisms, it is rare to find only one muscle in spasm. When a group of muscles are in spasm, the postural position changes thus altering the function and causing spasm on the opposite side (pain-spasm-pain cycle).

Muscle pain, in general terms, can be caused by decreased blood flow, ischaemic entrapment, localised inflammation, or nutritional factors. All treatments to relieve muscle pain have proved helpful (exercises to correct abnormal jaw movement, short wave therapy for muscle spasm and bite appliances which interfere with the proprioceptor feedback with the dentition during function, thus allowing recoordination of muscles. This is extremely effective in regressing a bruxism habit [29]).

V. CONVERSION

Weinberg [33] describes hysterical conversion as rare and involves pain without joint pathology, muscle spasm or neurologic disorders.

Weinberg [33, 34, 35] has incurred a great deal of criticism from certain quarters. According to Greene, Laskin [8] he rejects the classical occlusal concept of equilibration toward centric relation, saying that it is only valid in 20% of cases and appropriate for treatment only of anterior disk displacement. He maintains that mock equilibration is just as successful as real treatment (Goodman [60]). However, Laskin points out that the patients given mock equilibration were just 'anxiety ridden' mandibular pain-dysfunction patients who have no genuine temporomandibular joint problems.

Laskin also discredits Weinberg's radiographic methods and states mandibular pain-dysfunction syndrome should exclude radiographic evidence as this automatically changes the diagnosis to degenerative arthritis. Additionally, Weinberg rejects the stress theory by extrapolating that recent surveys state that men are more susceptible to stress and therefore surely more susceptible to mandibular pain-dysfunction syndrome. Indeed women have a much higher incidence of mandibular pain-dysfunction syndrome. In order to maintain a thorough overview of the etiology of temporomandibular joint pain-dysfunction syndrome, the relevant literature on the occlusion and stress theories is reviewed.

According to Ogus & Toller, occlusion has been widely implicated in the establishment of mandibular dysfunction [61]. Bronstein [29] concedes that some patients have a functional abnormality (cuspal interference, occlusal dysharmony, overclosure) which could cause joint damage. Treatment has been very successful but many authors believe this may be a placebo effect. Laskin [62] also questioned these tooth theories - 'how could occlusal interferences develop in a functioning dentition unless iatrogenically induced and why are early symptoms related to the musculature rather than the joint inself?' On a clinical basis, several studies [63, 64] showed no statistical correlation between the presence of various occlusal abnormalities and the incidence of mandibular pain-dysfunction symptoms.

Farrar & McCarty [47] observed that anterior disk displacement (internal derangement) occurred most frequently due to irregular dental occlusal inclines which caused a posterior deflection of the condyle. However, the authors also observed that this incoordination between the superior and inferior components of the lateral pterygoid muscle, resulted from drug reactions, para-occlusal mandibular function and neuromuscular disturbances induced by emotional stress.

Krogh-Poulsen & Olsson [65] assessed functional disturbances of the stomatognathic system. The authors believe there is general agreement that they have their origin in hypertonic muscle action and occlusal dysharmony and even a psychic component. Occlusal dysharmonies included cuspal interferences during habitual closing, cuspal interferences during terminal hinge closure, excursive interferences on the non working side and bruxofacets. The combination of these and a psychic component produces a deviation of the mandibular physiological movement. In turn this causes adaptive strain in the masticatory muscles and thus a hypertonic response and further injury to the muscles and joints.

Kinnie [24], in his article on temporomandibular joint syndrome describes the four conditions of the dentition which give rise to pathologic changes of the temporomandibular joint. These were initially reported by Ricketts [66] and can be observable by tomographic techniques.

i. abnormal range of function associated with retrognathic mandibles (there is an anterior thrust of the mandible in order to function.

ii. distal or posterior displacement of the mandible (the condylar head is located in the posterior aspect of the joint and frequently behind the articular disk.) This frequently leads to chronic head, neck and facial pain.

iii. occlusal interferences during function in patients with open bites, crossbites and malocclusions, drifting of posterior teeth due to extraction

iv. loss of posterior tooth support, allowing superior displacement of the condylar head.

According to some authors personality and behavioural disorders are the principle causes of temporomandibular joint pain-dysfunction syndrome This psychic component is explained by the psychophysiologic and psychosomatic theories. As Moulton [68] explains, 'although teeth lend themselves readily to a purely mechanical approach, it should be no surprise that even here emotional factors play a significant role, since the face and mouth have such deep psychologic meanings to human beings from infancy to old age'.

Ogus & Toller have categorised these behavioural responses:

a) conversion reactions - these have Freudian origins in which the mouth and associated muscles of expression become the focus of unresolved emotional and sexual tension (oral erotocism). This leads to parafunction or bruxism.

b) personality traits - (Rugh Solberg [69] and Schwartz et al [70]) found that short term emotional states may influence their personality.

c) emotion - this is mirrored on a person's face. Patients with temporomandibular disease respond to emotional stress with increased activity of the masseter and temporalis muscles (Rugh Solberg [69], Yemm [56,71]) This supports the psychophysiologic theory of Laskin [58, 62], which was developed from Costen's observations in 1934 [44], and Lupton [72]. In effect, masticatary muscle spasm is the primary factor responsible for symptoms of temporomandibular joint pain-dysfunction syndrome (clicking, limitation of movement). The most common cause is thought to be muscle fatigue produced by chronic oral habits (involuntary tension relieving

mechanism). This causes secondary organic changes such as occlusal dysharmony, degenerative arthritis and contracture which make the process self perpetuating [72]. Laskin [62] supports his theory with experimental evidence. Moulton [68] points out that those who are predisposed psychologically or physiologically because of great inner tension may get the pain-dysfunction syndrome when precipitated by mandibular movements excessive in extent or duration or by occlusal changes following dental treatment, thus creating spasm and pain.

3) Response specificity - financial, personal and social problems create a specific physiologic response to stress situation in the form of parafunction. Bronstein [29] also correlated this acute psychologic trauma with an onset and exacerbation of joint symptoms.

There is no definitive aetiology of temperormandibular joint pain-dysfunction syndrome. Modern evidence suggests it is one of a group of tension related functional disorders. Obviously it is a complex multi-factorial problem which requires accurate and thorough assessment before successful treatment may be instituted.

Management of Temporomandibular Joint Pain-Dysfunction Syndrome

Consensus is lacking as to whether the primary approach to the disorder should be considered through the consideration of an occlusal discrepancy, muscular imbalance, soft tissue joint derangement, a psychophysiological disorder, or some combination of these. However, Bronstein [29] concedes that most treatment, whether occlusal equilibration, bite-plate therapy, stress management, or surgery, is directed at symptomatic relief and in many cases only provides temporary relief. Although this author also discussed the role of disoccluding posterior teeth by bite plate therapy. By interrupting the pain-spasm cycle and reducing the microtraums to the joint, as well as the condylar displacement and its subsequent inflammation, the muscles will function over a new length and the bruxism habit will be decreased. Toller [73] reported that the majority of patients with temporomandibular joint pain-dysfunction syndrome are successfully managed by conservative treatment, i.e. reassurance, muscle training, thermotherapy, occlusal adjustment and normalisation and bite appliances. The author indicated that a few patients fail to respond and may need surgical correction, e.g. condylar shaving, condylectomy, intra articular meniscus removal.

Moulton [68] found that in those patients with a long standing pattern of bruxism with neurotic backgrounds, immediate relief through slow conservative therapy which provides some muscle relaxation, is not possible. Obviously,

under these conditions, the ultimate answer may lie in therapies which manage the underlying cause of stress, such as hypnosis. However, Bell [74] argues that a theory of muscle action as the key to occlusal function or dysfunction does not rule out the importance of identifying and correcting occlusal discrepancies. Ramford & Ash confirm this view.

Williams [6], in discussing the role of the condylar position in temporomandibular joint pain-dysfunction syndrome, suggested that if patients exhibited a healthy condylar position and good neuromuscular responses, then the existing occlusal schemes are excellent. 'If we reconstruct, conduct orthodontic treatment or equilibrate patients who have poor condylar positions, the temporomandibular joint may not be able to physiologically adapt in the long term'.

Farrar & McCarty[52] emphasized that some therapies may actually cause derangements.

However, in many internal derangements of the soft tissues of the temporomandibular joint, when diagnosed both clinically and arthrographically, surgery is the only treatment. Bronstein [29] points out though, to put a patient through this surgery, which is not a minor procedure, and neglect recommending or initiating follow-up occlusal, bite-plate or restorative therapy to correct or control the causes, may be inviting eventual recurrence of symptoms.

CLINICAL MANIFESTATIONS OF TEMPOROMANDIBULAR JOINT PAIN-DYSFUNCTION SYNDROME - PATHOLOGY, SYMPTOMS, RADIOGRAPHIC AND ARTHROGRAPHIC INTERPRETATION AND FINDINGS.

Pathology

The findings of Toller [73] confirmed, during capsular rearrangement surgery, that there is an associated pathology with temporomandibular joint pain and dysfunction. He found undulations on the condylar surface and dullness of the fibrous tissue covering the articular surface, suggesting fibrillation, similar to that found in early osteoarthrosis.

Histopathologic experiments have revealed the following changes:

a) fibrillation - this is the first indication of degenerative change and occurs in the area of articular contact across which the greatest loads pass. There is a progressive loss of cohesion between collagen bundles which make up the surface layer. Horizontal clefts in which fluid collects are formed between the bundles, which then fray off. There may be increased mineralisation of the subarticular cartilaginous zone which eventually causes remodelling of the bony trabeculae but leaving a normal bone marrow.

b) denudation and eburnation - with the loss of fibrous tissue surface, there is a simultaneous maturation of the bony endplate producing a 'polished' surface.

c) perforation - further progression produces a thinning of the endplate which may be sufficient to allow contact between the articular cavity and the bone marrow, producing a fibrotic reaction within the cancellous spaces.

d) subarticular collapse - small cysts deep in the bone may develop and may collapse causing generalised bone destruction.

e) erosion - with the loss of more and more trabecular bone, a deepening lesion occurs with frequent gross destruction of the normal condylar anatomy. Fibrous tissue replaces the marrow spaces and there is an increase in number and size of chondrocytes.

f) repair - there is evidence this may occur by development of a new fibrous articular surface with a cortical endplate underneath. Repair may occur at any stage of this pathologic process, however.

In cases of meniscal displacement (internal derangement) Ma Xu-Chen et al [21] found all cases had associated degenerative changes. This view was proved by Toller's study [73] and supports the author's claim that indolent and deferred cases with temporomandibular joint dysfunction do have internal

derangement of the temporomandibular joint and that associated degenerative changes actually are an early stage of degenerative arthrosis.

In an overview of the pathological effects of internal derangements, Farrar & McCarty [47] describe how anterior disk displacement stretches the posterior ligaments and they are drawn into the interarticular space where a herniated sac develops.

This becomes more fibrous and the vascularity decreases (an adaptive remodelling to abnormal function). In time this herniated portion of the ligament may perforate. As the perforation becomes more chronic, osteophytic spurring of the condyle in the region of perforation may occur. After many months, internal derangements of the disk and condyle invariably results in osteoarthritic degeneration of the temporomandibular joint.

Symptoms and Signs

Temporomandibular joint pain-dysfunction syndrome may be demonstrated by a variety of symptoms and signs. Some, such as clicking and locking of the temporomandibular joint are specific for this abnormality and will be discussed in some detail.

Kinnie [24] in his 1981 article attributed the following observations to temporomandibular joint pain-dysfunction syndrome : headaches, sinus problems, loss of hearing, dizziness, muscle pain, nausea, sleeplessness, head noises, slicking sounds and social and family maladjustments. Additionally, crepitus and tinnitus have been cited as clinical signs [67]. Solberg et al [75] in their thorough clinical appraisal, found further signs: dull occlusal sounds on repeated firm closure of teeth, limitation or uncoordination of mandibular movement, faulty relationships between mandibular reference points, e.g. retruded contact position, pain in face and neck, pain and tenderness of the muscles of mastication (especially lateral pterygoids), asymmetry in slide from retruded contact position to intercuspal position associated with other dysfunctional signs. They also noted that most patients were not aware anything was wrong and those who showed signs of bruxism were more likely to have masseter tenderness and limited opening.

The work of Greene et al [76] and Agerberg, Lundberg [19] confirmed many of these findings. Weinberg [77] in his 1976 analysis of posterior bilateral condylar displacement as an aetiological factor in temporomandibular joint dysfunction, found the following signs of temporomandibular joint dysfunction; joint noise, tenderness on muscle palpation and acute temporomandibular joint pain. He concluded that joint noise was an early sign of dysfunction

because of its higher incidence in comparison with other signs.

Kinnie [24] compared the set of symptoms of temporomandibular joint pain-dysfunction syndrome with those of rheumatoid arthritis of the temporomandibular joint and found they differed slightly. Local pain, warmth, swelling, inability to open jaws normally, bilateral nature of symptoms, symptoms present on awakening and which subsequently diminish during the day, are all specific for the latter condition [78]. Similarly, osteoarthritis differs in a specific fashion, i.e. dull pain in the region of the temporomandibular joint, joint stiffness following periods of rest and an increase in pain as the day wears on (especially if the joint is overused) and crepitus [79]. Kinnie [24] has developed indexes (K-F-S and F-S) according to visual signs and symptoms to accommodate diagnosis of pain dysfunction and aid in its treatment.

There is a general consensus of opinion that the symptoms attributed to anterior meniscal dislocation and temporomandibular arthropathy (a broad term applied by some to cover a range of degenerative disorders of the temporomandibular joint) have similar, if not identical, history and clinical symptoms [80]. Early temporomandibular arthropathy is characterised by decreased maximal opening and pain (on maximal opening, tenderness of temporomandibular joint capsule, tenderness of lateral pterygoid muscles) As the erosion continues and late temporomandibular arthropathy is reached, a different set of symptoms is evident, i.e. crepitus and constriction are most frequent. It is possible, also, that complete resolution may occur. The correlation of the relative histories of temporomandibular arthropathy and anterior disk dislocation has not been studied [47,77,80,81,82,83,84,85] though it is possible that temporomandibular arthropathy may be triggered by anterior disk displacement.

The Clicking Joint

There have been several explanations for this phenomena but recent literature supports the theory of mechanical obstruction (build up of fibrous cartilage) and a subsequent release of energy producing the click [80]

Ogus & Toller described the progression of clicking symptoms:

a) meniscal hesitation - repetitive overload of the joint changes the physical nature of the articular surface and its frictional characteristics. The disk starts to 'stick' in normal joint movement.

b) early click - 'sticking' in the lower joint component. On opening, the meniscus, instead of remaining stationary, adheres to the condyle as it rotates. As forward translation in the upper compartment begins, tension builds up in the disk and with further opening, friction between

the condyle and disk is overcome with a release of energy (click) and it assumes its normal functional path; an observation confirmed by Toller.[73]
c) late click - during forward translation of the condyle as it moves down onto the articular crest. When meniscal hesitation occurs in the upper compartment the meniscus may stick and tension is built up until on further opening there is an audible release of energy (click).

In Westesson's[20] 1984 study in double contrast arthrography, he concluded that clicking was associated with uncoordinated movement between the condyle and disk. Weinberg[77] discussed the etiology of joint noise in condylar retrusion. He supplies radiographic evidence to support the fact the joint noise immediately precedes complete closure due to posterior condylar displacement. That same noise is produced on opening. He hypothesises that perhaps the ligamentous attachment of the condyle is less rigid than that of the disk, thus producing condylar rotations as well as sliding movements. Secondly, he observed that no noise is produced if the mandible is held in retruded position and only hinge movements are allowed. This confirms that noise is produced by anteroposterior malposition of the condyle/disk prior to complete closure. It is for this derangement that Dolwick et al[41] have adopted the term anterior subluxation, instead of clicking. Weinberg[77] supports his conclusions with a finding of 70% of individuals with temporomandibular joint pain-dysfunction syndrome had condylar retrusion.

Further evidence confirms that clicks will not occur in retrusion unless preceded by clicks in mandibular opening. The reciprocal click can be early, intermediate or late, depending on the relative position of condylar protrusion at which the click occurs. The same author cites that clicking is due to internal derangement (anterior disk displacement associated with posterosuperior displacement of the condyle when the teeth are in centric occlusion)[47].

However, Hellsing et al[30] arthroscopic investigation is more suggestive of degenerative changes within the joint as the cause of the physical obstruction rather than an anterior displaced disk. Another study[86] in 1984 correlated joint sounds with joint morphology using ausculation in cadavas. They found two-thirds of those joints with sounds were normal and the other one third had disk displacement. Joints with clicking or crepitation showed constant disk displacement (50% had osteoarthrosis). Therefore joint sounds were a consistant sign of pathology, whereas no sound was a weaker sign of a normal joint.

Limitation of Movement (Locking)

Nanthaviroj [80] describes a mechanical obstacle (deviations in the form of the articulating components due to a build up of fibrous cartilage) which leads to locking. He supplies tomographic evidence to support this. Ogus & Toller suggest that if a click persists and the overload on the joint increases, then the sliding properties of the articular surfaces deteriorate and the symptoms worsen. The disk fails to complete its excursion and remains stuck at the point it used to click. This may be aggravated further by reflex muscle spasm and by an increased load on the joint. Thus the symptoms of locking and clicking are closely related [73].

Locking has also been attributed to constant anterior disk dislocation which block the anterior translation of the condyle [20]. It may act as a physical barrier to opening. Some patients with a distinct loud click and sharp pain at click, occasionally experience an intermittent closed lock condition [41]. Furthermore, Katzberg et al [28] suggests disk displacement is a result of damage to the posterior attachment of the meniscus. Arthrographic evidence supported his claim relating to soft tissue internal derangement and associated capsular damage.

As the condition becomes more chronic, often the range of condylar movement actually increases because the functioning condyle begins to push the dislocated disk even further forward [47].

Stiffness and Pain

Although temporomandibular joint dysfunctional pain has been thoroughly discussed previously, it nevertheless is an important diagnostic symptom. Ogus & Toller maintain that stiffness and pain may be a function of the changes in frictional quality of the articular surfaces, but more frequently is associated with muscle fatigue (i.e. the morning after bruxing). Pain may originate from -

a) the joint - when the joint is locked the condyle tends to ride up the anterior meniscus forcing the condyle and eminence apart thus irritating the nociceptive receptors in the fibrous capsule.

b) retrocapsular tissues - due to prolonged compression on the dense plexus of unmyelinated nerve fibres within the pad of fat.

c) Reflex muscle spasm.

Bronstein [29] is of the opinion that pain is caused by pressure on the neurovascular tissues. In his 1981 article he confirmed this opinion by arthrographic means. A chronically directed condyle in an upward and

backward direction produces pain. On an opening movement, the retruded condyle may invoke a stretch reflex in the lateral pterygoid muscle (especially those fibres attached to the meniscus) and consequently myospasm of that muscle may occur, thus placing the meniscus in front of the condyle in closed positions. The condyle is allowed to articulate on the posterior attachment and neurovascular tissue, thus causing pain. This condition may be aggravated by contraction of the lateral pterygoid muscle during closing movements of chewing and swallowing. The combined effect of stretching and myospasm leads to bilaminar redundancy and anterior disk displacement .

It must be emphasized though, that if the overload on the joint is arrested, these symptoms may resolve.

Radiographic Findings:

Ogus & Toller stress that the majority of patients with mandibular stress syndrome have no radiographically detectable lesions. Only rarely do radiographs reveal an internal derangement. However, there is a definite radiographic sequence. Initially there is a loss of density of the condylar surface opposite the principal point of articular contact. A rough woolly appearance develops with a spreading rarefaction in the bone and eventually becomes a saucer shaped erosion which may or may not heal. Occasionally osteophytic 'lipping' may be observed at the anterior edge of the condyle. The articular surfaces may range from being slightly flattened to being completely remodelled.

Nanthaviroj et al [80] tomographic findings showed that the distance between the mineralised parts of the condyle and temporal components in all patients was larger just before and after locking and clicking, suggesting that there are deviations in the form of the articular components due to a build up of fibrous cartilage which creates a mechanical obstacle.

Farrar [81] employed transcranial oblique radiographs to diagnose loose capsular ligaments and anterior disk dislocation in 5% of those patients with temporomandibular joint complaints.

Ma Xu-Chen et al [21] found the following radiographic changes.

a) sclerosis of the condyle, glenoid fossa and articular eminence
b) various destructive cyst-like changes, flattening, lipping and shortening of the condyle
c) communication between the superior and inferior joint cavity.

Rasmussen [13] used transpharyngeal radiographs in his work on temporomandibular arthropathy. His findings related to healing of the fibrous articular cartilage [14] and this accounts for improved symptoms, i.e. crepitus. However, radiographic detection of the onset of early temporomandibular arthropathy may not be important because early changes may take place in unmineralised tissues.

Arthrographic Interpretation

Normal joint space arthrograms [27]

a) lower joint cavity in closed position - the radiopaque contrast medium fills the small anterior sulcus which lies between the meniscal and condylar insertions of the lateral pterygoid muscle and the larger posterior sulcus extending down the posterior aspect of the condylar head..

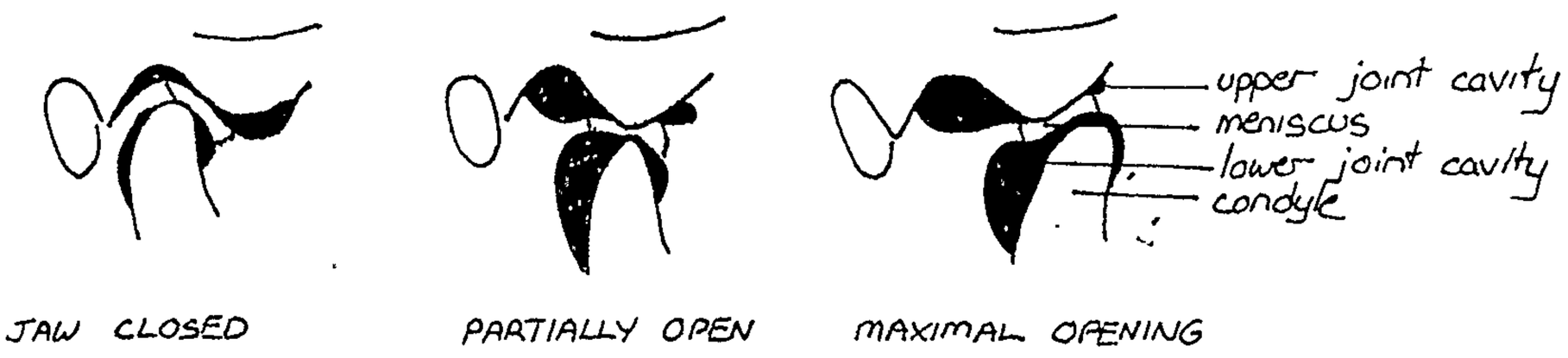
b) lower joint cavity on jaw opening - the anterior sulcus is reduced and fluid is forced back into the posterior sulcus, assuming a rounded humped shape behind the condyle and below the disk.

c) upper joint cavity in closed position - the dense lateral edge of the filled cavity has a sigmoid shape.

d) upper joint cavity on jaw opening - upward pressure by the condyle squeezes the radiopaque filling medium distally, so that the posterior sulcus becomes full and assumes a D shape with the flat side below. Anterior sulci filling is diminished to near invisibility.

e) simultaneous filling in closed position - the meniscus is seen clearly a dark shadow (biconcave wafer) between the normal shapes of the upper and lower filled sulci. The posterior aspect of the meniscus merges imperceptibly with the posterior attachment.

f) simultaneous filling on jaw opening - the anterior sulci are obliterated but the dark shadow of the posterior part of the disk is depressed by the quantity of contrast medium in the upper sulcus so that the normal hump of filling material seen in the lower sulcus is not observed.



Blaschke [26] makes some important observations of normal joint space arthrography.

i. The relationship of the condyle to the thin portion of the disk is constant throughout opening and closing sequences of joint movement.

ii. The injected joint spaces are distended considerably beyond their physiological limit but the diagnostic information is not compromised.

Abnormal Joint Space Arthrograms [26]

a) anterior displacement of disk with reduction (i.e. clicking phenomena)

There is a rapid posterior recoil of the disk over the anteriorly moving condyle observed at the precise moment the patient experiences a click. In the closed mouth position, the disk is situated decidedly anteriorly to its normal anterosuperior position. The condylar articular surface is positioned just posterior to the thick posterior band of the disk. The lower anterior recess is elongated and more horizontal compared to normal, giving it a lobular appearance [47].

As the jaw opens and the condyle moves forward the disk is carried forward by a shearing force on the condyle (hesitation of normal movement). At some instant the posterior band of the disk recoils over the condylar articular surfaces and establishes a normal disk-condyle relationship. There is a harmonious relationship for the remainder of the opening sequence. The reciprocal click is basically a similar return to the abnormal disk position with the lower posterior recess returning to a less exaggerated sigmoidal form [47].

Toller [27] describes a condition of recurrent subluxation or dislocation. There is generally a normal arthrographic appearance of the upper and lower joint spaces in the closed position. On opening, the anterior sulci are obliterated but the meniscus translates further forward than normal indicating the presence of more extensive anterior sulci. This derangement

suggests synovial looseness and is more significant diagnostically in the upper joint compartment. The anterior recess of the lower joint space shows a concave impression and may assume an angled shape. The contrast material does not completely flow posteriorly from the anterior recess [41].

Dolwick et al [41], however, differentiates between dislocation and anterior subluxation (clicking) and his observations are concurrent with those described above by Blaschke [26] who also employed fluoroscopy.

b) anterior displacement of disk without reduction (i.e. locking phenomena).

A closed lock is an advanced abnormality with no reciprical click. Arthrography confirms the radiographic finding that the condylar translation is limited, but also gives an illustration as to its cause, i.e. obstruction by a non reducing anteriorly displaced disk [26].

In the closed position, the disk position is even further anteriorly placed in reference to the articular surface of the condylar head.

As the condyle goes through early and midrange translation it picks up the posterior rim of the disk and forces it further forward. At no point does the disk spontaneously recoil. At maximum opening, the disk physically blocks the condyle from achieving a normal excursion. The disk is often progressively deformed during such an opening lock sequence.

JAW CLOSED PARTIALLY OPEN MAXIMUM OPENING.

Toller [27] describes a tension deformity brought about by the condyle attempting to complete its excursion. Dolwick et al [41] also has shown that the lower anterior sulcus may not be obliterated and the posterior sulcus is pulled into a sharp tented shape on opening. When only the upper cavity is filled, the meniscus is seen to fail to complete its forward translation and it sticks. Transfer of contrast medium into the upper posterior sulcus is incomplete [27]. Sometimes the contour of the meniscus is seen to have a buckled and distorted appearance giving heed to the opinion that the symptom of locking is in fact an undischarged click. Farrar McCarty [47] support the

finding of a change in configuration of the usually sigmoid shaped lower posterior recess.

c) meniscal perforations.

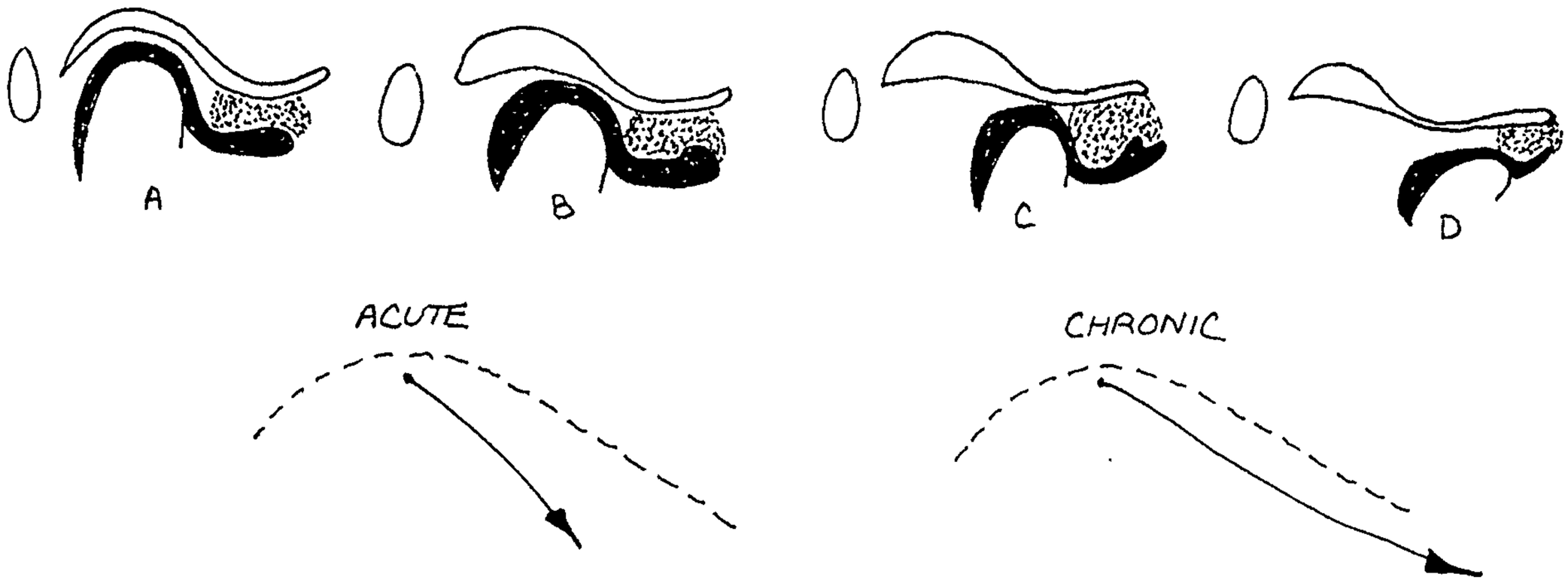
Perforation of the meniscus is easily recognised by simultaneous filling of the upper joint space during introduction of contrast medium into the lower joint space [41]. This characterises either perforation of the disk or posterior attachment. Farrar McCarty [47] point out, though, that this may be an artifact due to the need to pass a cannula through the superior joint space into the inferior joint space. However, Blachske [26] argues that if arthrography is performed properly the paths of the angiocatheter placement into the joint should not come near the disk and thus this iatrogenically induced condition should not occur.

d) degenerative changes and adhesions.

These are recognised by arthrographic means as decreased joint volume and multiple irregularities in the contours of the lower joint space [41].

e) abnormal condylar paths.

Farrar, McCarty [47] correlated inferior joint space arthrography and the characteristics of condylar paths in acute and chronic locking.



In acute dislocations of the disk, the range of movement is limited because the condyle is blocked by the disk (see A,B). The condylar path in recent dislocations is short and steep. As locking becomes more chronic the range of movement increases (see C,D) and the condylar path is normal.

Arthrographic Findings

Over the past ten years there have been a great many arthrographic studies performed. Some important observations have occurred which have led to the current concepts of the temporomandibular joint pain-dysfunction syndrome.

In Toller's 1974 survey [27], it was found that most arthrographs showed the features of normal joints, i.e. normal volumes, meniscal integrity, normal extent of capsular sulci and absence of adhesions. However, he concluded that in those abnormal arthrograms, periarticular fibrosis could restrict the extensibility of a synovial capsule and thus decrease the movement of the meniscus and the condyle.

There was a consistant arthrographic finding dealt with in several articles in 1979 [9, 2, 6], whereby, in 70% of those patients with temporomandibular joint pain and dysfunction had condylar retrusion.

Dolwick et al [41] in 1979 attempted to explain the significance of 'tenting' of the posterosuperior aspect of the lower joint space, as adhesions of the posterior meniscal attachment. Clinically they all had symptoms referable to the temporomandibular joint but there was no common finding. They found, however, that in cases of meniscal perforations, all had a history of trauma.

Katzberg et al [28] in 1981 found that most patients with anterior meniscal displacement had unilateral pain and limitation of opening. Subsequently, they found that leakage of contrast medium along the lateral condylar neck in patients with anterior meniscal displacement without reduction, suggested associated tearing of the lateral capsular attachment.

Bronstein et al [29] discussed his 1982 finding that 40% of patients with mandibular pain-dysfunction syndrome had anterior disk displacement and in many cases had a history of acute trauma. Fundamental to this conclusion were these arthrographic findings.

a) anterior meniscal displacement - the meniscus is observed to be forward of the condyle in closed jaw position and possibly showed some folding. However, while many primary and secondary anterior disk displacements show bilaminar redundancy on arthrography, they are often otherwise asymptomatic.

b) meniscal perforations - these typically are illustrated by the filling of both joint compartments, even though only one is entered.

c) posterior meniscal displacement - they found this entity was difficult to interpret arthrographically and clinically.

Ma Xu-Chen et al [42] (1983) made the observation that anterior displaced disks are often accompanied by rotation.

(a torque is set up by muscle action and the anterior part of the disk is displaced medially and the posterior part displaced laterally). Additionally the authors state that bony degenerative changes may occur in the temporomandibular joint in cases of disk displacement without any initial bony change. Similarly, joints both with and without bony changes can develop

communications between superior and inferior joint cavities (perforations) [21]. They hypothesised that when the disk is displaced anteriorly, the bilaminar zone may be stretched and struck repeatedly by the condyle during mandibular movements. Thistraumatised area of the disk becomes thin and leads eventually to a perforation or laceration of the disk and/or disk attachments. Farrar, McCarty [47] supports this view that the bilaminar zone is the most frequent site of disk perforation and that it is caused by anterior disk displacement in cases of no bony changes.

Most recently, Westesson [20] claims that results from his double contrast arthrography survey, confirm that the anteriorly displaced disk is the most frequent internal derangement of the temporomandibular joint. He proceeds to supply evidence that the 'clicking' mechanism is caused by a physical obstruction of the anteriorly displaced disk.

SUMMARY:

In reviewing the role of the temporomandibular joint radiography and arthrography in the diagnosis of temporomandibular joint problems, several features stand out for the clinician.

1. The importance of adequate criteria and data gathering for patient symptoms and clinical examination procedures.
2. A thorough knowledge of the anatomy and function of the temporomandibular joint(s).
3. The limitations of temporomandibular joint radiography and the advantages of each radiographic method.
4. The enhanced status of the tomogram for evaluation of the joint.
5. The beneficial results obtained with arthrography when functional abnormalities are under investigation.
6. The importance of strict criteria for the choice of arthrography of the temporomandibular joint.
7. The necessity to assess radiographically or arthrographically the dynamics of temporomandibular joint function.

Recent developments encourage an optimistic view for the future of "radiographic" examinations, as an aid in the diagnosis and treatment of temporomandibular joint abnormalities. Further, it emphasizes the frequency of temporomandibular joint disorders, and the need for suitable training and the availability of experienced consultants in all aspects of temporomandibular joint radiography and arthrography.

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PART 111

ROENTGENOGRAPHIC ANALYSIS OF DENTINAL CARIES

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Introduction

The objective of this survey was to assess the optimal exposure factors in the detection of early caries in dentine. Because of widespread fluoridation in the Australian community and in view of its specific effect on dental caries diagnosis, often decay in enamel may be undetected both clinically and radiographically. The rapid erosion of dentine with the concomitant loss of stability of the enamel shell in fluoridated teeth emphasizes the importance of detecting early carious lesions in dentine.

Ironically, though, most research thus far, has centred on mechanisms for detecting incipient carious lesions in enamel radiographically. The resultant radiographic factors invariably sacrifice the densitometric contrast of caries in dentine which is essential for diagnosis of decay.

Specifically, the determination of caries at the dentino-enamel junction is the prime objective of this survey. This may prove to be a more reliable method of diagnosing more carious lesions by radiographic means.

What radiographic determinants maximise the detection of caries in dentine? Several assumptions have been made prior to proceeding with this endeavour. Of primary importance was the implementation of the paralleling technique, using film holding equipment as the radiographic system of choice. It has overriding advantages compared with other techniques in terms of accuracy of projection geometry and reproducibility. It can be manipulated easily in the vast majority of patients. The second assumption involves the simulation of in vivo radiographic examination using a static artificial set up. These procedures will be discussed at length at a later stage.

Ennis & Berry [1] stated that 'any roentgenologic standard less than perfection, or an interpretation less than thoroughly accurate, defeats its own purpose and renders valueless the most perfect clinical diagnosis'. It is my endeavour in this study to obtain perfect radiographic diagnosis of dentinal caries through faultless methodology.

Pre-Survey Introduction

The Clinical and Radiographic Appearance of Dentinal Caries

Of immense importance is an understanding of the caries process before any radiographic study can be performed. It is noticeable that differing anatomical locations of dentinal caries (i.e. molars and incisors, occlusal and proximal regions) have varying histopathological patterns and therefore probably varying radiographic features.

Clinical Appearance:

Caries of dentine is characterised by progressive softening and staining with concomitant loss of structural integrity. It spreads rapidly at the dentinoenamel junction causing a large frontal area of demineralisation. It can be active, chronic or a combination of the two. There are four distinct layers present and visible in actively carious dentine.

a) a soft superficial surface layer - includes a narrow necrotic zone where plaque proliferates and is devoid of normal dentine (i.e. bacterial invasion and protein breakdown).

b) a zone of bacterial spread - this region is firm and rubbery with bacteria found in the dentinal tubules.

c) a decalcified deeper zone devoid of bacteria - there occurs acid demineralisation on an advancing front, and a wide normal tubular arrangement. However, when advanced, transverse cleftings of the dentinal tubules may occur.

d) a sclerotic zone - this region is narrow and difficult to see but is apparent in the arrested lesion. It outlines the decalcified layer and unlike the others is radiopaque radiographically. [2]

Backer-Dirks [3, 4,] states that caries develop slowly and on an average, it takes 3-4 years for a proximal enamel lesion to spread into detine.

Histological Appearance:

In the walls of dentinal tubules exist an acid mucopolysaccharide, a lipid and alkaline phosphatase and these organic compounds coincide with the primary pathway of decay. Soggnaes, Wislock [5] performed histochemical reactions on dentinal decay. They took radiographs before sectioning in order to establish the extent to which carious lesions had produced demineralisation. Their findings were confirmed later by Sarnat, Massler [6]. They concluded that 'once invaded, the organic matrix of the tooth slowly disappears. Thus, there is a widening of the dentinal tubules (distending) at the expense of the surrounding ground substance. Finally, the tubules may fuse with one another to produce familiar pictures of advanced caries (i.e. clefts, in which complete disintegration of the dentine has occurred)'

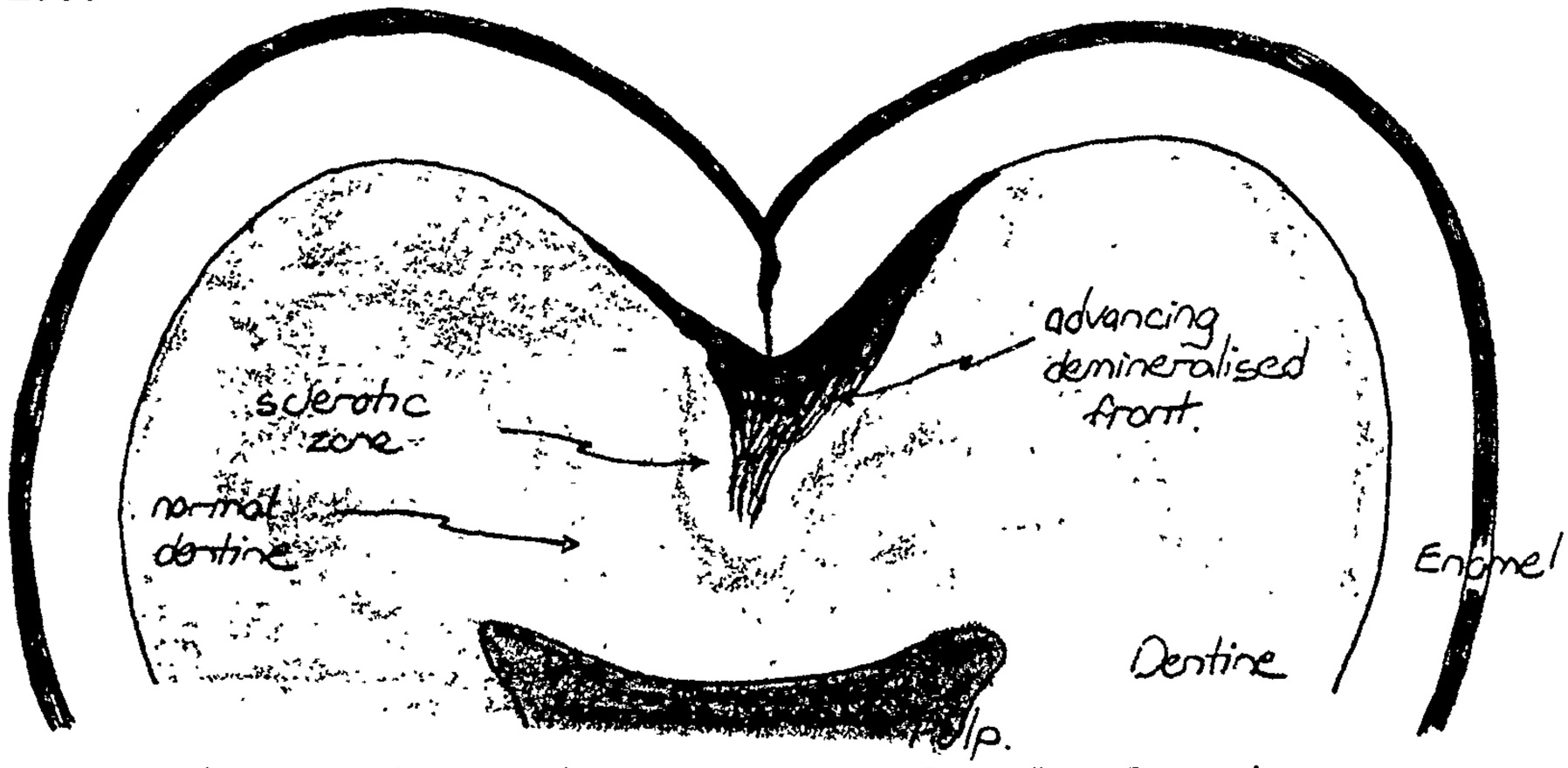
Caries of dentine is initiated by demineralisation and a change in acid mucopolysaccharides produces a loss of metachromatic and basophillic reactions normally present in the walls of dentinal tubules (Neumann's Sheaths), thus further causing their demise.

In Sognnaes' microradiographic study [7], it was found that in acute dentinal caries, the demineralisation extended a considerable depth beyond areas of microscopic breakdown of the organic matrix. At an early stage, low density areas appeared to proceed partly by way of incremental zones and along dentinal tubules so as to make them more apparent than in normal underlying dentine. In deeper zones, the walls of the dentinal tubules appear distended because of demineralisation of the adjacent matrix. He found a direct relationship between the relative microradiographic density and the concurrent microhardness.

Dentinal Layer		Relative Microradiographic Density	Microhardness (Knoop Number Average)
Subsurvace)		+	43
Central Zone)	Carious Dentine	++	52
Inner Zone)		+++	63
Intact Dentine		++++	75

Radiographic Appearance:

a) Interproximal lesions - there is a relatively small penetration usually observable through enamel. It may be the classical radiolucent triangular shape (with the base of the triangle being the original surface and the apex pointing toward the dentinoenamel junction), or it may be diffuse or a combination of both. There is a spreading of the demineralisation process at the dentinoenamel junction, undermining enamel and extending into dentine. This forms a second triangular radiolucent image in the dentine (with the base at the dentinoenamel junction and the apex directed toward the pulp). Some radiographs show only a thin line penetrating enamel lamellae with the usual lesion formation in dentine. Pulp exposures cannot be determined on the basis of radiographs alone, since a radiograph is a 2D image and the full extend of the carious progress may not be revealed or it may be exaggerated. One must remember that the examiner is looking at images caused by varying densities due to demineralisation and cavitation [8].



Typical Radiographic appearance of Occlusal Caries.

The Mach Effect:

The apparent density in the region of a common boundary between two areas on a radiograph that has received considerably different levels of exposure (relatively light, less exposed area adjacent to a dark more exposed area) will not show the expected density versus exposure relationship when viewed. There is an apparent enhancement of radiographic density at the edge of the denser region (relatively highly exposed area) and an apparent depression of the density just over the boundary in the less exposed part of the image. They may be sharp or gradual bands. They may be confused with occlusal caries at the dentinoenamel junction interface.

This optical illusion is produced by lateral inhibition of neural receptors in the eye, and may produce fictitious radiolucent areas, particularly in the following clinical regions -

1. Inside the proximal dentinoenamel junction in incisors and canines, and less frequently in premolars and molars.

11. In dentinal peaks bounded by occlusal and proximal enamel in premolars (especially in the mandible)[10].

Methodology:

In the setting up of a standardized and diagnostically relevent experiment with minimal errors associated with it, a complex series of pre-survey experiments and observations were undertaken.

Initially, 100 teeth were selected from those extracted from the Exodontics Department, United Dental Hospital, Sydney. The only pre-requisites which applied to their selection were that they were not grossly carious and that their roots were intact. Consequently, those teeth selected consisted both of non-carious teeth, and those showing clinical evidence of approximal and occlusal caries.

Evaluation of Suitable Teeth:

All teeth were examined radiographically using arbitrary values - 90kVp, 15mA, 25cm focal/object distance, and with the teeth sitting directly on the film. The exposure times were selected according to a "Procedure For Determining Optimal Exposure Times" [11]. This exercise determines the proper exposure times for intraoral films which will be satisfactory for all teeth. The above values are kept constant and by varying the time exposure on a selected tooth type (i.e. mandibular molars) a sequential series is produced which can then be subjectively examined for the most diagnostically accurate exposure time. (See Appendix A). The best exposure time for the mandibular molar group was 0.64 seconds. This value is inserted into the predetermined equations in a "Procedure For Determining Optimal Exposure Times".

Tooth Group	% Increase or Decrease From Mandibular Molar Time	Calculated Exposure Time (secs.)
Mand.molars	0	0.64
Mand.bicuspids	-15	0.50
Mand.cuspids	-33	0.40
Mand.incisors	-50	0.32
Max.molars	+33	0.80
Max.bicuspids	0	0.64
Max.cuspids	0	0.64
Max.incisors	-15	0.50

Of course, most x-ray machines have a limited range of exposure times. The General Electric machine which was employed in the survey proper (see Photograph A) has the following exposure gradations: 0.15, 0.20, 0.25, 0.30, 0.35, 0.40, 0.50, 0.60, 0.80, 1.00, 1.25, 1.50, 1.75 seconds. Consequently, minor variations to the optimal exposure times were observed, when the subsequent survey of these individual tooth groups was

carried out.

These radiographs were examined for the presence of dentinal caries (see Appendix B). Suitable teeth were selected and further sub-grouped into those showing occlusal caries, proximal caries, occlusal and proximal caries, or non-carious control teeth. Eliminated were those teeth with buccal or lingual carious lesions or gross caries extending into the pulp. Those teeth with a certain ambiguity as to whether caries had extended into dentine were certainly included.

Overall, 43 carious and 10 non-carious teeth remained. They were numbered and mounted in plaster blocks. This selection consisted of 21 molars, 20 premolars, 6 canines, 6 incisors. Each were clinically evaluated, using a good light source and a probe, for proximal cavitation, occlusal sticky fissures or subsurface demineralisation. This information was transferred to a reference chart, and the following categories were established with the bracketted numbers indicating how many teeth existed in each.

Non carious control (10)
Cavitation and proximal caries radiographically (9)
No cavitation but proximal caries radiographically (13)
Sticky fissure and occlusal caries radiographically (18)
No sticky fissure and occlusal caries radiographically (2)
With clinical and radiographic evidence of proximal and occlusal caries (1)

The ultimate make-up of teeth for the study was designed to incorporate teeth of all these categories. Further pruning was undertaken to achieve a manageable experimental number of 18 teeth. Of these 18 teeth, 7 were molars, 7 were premolars and 4 were either canines or incisors. One control tooth was included in each category. Of those remaining 15 teeth, 7 had evidence of proximal dentinal caries, 7 had evidence of occlusal dentinal caries and 1 had evidence of both.

Radiographic Equipment and Materials

The prime objectives are to most closely simulate the clinical situation and to satisfactorily standardize all aspects of experimentation. In this regard, Benkow [12] listed factors which must potentially be kept constant: film/object distance, source/film distance, x-ray beam angulation, film density, film contrast, position of the film in relation to the object.

1. Use of the Paralleling Technique: This method was chosen as it incorporates the most accurate imaging principles. It can be more readily standardised with the film and teeth being parallel. Because it involves a long cone (increased source/film distance) rays are more parallel. It also decreases the chance of image shape distortion (unequal magnification) which is seen in the bisecting angle technique.

The paralleling technique employs a film holder. This avoids cone cutting (the film is kept exactly in the centre of the beam), it reproduces distances and controls the geometric relationship between the film, object and x-ray source. It also avoids film displacement and bending [13,14].

Additionally, the use of collimation restricts the x-ray beam to a size slightly larger than the film. Thus it reduces scatter and the likelihood of fogged films. Highly collimated beams have superior quality, give high contrast radiographs with better definition and decrease dose irradiation. Radiographs of high quality are necessary in dental radiography if small differences between healthy and pathologically altered tissues are to be detected [15].

Most epidermiological surveys employ a fixed rigid connection between the cone and film holder to eliminate movement [16]. However, with the experimental use of the Rinn XCP model an arbitrary connection is employed.

Pitts [17] gives objectives of film holders and aiming devices:

a) quick, practical, easy to use
b) supplies additional collimation to reduce close exposure
c) fixed, rigid system with the film flat and perpendicular to the central beam
d) may incorporate a step wedge

He states that 'comparisons are only valid if films are standardized with respect to angulation, distance, and exposure and processing. The use of film holders and aiming devices greatly assists standardisation of variables'.

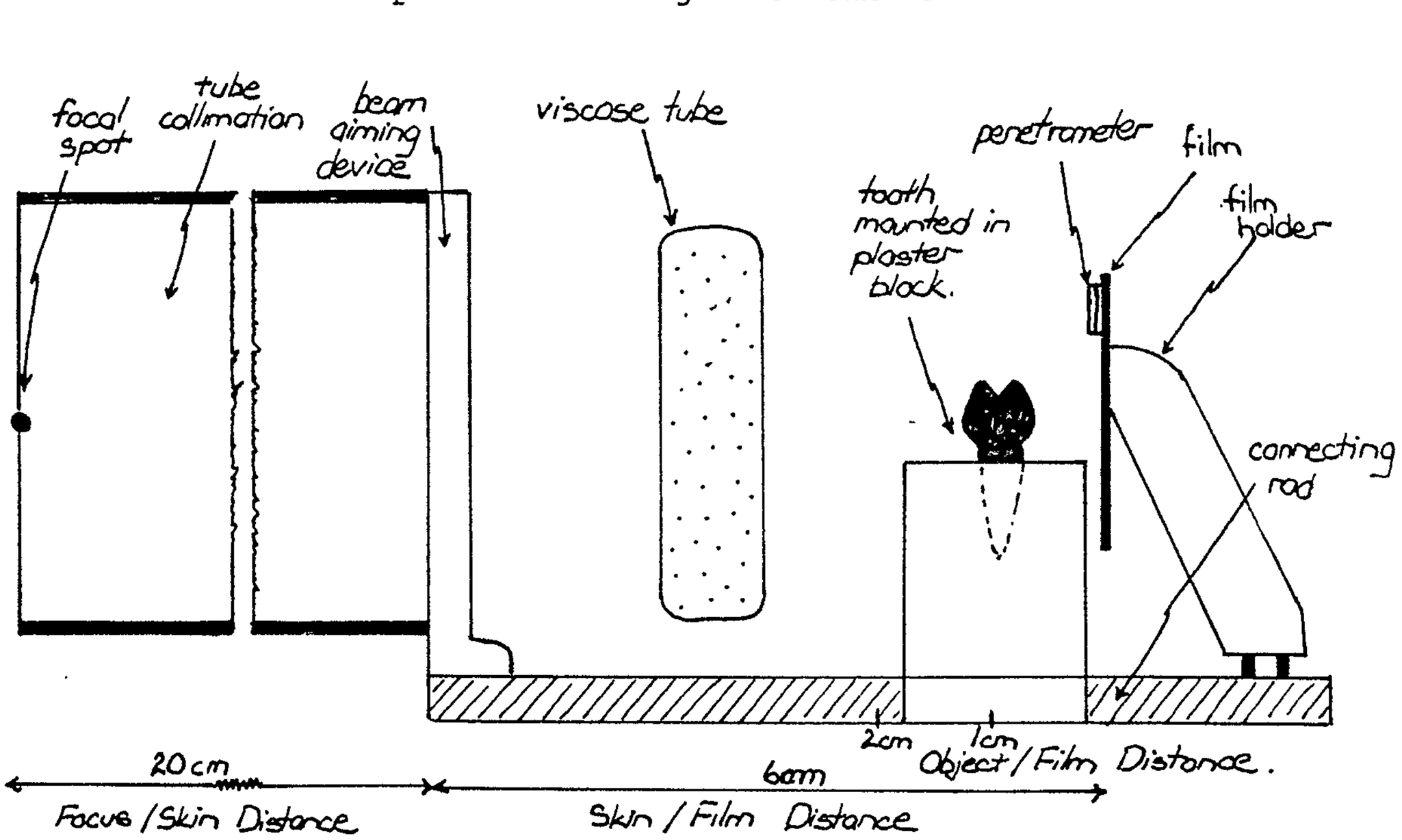
11. Type of Film: The film most widely used in the United Dental Hospital, Sydney for intraoral purposes, is Kodak DF58. Films from the same batch were used and storage was provided in a light-proof area away from radiation. These fast films may not produce as sharp an image as slow fine-grained films, but are required, due to the excessive exposure times needed for slow films in intraoral radiography.

111. Soft Tissue Simulation: A study by Hedin,,Halse [15], used 1 cm of water to simulate the scattered radiation from a patient's soft tissues. In this experiment, a viscose tube full of water was positioned between the collimator and the tooth.

lV. Use of a Penetrometer: A step-wedge determines whether the film has a high (short scale) or low (long scale) contrast. It correctly records the actual rays used and their suitability. For epidermiological surveys, it produces a suitable range of densities comparable to those produced by oral tissues. It opens the possibility for quality control by either visual or densitometric means because differences may effect the diagnostic threshold of an observer. It may also allow measurement of the degree of demineralisation of carious lesions. A step-wedge was affixed to each film in the survey.

V. The X-ray Machine: Focal spot size 1 m.m., Filtration 2.5 m.m. Al equivalent, Beam diameter 2.75", Type General Electric 100.

Constant angulation and distance between the film and tooth were thus ensured. The plaster mounting block can be



Diagramatic Representation Of Experimental Set-Up

placed at any required distance along the connecting rod. For the purposes of the study, the object/film distance was varied between 1 or 2 cm. Similarly with the addition of extra cone length, the focal/skin distance can be varied between 20 cm and 30 cm. (see photograph B).

A Method of Determining A Range Of Experimental Exposure Times:

With the standardisation of equipment reproducibility is assured and a range of acceptable exposure times for a varying tooth type and kVp can be attained, which is the basis upon which this survey is schemed. The constant values in this assessment were as follows: 10mA, 20 cm source/skin distance, 2 cm film/object distance.

One representative tooth from each category was selected. A series of 5 progressive exposures at each kVp was carried out and subsequently a subjective decision was made by 2 examiners as to the optimal exposure time. The exposure times included 0.20, 0.25, 0.35, 0.50, 0.80 and in some instances 1.25 seconds. Handicapped by the machine's inherent exposure time gradations, these values were the closest estimation for a scheme of increasing the exposure time by one half each time.

The radiographs were viewed in a darkened room with a viewbox and magnifying glass (see Photograph C) bearing mind the fundamentals of optimal image detection and with the aid of the penetrometer reading one can assess optimal contrast. X-ray machine gradations immediately above and below this optimal value (x) were incorporated into the tables as $(x)^{-}$, $(x)^{+}$. Optimal exposure values of 1.00 seconds or more were deemed to be inappropriate for clinical implementation and on the whole, kVp's with these high exposure time values were eliminated from the survey.

Thus the following values represent the experimental variables which were to be used in the experiment proper on each of the survey teeth.

Obviously, if we apply the inverse square law when we change the source/film distance from 26 cm to 36 cm, a similar table can be formulated for those values.

TABLE 1 - Canine/Incisors

Range of exposure times (secs.).

* Require too high an exposure.

		kVp					
		50	55	60	70	80	90
20 cm source/ skin distance	$\bar{x}$			0.6	0.6	0.4	0.3
	x(opt)	*	*	0.8	0.8	0.5	0.35
	x^+			1.0	1.0	0.6	0.4
30 cm source skin distance	$\bar{x}$				1.25		0.5
	x(opt)	*	*		1.5		0.6
	x^+				1.75		0.8

TABLE 11 - Premolars

(Range of exposure times (secs.))

* Require too high an exposure

		kVp					
		50	55	60	70	80	90
20 cm source/ skin distance	$\bar{x}$				0.6	0.4	0.4
	x(opt)	*	*	*	0.8	0.5	0.5
	x^+				1.0	0.6	0.6
30 cm source/ skin distance	$\bar{x}$				1.25		0.8
	x(opt)	*	*	*	1.5		1.0
	x^+				1.75		1.25

TABLE 111 - Molars

(Range of exposure times (secs.))

* Require too high an exposure

		kVp					
		50	55	60	70	80	90
20 cm. source/ skin distance	$\bar{x}$			1.0	0.6	0.6	0.4
	x(opt)	*	*	1.25	0.8	0.8	0.5
	x^+			1.5	1.0	1.0	0.6
30 cm source/ skin distance	$\bar{x}$				1.25		0.8
	x(opt)	*	*		1.5		1.0
	x^+				1.75		1.25

Only 70 and 90 kVp values were used in the comparison of varying source/film distances.

Processing Standardisation

Automatic processing is efficient and prevents darkroom errors which are more prevalent in fast films. According to Buckholz [18], the 'manufacturers directions should be followed in absolute detail as it critically effects quality and standardisation'. Unfortunately, in the United Dental Hospital's Processing Department, there exists an unhealthy disregard for Buckholz' principles and one can only speculate as to the reproducibility of solution concentrations and temperatures from day to day. Nevertheless, an endeavour was made to process films in as few visits as possible and then only when the processing solutions were fresh.

At a subsequent date all radiographs were viewed under the standardized conditions previously described employing random labelling of the films. The two examiners observed all of the fundamentals of examiner reliability required for such a trial.

Methods of Reducing Examiner Error

The following procedures were employed in an endeavour to enhance examiner reproducibility. Experienced scientists such as Backer-Dirks [3] recommend four observers. However, logistics demanded this experiment be limited to two persons.

The observers' results can deviate markedly [19,20].

Initially, a rigorous calibration exercise was undertaken by the two examiners prior to the survey. Random films were assessed by group consulatation in an endeavour to eliminate diagnostic reversals and obtain consistency between the examiners. Many investigators [21, 22, 23] have acknowledged the value of standardised readings. Absolute concordance between observers may not be reached, but minimising this discordance through calibration is possible [24].

Grondahl [25] emphasized the necessity to remove any biases prior to examination. Apart from randomly numbering the films, this was achieved by -

a) preventing the use of the clinical evaluation as a reference.
b) making a conscious effort not to read literature relating to the experiment before the examination.
c) by implementing total removal of the examiners from the experiment for one month before the examination took place.
d) preventing any assessment or computation of the results until all viewing had ceased.

Many experimenters have examined the need for a 'control' group to supplement the 'test' group [26]. A non carious control tooth was placed in each test group and examiner error tested by this yardstick.

Random sampling and viewing was based on the Blind Technique [26], whereby the individual films are randomly numbered and a coded template keeps the data for later analysis.

Rugg-Gunn, Holloway [22] found that random error was far greater than systematic error. However, his suggestion that 1 in every 10 films be randomly re-examined was not applied to this investigation.

Only diagnoses made with absolute certainty were counted. This decreases the chances of false positives [27].

Additionally, a conscious effort was made to only assess films when the examiners were alert and fresh. Obviously, in a survey of over 500 films, this is an extremely influential variable. Haugejorden [27] stated that 'there is a need for a critical acceptance level for diagnostic performance of examiners when diagnosing caries. In addition to the problem of reproducibility of diagnosis, it is important to maintain a constant diagnostic level throughout the duraction of clinical trials'.

Radiographic Viewing Equipment and Viewing Conditions

Basically, the equipment involved included a view box, magnifying lens and a light masking screen (see Photograph C). Analysis was undertaken in a darkened room free from distraction. The assumption is made that if the number of extraneous signals to the retina is decreased, then one's capacity to perceive pertinent information will be enhanced.

Unfortunately, a variable intensity viewbox and 'hot light' were not available. It is believed that these increase grey scale discrimination and point resolution to enable the evaluation of those radiographs which are slightly overexposed and allow the determination of the usefullness of those which are underexposed [28].

The light masking screen was fabricated out of exposed panoramic films and a hole the size of the films to be examined was cut into it. It is perceived that this masking of extraneous light stops eye weariness and maximises grey level discrimination diagnosis.

The following instructions were given to the examiners prior to their evaluation of the random films. The dot on the films was always placed on the top right corner.

'Observe the following radiographs and detect on the occlusal and proximal surfaces, radiographic caries, if present, according to this scoring system.[29]

0	Non carious
1	lesion confined to enamel
2	lesion extending to the dentino enamel junction
3	lesion extending to the outer half of dentine
4	lesion extending to the inner half of dentine

Assess the film subjectively for its diagnostic value according to the following scoring system

+	Poor
++	Fair
+++	Good
++++	Excellent

Inter and Intra X-Ray Machine Variability Trial:

An assessment was made of the inherent reproducibility of the General Electric 100 machine which was used in the experiment. Simply, three separate films were taken of a penetrometer at exactly the same exposure, kilovoltage and source/film distance values. A densitometer measured the density at each of the gradations on the step-wedge in each of the films.

Similarly, films were taken employing the other machines available for use in the radiology department. There were two other General Electric 100 machines and a Siemens Heliodent 70 machine. However, because the Siemans machine only ran at 7mA, the exposure time had to be altered from 0.35 secs for the 10mA machine, to 0.50 secs. All radiographs were taken at 70kVp with a source/film distance of 26 cm.

The intra and inter machine reproducibility can be referred to in the results section in the appropriate tables.

Results from both examiners were uncoded and transferred to a large table "Visual Grading Results". The processes of statistical evaluation to conclude any significant findings were undertaken.

Results

1 Analysis of the Diagnostic Value of the Film

Each film was assessed by each examiner for its diagnostic value, according to the scoring system outlined previously.

+	Poor
++	Fair
+++	Good
++++	Excellent

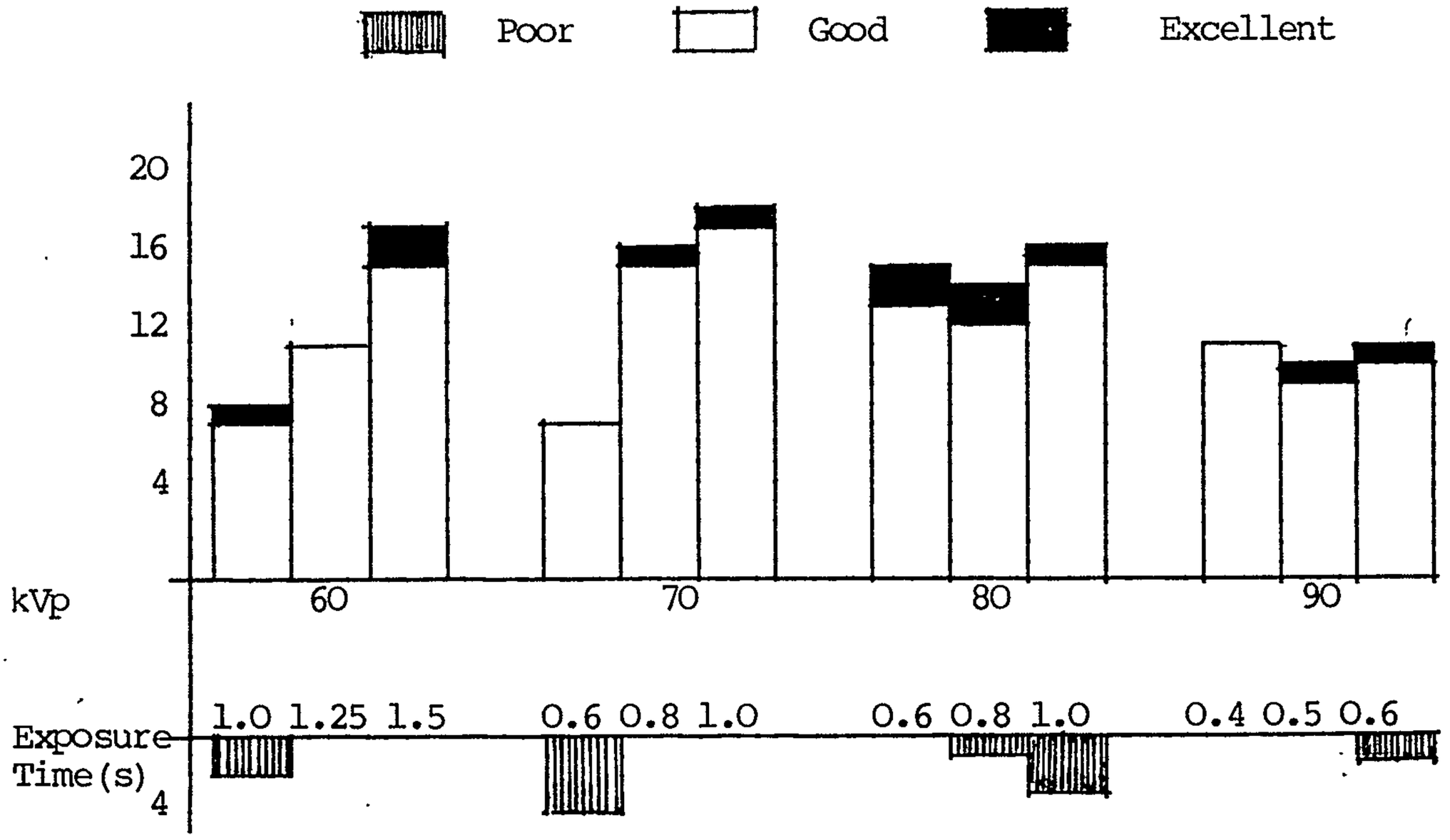
For convenience let us consider only those results which we deem pertinent to this experiment, i.e. in the endeavour for 'Good' and 'Excellent' results and the elimination of 'Poor' results.

If we assume that clinically altering the film/object distance from 1 cm to 2 cm will not effect the standard of the film, providing all other values are kept constant, we can statistically combine the two sets of scores to enable the detection of a perceivable trend. Similarly, we can combine the scores of both examiners.

A comparison can also be made between source/skin distances of 20 and 30 cm. It may be significant that more parallel rays give better diagnostic films.

a) Molars

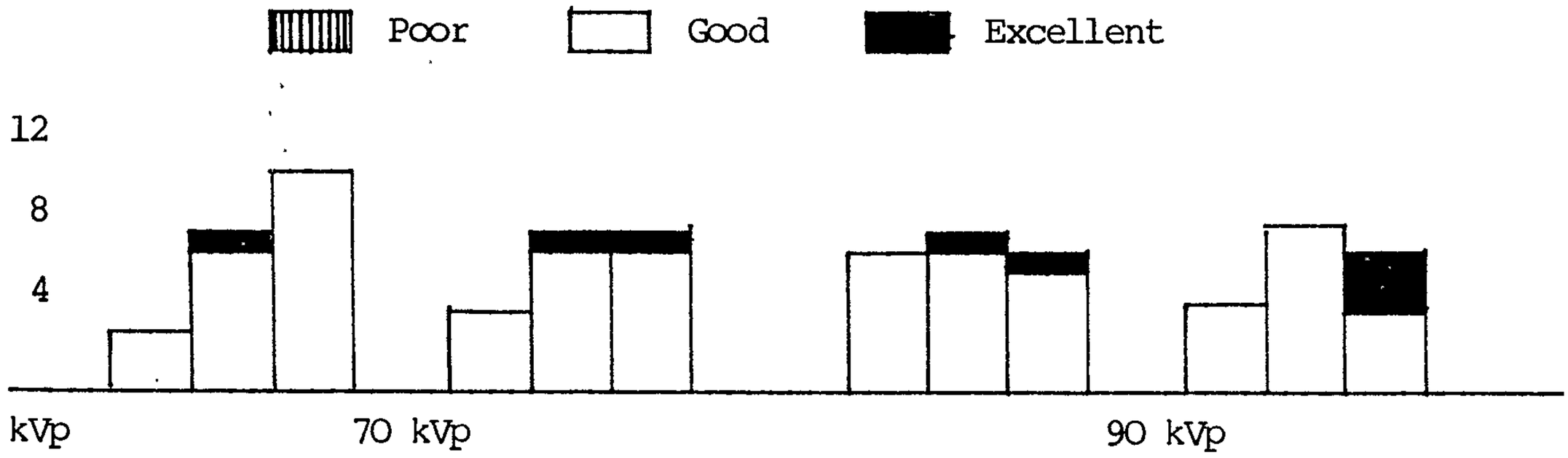
Table 1. Film quality assessment at 1 or 2 cm film/object distances with a constant 20 cm source/skin distance.



Assuming that a realistic clinical exposure time does not exceed 1.0 secs. best overall results were achieved at the following values.

60 kVp - none
70 kVp - 0.8, 1.0 s.
80 kVp - 0.6, 0.8, 1.0 s.
90 kVp - 0.4 s.

Table 2. Comparison of 20 and 30 cm. source/skin distances with a constant film/object distance of 1 cm.



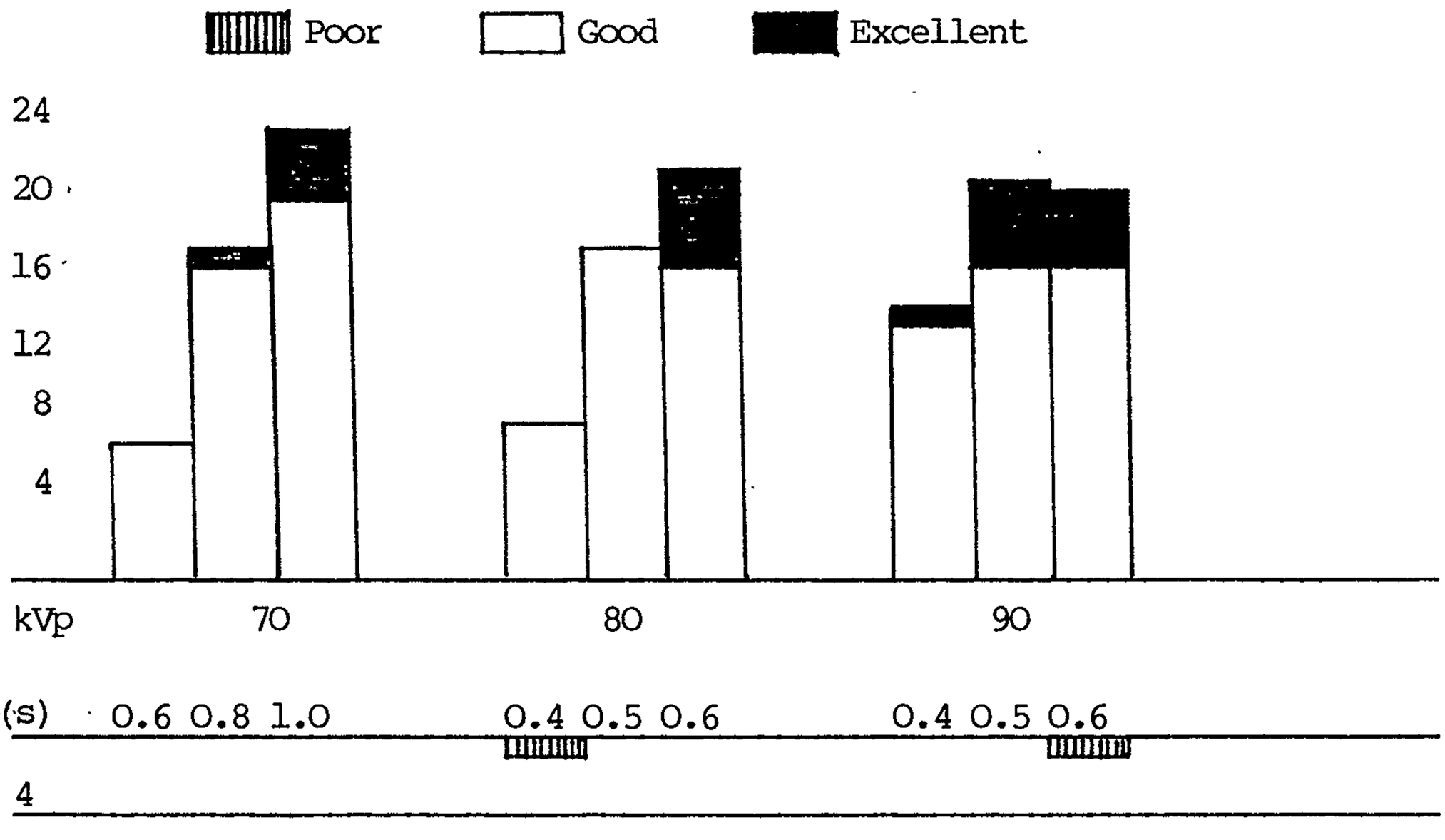
Similarly, at 30 cm. source/skin distance, good overall results were achieved at:

70 kVp - none
90 kVp - 1.0 s.

There are no significant trends in comparing 20 and 30 cm. source/skin distances in molars. The optimal exposure times have consistant findings for each kVp. It is apparent that in the 70 kVp range increased film quality occurs along with increased exposure time.

b) Premolars

Table 3 Film quality assessment at 1 or 2 cm film/object distances with a constant 20 cm source/skin distance.

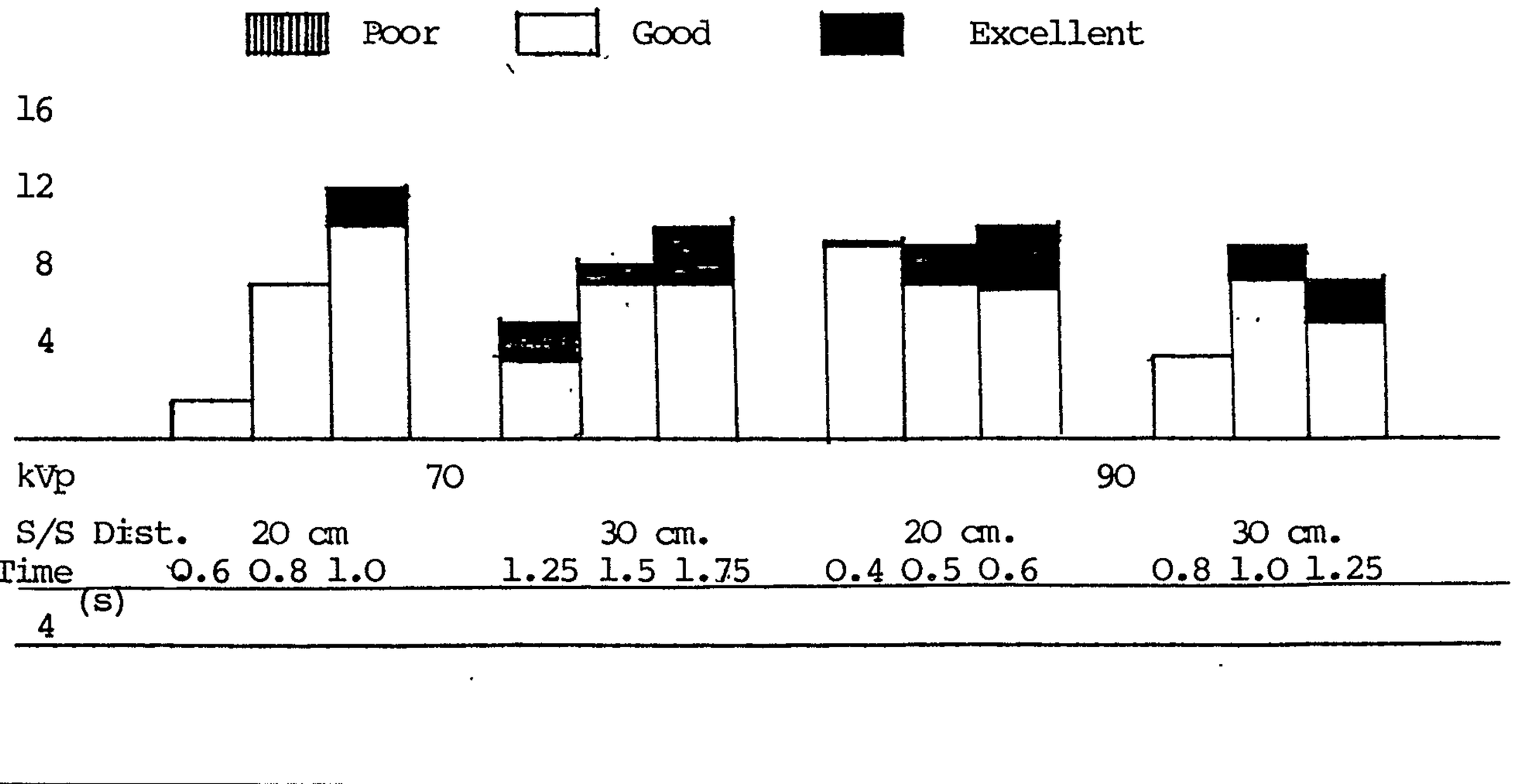


Best overall results were achieved at the following values.

70 kVp	0.8, 1.0 s
80 kVp	0.5, 0.6 s
90 kVp	0.5, 0.6 s.

It is significant that the most diagnostically valueable results were assessed in the higher range of exposure times used.

Table 4. Comparison of 20 and 30 cm. source/skin distances with a constant film/object distance of 1 cm.



Similarly, at 30 cm. source/skin distance good overall results were achieved at:

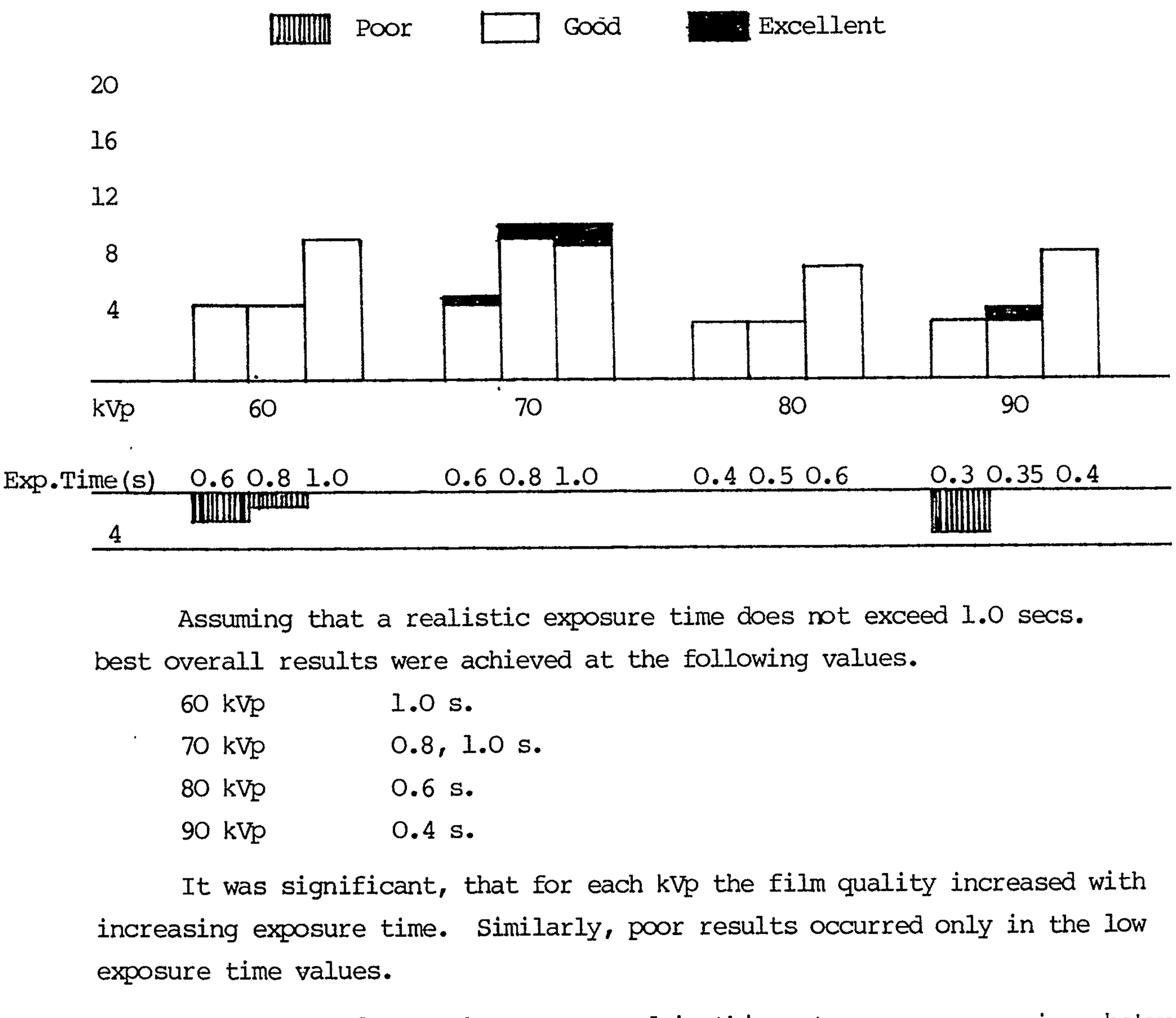
70 kVp - None

90 kVp - 1.0 s.

There are no significant trends in comparing 20 and 30 cm. source/skin distances. However, it is apparent in the 70 kVp range that film quality increases with increased exposure time.

c) Canine/Incisors

Table 5. Film quality assessment at 1 cm or 2 cm. film/object distances with a constant 20 cm. source/skin distance.



Assuming that a realistic exposure time does not exceed 1.0 secs. best overall results were achieved at the following values.

60 kVp 1.0 s.

70 kVp 0.8, 1.0 s.

80 kVp 0.6 s.

90 kVp 0.4 s.

It was significant, that for each kVp the film quality increased with increasing exposure time. Similarly, poor results occurred only in the low exposure time values.

Because, so few teeth are assessed in this category, a comparison between 20 and 30 cm source/skin distances, is not justified. However, in the sample tested, there appeared to be little variation in film quality between the two.

11. Analysis of Occlusal Caries in Dentine

All teeth were examined radiographically for the existance of and depth of penetration of occlusal caries according to the scoring system outlined in the Methodology. All raw data can be reviewed in the relevant tables in Appendix C. The only relevant scores are those of 2 or above, i.e. of those showing radiographic evidence of caries at the dentinoenamel junction or beyond.

a) Molars:

Clinically, 6 showed evidence of occlusal caries and 1 was a control. Again, for convenience, the scores of both observers are added together so that in effect 14 teeth are examined with (12) showing clinical evidence of decay.

Table 6. Evaluation of Occlusal Caries in Dentine in Molar Teeth.

Where: S/S Denotes source/skin distance

F/O Denotes film/object distance

Exposure Times		20 cm S/S 1 cm F/O	20 cm S/S 2 cm F/O	Exposure Time(s)	30 cm S/S 1 cm F/O
60 kVp	1.0	7	10		
	1.25	10	9		
	1.5	9	9		
70 kVp	0.6	7	9	1.25	8
	0.8	9	8	1.5	10
	1.0	9	10	1.75	10
80 kVp	0.6	11	10		
	0.8	11	11		
	1.0	9	9		
90 kVp	0.4	9	9	0.8	9
	0.5	11	11	1.0	10
	0.6	10	9	1.25	9

There are several significant features in the radiographic analysis of occlusal caries in molars.

- There was no significant variation between 30 cm S/S and 20 cm S/S distance values at 1 cm F/O distance.
- A greater number of lesions which extended to the DEJ were picked up at the 80 kVp, 90kVp, and high 70 kVp ranges.
- The optimal exposure time for each kVp (x) distinguished more lesions extending to the DEJ than (x^-) (x^+) values in most instances.

In a closer analysis of the individual observers' results (Table 7), there was an increase in radiographic performance as the exposure time was increased at lower range kVp's in the more experienced observer and in the other observer, lower range kVp,s appeared to be quite variable in performance.

Table 7. Comparison in Observer Performance, at 20 cm. S/S distance.

Observer	Exposure Time (s)	60 kVp 1.0	1.25	1.5	70 kVp 0.6	0.8	1.0	80 kVp 0.6	0.8	1.0	90 kVp 0.4	0.5	0.6
1	1 cm F/O dist.	4	5	5	4	4	5	6	6	6	5	6	5
	2 cm F/O dist.	4	5	6	4	5	6	6	5	5	5	6	5
2	1 cm F/O dist.	3	5	4	3	5	4	5	5	3	4	5	5
	2 cm F/O dist.	6	5	3	5	3	4	4	6	4	4	5	4

b) Premolars

Of the 7 premolar teeth in the survey, 4 showed no clinical evidence of caries (1 was a control) and 3 showed evidence of caries. This represents (6) teeth with caries if both observers' are combined.

Table 8. Evaluation of occlusal caries in dentine in premolar teeth, where -

S/S denotes source/skin distance
F/O denotes focal/object distance
Bracketted numbers are those occasions where lesions extending to the DEJ were found radiographically, but not clinically.

	Exp. Time(s)	20 cm S/S 1 cm F/O	20 cm S/S 2 cm F/O	Exp. Time(s)	30 cm S/S 1 cm F/O
70 kVp	0.6	4 (2)	5 (1)	1.25	3
	0.8	4 (2)	5 (2)	1.5	4
	1.0	7 (4)	5 (3)	1.75	4
80 kVp	0.4	5 (2)	4 (2)		
	0.5	6 (3)	6 (3)		
	0.6	5 (2)	3 (1)		
90 kVp	0.4	5 (2)	4 (1)	0.8	4
	0.5	5 (2)	6 (3)	1.0	3
	0.6	3 (1)	3 (1)	1.25	4

Significantly, best results were achieved at the following values -

70 kVp 1.0 s.
80 kVp 0.5 s.
90 kVp 0.5 s.

These values included many lesions which were only apparent by radiographic observation, i.e. could have been clinically evident as subsurface

demineralisation.

However, many lesions which were apparent clinically, were missed radiographically.

c) Canines/Incisors

No results were compiled for this category due to the obvious infrequency of occlusal decay in these teeth.

111. Analysis of Approximal Caries in Dentine

All teeth were examined radiographically for the existance of and the depth of penetration of approximal caries according to the scoring system previously outlined. All raw data can be reviewed in the relevant tables in Appendix D.

Again, only scores of 2, 3, 4, are considered as this represents decay at the DEJ or beyond.

a) Molars

Of the 14 proximal surfaces in the 7 molar teeth, only 2 showed clinical evidence of cavitation, and 6 showed evidence of subsurface demineralisation. If both observers' results are combined this represents (4) surfaces with cavitation.

Table 9. Evaluation of Approximal Caries in Dentine in Molar Teeth.

	Exposure Time (s)	20 cm S/S 1 cm F/O	20 cm S/S 2 cm F/O	Exposure Time (s)	30 cm S/S 1 cm F/O
60 kVp	1.0	3	4		
	1.25	5	5		
	1.5	3	5		
70 kVp	0.6	3	4	1.25	4
	0.8	4	5	1.5	4
	1.0	6	6	1.75	5
80 kVp	0.6	4	7		
	0.8	6	6		
	1.0	5	4		
90 kVp	0.4	5	3	0.8	6
	0.5	5	5	1.0	5
	0.6	5	4	1.25	4

Best results were achieved at the following values, assuming that a realistic exposure time does not exceed 1.0 secs.

60 kVp	None
70 kVp	1.0 s.
80 kVp	0.6, 0.8 s.
90 kVp	0.5 s.

Again, in many instances, caries was found to extend to the DEJ in those teeth which did not show clinical evidence of cavitation. These lesions may be clinically apparent as regions of subsurface demineralisation.

Significantly, in most instances, the optimal exposure time [(x)] gave the most diagnostic results.

b) Premolars

Of the 14 proximal surfaces in the 7 premolar teeth, 5 showed clinical evidence of cavitation and 5 showed subsurface demineralisation. When both observers' results are combined, this represents (1) surfaces with clinical cavitation.

Table 10. Evaluation of Proximal Caries of Centine in Premolar Teeth.

	Exposure Time (s)	20 cm S/S 1 cm F/O	20 cm S/S 2 cm F/O	Exposure Time(s)	30 cm S/S 1 cm F/O
70 kVp	0.6	7	8	1.25	9
	0.8	11	10	1.5	10
	1.0	10	11	1.75	9
80 kVp	0.4	10	8		
	0.5	8	9		
	0.6	10	10		
90 kVp		8	8	0.8	9
		8	11	1.0	12
		10	13	1.25	11

Significantly, best results were achieved at the following values:

70 kVp	0.8, 1.0 s.
80 kVP	0.6 s.
90 kVp	0.5, 0.6 s.

In many instances, the radiograph picked up those surfaces which showed subsurface demineralisation, as lesions extending to dentine.

c) Canine/Incisors

Of the 8 proximal surfaces on the 4 teeth in this category, there was evidence of clinical cavitation on 4 surfaces and subsurface demineralisation on 1. This represents (8) surfaces with clinical cavitation if both

observers' results are combined.

Table 11. Evaluation of proximal caries of dentine in canine and incisor teeth.

Exposure Time (s)		20 cm S/S 1 cm F/O	20 cm S/S 2 cm F/O	Exp. Time (s)	30 cm S/S 1 cm F/O
60 kVp	0.6	9	8		
	0.8	8	6		
	1.0	9	8		
70 kVp	0.6	7	8	1.25	8
	0.8	8	8	1.5	8
	1.0	8	8	1.75	8
80 kVp	0.4	8	8		
	0.5	8	8		
	0.6	8	8		
90 kVp	0.3	8	8	0.5	8
	0.35	8	8	0.6	8
	0.4	8	8	0.8	8

These results are significant in their reproducibility. It would seem that these teeth with small buccal-lingual widths have less variation in radiograph evidence of clinical caries.

However, at the lower range 60 kVp scale, there does appear to be some variability.

In all control teeth, there were no false positives of lesions extending to the DEJ.

lV. Analysis of Inter and Intra X-Ray Machine Variability

Machine A - General Electric 100

	Densitometric Readings Along Step Wedge				
Radiograph 1.	0.35	0.30	0.22	0.09	0.01
Radiograph 2.	0.35	0.28	0.20	0.09	0.01
Radiograph 3.	0.34	0.28	0.20	0.08	0.01

Machine B - Siemans Heliodent 70

	Densitometric Readings Along Step Wedge				
Radiograph 1.	0.26	0.20	0.12	0.04	< 0.01
Radiograph 2.	0.26	0.20	0.11	0.03	< 0.01
Radiograph 3.	0.26	0.20	0.12	0.04	< 0.01

Film density can be measured as a logarithm of the intensity of light transmitted through a radiograph [30] and it is possible to plot graphically its diagnostic limits [31].

Both machines exhibited a functional intramachine reproducibility with perhaps the Siemans Heliodent 70 machine being slightly better in this regard. Any intramachine variation of this magnitude can be generally disregarded as being acceptable by experimental standards.

However, the Siemans machine produced a more dense radiographic pattern of the step wedge. It would appear that, even by naked eye observation of the radiographs, that this machine produced x-rays with greater penetration than the General Electric Model. Considering that the mAs and source/film distance were kept constant in both these machines, the lower penetration power of x-ray produced by the General Electric machine is significant.

Discussion:

It is essential to review the factors which give a radiograph optimal diagnostic value and to relate these factors to the results of this experiment. An appraisal of the methodology, its inherent flaws and advantages and the way they may have affected the results, is paramount to achieving a realistic evaluation of the experiment as a valid document.

1. Effect of Altering Radiographic Variables

In effect, to achieve the optimal diagnostic aid from a radiograph, a juggling act must be performed between the kVp, mAs, source/skin distances and film/object distances, to enable the proper balance of contrast (grey scale discrimination([32], density (degree of film darkness) and latitude (the range in which a dental film can be over or under-exposed and still produce a diagnostically valid result).

. kVp - Kilovoltage potential.

The kVp indicates the quality of the x-ray beam. It controls the velocity, wavelength and penetrative power of the rays and thus effects the contrast and density.

Wuerhmann, Curby [33] in 1952 attempted to establish the kVp that would produce penetration of the maximum thickness of enamel and dentine in vitro without overexposing the minimum thickness of soft tissue. The most suitable mAs for each kVp was selected and the experiment carried out intraorally. They found that an increase in kVp causes an increase in the range of structural density that can be demonstrated on one film. They found that the optimum kVp was 95 to attain the greatest amount of perceptable density differences.

However, Buckholz [18] held the contrary view; that 65 kVp has greater diagnostic value. He explained that "the longer scale low contrast radiographs made with 90 kVp seems to have overall fogging and this wide range of greys complicates the interpretation of small images. The shorter scale, high contrast radiograph at 65 kVp has greater visual difference between black and white with fewer shades of grey.

Wuerhmann, Manson-Hing [9] concludes that 'lower kVp's are indicated where deviations of small structures are to be studied because of limitations of the human eye to determine small deviations of grey in small areas under high kVp's". Furthermore, they suggest that 'the diagnostic value shouldn't be jeopardised by the concept that the patient receives a marked longer radiation dose of low kVp'. Wainwright [34] concludes that "it is now known

that the sum of primary and secondary radiation is also identical for exposures made at both high and low kVp'. However, Goaz and White[35] maintain that by changing the kVp from 65 to 90, it is possible to reduce incident skin exposure by 40%.

Updegrave [36] who did considerable work in this field, recommends that the long scale, low contrast high kVp technique will improve detail rendition at the extremes of the range of absorptions but will sacrifice some detail visibility in the intermediate regions. However, when compared carefully under proper viewing conditions, the greater range of interpretive details seen in low contrast radiographs enhances diagnostic value. Furthermore, the use of 90 kVp is advantageous for portrayal of not only denser structures of the dentoalveolar region but also thinner and more penetrable regions. Since exposure latitude becomes greater as the contrast scale increases, 90 kVp produces radiographs of greater uniformity, displaying the optimum densities necessary for accurate interpretation. This view of exposure latitude is supported by Gratt, Sickles[37] in their xeroradiographic findings.

Grondahl [38], in reference to high kVp data, suggests that an increase in film density leads to an increased willingness to report positive findings. This observation will be discussed later.

. mAs - Milliamperage Seconds

This is a measure of the electric current passing through the tube, or the quantity of radiation produced, and is controlled by the temperature of the filament and the exposure time. As the kVp is increased, the mAs must be decreased to counter the increased density. There is no precise method of doing this though.

Film latitude is closely related to the exposure time. A film that has a wide latitude can record an object having a wide range of inherent subject contrast. Therefore, the wider the latitude, the greater the range of object densities that may be visualised. Typically, a wide latitude has a relatively low contrast. If we decrease the exposure time, the resultant image will be lighter and have slightly wider latitude.

In this experiment it was assumed that the majority of dental x-ray machines in use ran at 10mA and consequently there was no comparison made at a different mA. However, the optimum exposure time results can be altered for a change in milliamperage simply by incorporating those values into the following equation.

$$mA_e\ S_e = mA_1\ S_1$$

Where e = experimental values.

. Source/skin distance

Obviously, with the incorporation of beam aiming devices there can be no such item as a source/object distance. Only an approximation can be made. For intra oral radiographs the end of the aiming device is placed as close as possible to the skin on the face. For the purposes of this study it was assumed that there existed another 6 cm between the film and the end of the cone with the tooth under examination being either 1 or 2 cm from the film.

As the source/skin distance increases the image sharpness and resolution are enhanced by decreasing the penumbral blur. Similarly, by increasing the source/skin distance, we decrease divergence of photons in the beam and therefore decrease image size distortion (magnification). Thus, long open-ended aiming devices are best.

The effect of altering the source/skin distance on the diagnosis of dentinal caries and yet keeping variables such as the exposure time consistant with this change is one of the objectives of this experiment.

. Object/film distance

Decreasing object/film distance also increases image sharpness and resolution and decreases magnification by the same imaging principles outlined above.

Another endeavour of this experiment is to glean any differences in interpretation of dentinal caries if we adjust the object/film distance from 1 cm to 2 cm.

11. Experimental Methodology

The presurvey trials were designed to give an indication of the optimal values for detected caries of dentine in different tooth types. This was achieved very effectively as the results of the survey proper bore out. They were also designed to enable a selection of teeth which had questionable caries depth.

The elimination of optimal exposures of greater than 1.0 secs. meant the elimination of kVp's less than 60 in all instances. It is assumed that an exposure time of 1.0 seconds or greater is just not realistic in practice, due to the excessive scattered radiation exposure and the chance of patient movement during that time.

There was some concern that the enlistment of only 18 teeth in the survey proper may well make the experiment manageable, but not valid. Certainly, this was the case when estimating the film quality in the canine/incisor category and questionable in assessing proximal lesions in molar teeth due to the small sample used.

The institution of the paralleling technique is well advised in any instance for either clinically or experimentally detecting carious lesions. It employs the most accurate imaging principles, avoids film displacement and bending, provides unmatched reproducibility, and avoids clinical overlapping [39].

The soft tissue simulation employed by Hedin, Halse [15] was applied to this experiment by using a 1 cm. diameter viscose tube filled with water. It is believed that soft tissue around the mouth is 90% water and that it causes considerable scatter and fogging. The viscose tubing was placed between the tooth and the x-ray source with the greatest diameter of the tube placed at a level coinciding with the proximal and occlusal lesions under examination. Indeed, on all those light films without very penetrative photons, the scatter produced by the water can be seen with the unaided eye, as a diffuse radiopaque cylinder overlying the tooth.

The penetrometer measures the quality of the x-rays and provides a comparison in radiographic contrast and density between films. Its use was primarily as a control measure, but was extremely effective in assessing inter and intra machine reliability. In this respect, it was significant that the General Electric machine produced less dense radiographic patterns of the step wedge than the Sieman's model. This can be explained by the age of the General Electric model, in comparison with the Sieman's unit which had just been installed and it is conceivable that its functional capacity had decreased over the years. It is unestimable how significant this lack of penetrative x-rays is with respect to the kilovoltage/ exposure time recommendations which this survey has concluded.

However, the General Electric machine was very reliable in densitometric readings from one exposure to the next (intramachine reliability) which is important in view of the multiple film exposures required by this experiment.

The automatic processing of films was of questionable reliability, as previously discussed, but an attempt was made to process as many films at a time and at a stage when the processing solutions were not exhausted chemically.

Experimental viewing conditions were designed to coincide with the optimal situation an average practitioner would have available at his disposal. These have been previously described and decrease markedly extraneous stimuli and improve image enhancement.

According to Goaz & White [40], there are four aspects of visual acuity (1) detection of single lines or grey level variation, (2) pattern recognition, (3) resolution of fine image detail, (4) recognition of a 3D object in a 2D image. All are important. However, item (1) has specific involvement in this experiment..

Jensen [41] examined differences in visual acuity and image perception. He found the eye could perceive up to 20 line pairs per mm. However, with the use of a magnifying lens, the diagnostic yield can be increased by 135%. He states that 'image perception also depends on the illumination and contrast of the image. It is not known whether the decreased contrast of high kVp will decrease the performance of the human visual system in the perception of small structures'. It is known that high kVp exposure techniques increase the image latitude to show more density grades in each radiograph. If the results of this experiment show an inclination towards better resolution of dentinal caries at low contrast, higher kVp, is it not possible that the human visual system <u>can</u> perceive such small variations in density? Or are these results just an extension of Grondahl's findings [37] that with an increase in film density there is an increased

willingness to report positive lesion findings? Unfortunately, there is no clear cut answer to such diverse observations.

Visual pattern bias is not important in this study because the observers' eyes need to be solely attuned for caries detection and nothing else and the examination was conducted in an ordered sequence, i.e. mesial, occlusal, distal.

Concerned with visual reliability is the role the Mach effect plays in the determination of caries at the DEJ. One cannot say with any certainty whether it has no effect at all on the perceived radiolucent decaying front or whether it becomes more apparent as we decrease the contrast by increasing kVp and exposure time. It may well have a profound effect on the experimental results.

Every attempt was made to obtain optimal examiner reliability by reducing any bias. This included the use of 2 observers who underwent a rigorous pre-survey calibration by randomising all films, by eliminating the examiners from literature related to the topic and to the clinical evaluation of the survey teeth, by preventing any assessment of the results until all viewing had ceased, and by making an effort only to assess films only when the observers were alert and fresh.

However, despite the pre-survey calibration, interexaminer reliability is questionable (see Table 7), wherein observer performance varied considerably in categories where a deal of ambiguity existed. In those categories which were not, examiner performance was consistant, e.g. evaluation of proximal lesions of dentine in canine/incisor teeth.

It was found, however, that because there were only 18 different teeth in the survey and 33 films had to be viewed of each, that after a while, one could discern what tooth was being viewed. This no doubt resulted in some biases, despite a conscious effort not to do so.

Another problem occurred with the arbitrary values designated to the 30 cm source/skin distance exposure times. Because of the limited exposure time scale available to the experimenter in these machines, the optimal exposure time calculation when altering the source/skin distance may well have had to be adjusted by $\pm$ 0.1 secs.

111. Evaluation of Diagnostic Film Quality in the Interpretation of Occlusal and Proximal Lesions in Dentine

Diagnostic Value

The results indicated, that for a constant current of 10mA, the most diagnostically valueable films occurred at the following kilovoltage and exposure times.

Molars 70 kVp at 0.8, 1.0 secs.
80 kVp at 0.6, 0.8, 1.0 secs.
90 kVp at 0.4 secs.

Premolars 70 kVp at 0.8, 1.0 secs.
80 kVp at 0.5, 0.6 secs.
90 kVp at 0.5, 0.6 secs.

Canines/Incisors
60 kVp at 1.0 secs.
70 kVp at 0.8, 1.0 secs.
80 kVp at 0.6 secs.
90 kVp at 0.4 secs.

An initial assessment was made between the 1 and 2 cm focal/object distances and it was found that these two values were comparable in each tooth group and thus were grouped together for statistical purposes. There was no significant difference in film quality between the 20 and 30 cm source/skin distances in any category.

As discussed previously all exposure times greater than 1.0 secs were omitted.

It was noticeable that at lower kVp's, i.e. 60, 70, all tooth categories exhibited an increase in this subjective quality evaluation as the exposure time was increased. This trend was followed at the 30 cm source/skin distance. To a lesser degree, in the analysis of dentinal caries this lower kVp trend was reestablished for an increasing exposure time. The extent of the lesion increased with increasing exposure times in the 60, 70 kVp range.

Significantly, most poor radiographs occurred at the ends of the experimental spectrum, i.e. 60 kVp at 0.6s, 90 kVp at 0.6 s., and at the ($\bar{x}$) exposure times.

Occlusal Caries

In the analysis of occlusal caries there was no significant differences between 20 and 30 cm source/skin distances in the molar category. However, slightly fewer lesions were picked up at the 30 cm distance in the premolar group.

Importantly, a greater number of lesions extending to the D.E.J. were picked up at the 90 kVp, 80 kVp, and high exposure 70 kVp ranges, in molar teeth. In premolar teeth, best results were achieved at the optimal exposure times, except for 70 kVp at 1.0 secs. This was consistant with the molar category where the optimal exposure time (x) was the most prolific, for each kilovoltage.

Many lesions were assessed as having reached the D.E.J. in the premolar category, that had clinical evidence of only subsurface demineralisation, and some lesions apparent clinically as a sticky fissure did not reach the D.E.J. radiographically. Whether these results are false positives is debateable. Conversely, it could just be excellent radiographic diagnosis. Nevertheless, it is apparent that whether there exists a sticky fissure or just subsurface demineralisation, these clinical signs are not a good guide to the depth of the lesion.

The existance of these radiographic/clinical disparities brings us to another issue. These reversals did not necessarily increase with an increasing kVp or exposure time as Grondahl would have expected, with 'false positives'.

Any attempt to study a pathological process by a radiographic technique presents the problem of validity, where false positives and false negatives are always possible. [42]

In no instance, however, was a lesion extending into the D.E.J. reported in the control tooth.

Proximal Caries

The results indicated that for a constant current of 10mA, the diagnosis of carious lesions was best achieved by the following values, assuming we omit exposure times >1.0 secs.

Molars	70 kVp at 1.0 secs.
	80 kVp at 0.6, 0.8 secs.
	90 kVp at 0.5 secs.
Premolars	70 kVp at 0.8, 1.0 secs.
	80 kVp at 0.6 secs.
	90 kVp at 0.5, 0.6 secs.
Canines/Incisors	70 kVp at 0.8, 1.0 secs.
	80 kVp at 0.4 , 0.5, 0.6 secs.
	90 kVp at 0.3 , 0.35, 0.4 secs.

In many instances in the molar and premolar teeth categories, caries were found radiographically to extend to the D.E.J. in those teeth which did not show clinical evidence of cavitation. It is possible that these lesions were clinically apparent as subsurface demineralisation.

In the molar category, the best diagnostic results were found at the optimal exposure time (x). However in the premolar category, most lesions were detected at the higher exposure times (x^{+}) in each kVp. This trend does significantly approximate Grondahl's observations that low contrast, high kVp dense radiographs may be associated with an increased willingness

to report positive findings. However, the human visual system may well have the capacity to detect such small changes in density associated with such films.

In the canine/incisor dategory in which teeth have a smaller buccal-lingual width and x-ray penetration can be more effective, there was little variation in the results. The only variability occurred in the lower range 60 kVp values. In no instance did the 'control' teeth report lesions extending to the D.E.J., which confirms examiner reliability between the 'test' and 'control' groups.

Significantly, there was no difference in results reported at the 30 cm source/skin distance when compared with the 20 cm source/skin distance, in any tooth category.

By comparing the results of visual grading the lesion depth and the diagnostic value of the film recommendations for the most diagnostically accurate values for determining early dentinal caries and the depth of more advanced lesions, can now be documented. The following recommendations are effective if the following values are kept within these limits : 10mA, 1-2 cm focal/object distance, 20 cm source/skin distance.

a) <u>Molars</u>	70 kVp at 1.0 secs.
	80 kVp at 0.6-0.8 secs.
	90 kVp at 0.4-0.5 secs.
b) <u>Premolars</u>	70 kVp at 1.0 secs.
	80 kVp at 0.5-0.6 secs.
	90 kVp at 0.5 secs.
c) <u>Canines/Incisors</u>	60 kVp at 1.0 secs.
	70 kVp at 0.8-1.0 secs.
	80 kVp at 0.5-0.6 secs.
	90 kVp at 0.3-0.4 secs.

These results are probably considerably higher than those values used in most clinical situations, but it is genuinely believed that low contrast and high density films have a greater capacity for determining caries of the dentine. An effort must be made to enhance these more diagnostic radiographs by the implementation of optimal viewing conditions.

CONCLUSIONS:

Using the parallel periapical radiographic technique in which the source to film distance was approximately 30 cm. and tooth to film distance was 2 cm. , optimal factors for visualization of dental caries within dentine were as follows:

Molars	70 kVp at 1.0 secs.
	80 kVp at 0.6-0.8 secs.
	90 kVp at 0.4-0.5 secs.
Premolars	70 kVp at 1.0 secs.
	80 kVp at 0.5-0.6 secs.
	90 KVp at 0.5 secs.
Canines/Incisors	60 kVp at 1.0 secs.
	70 kVp at 0.8-1.0 secs.
	80 kVp at 0.5-0.6 secs.
	90 kVp at 0.3-0.4 secs.

It is worthy to note that kVp ratings below 70 were not generally acceptable, especially due to long exposure times.

As might be expected, increased kVp to 80 and 90 kVp respectively, reduced the optimal exposure times and eliminated the scattering effect of the tissue representation (water) which reduces the diagnostic quality of the films.

In general kVp ratings above 70 kVp are necessary to outline the carious lesion in dentine, due to a lower contrast.

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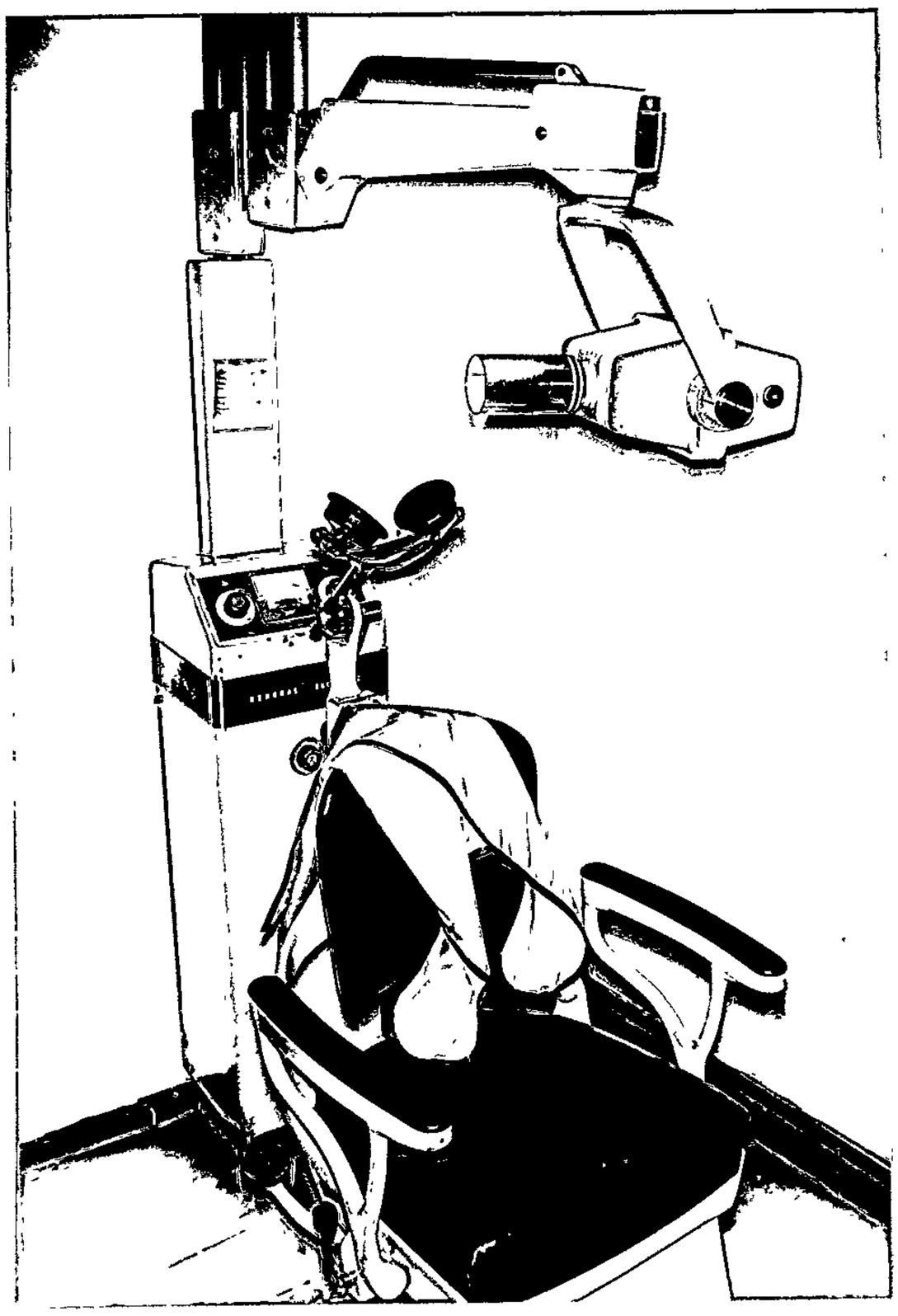
PHOTOGRAPH "A"



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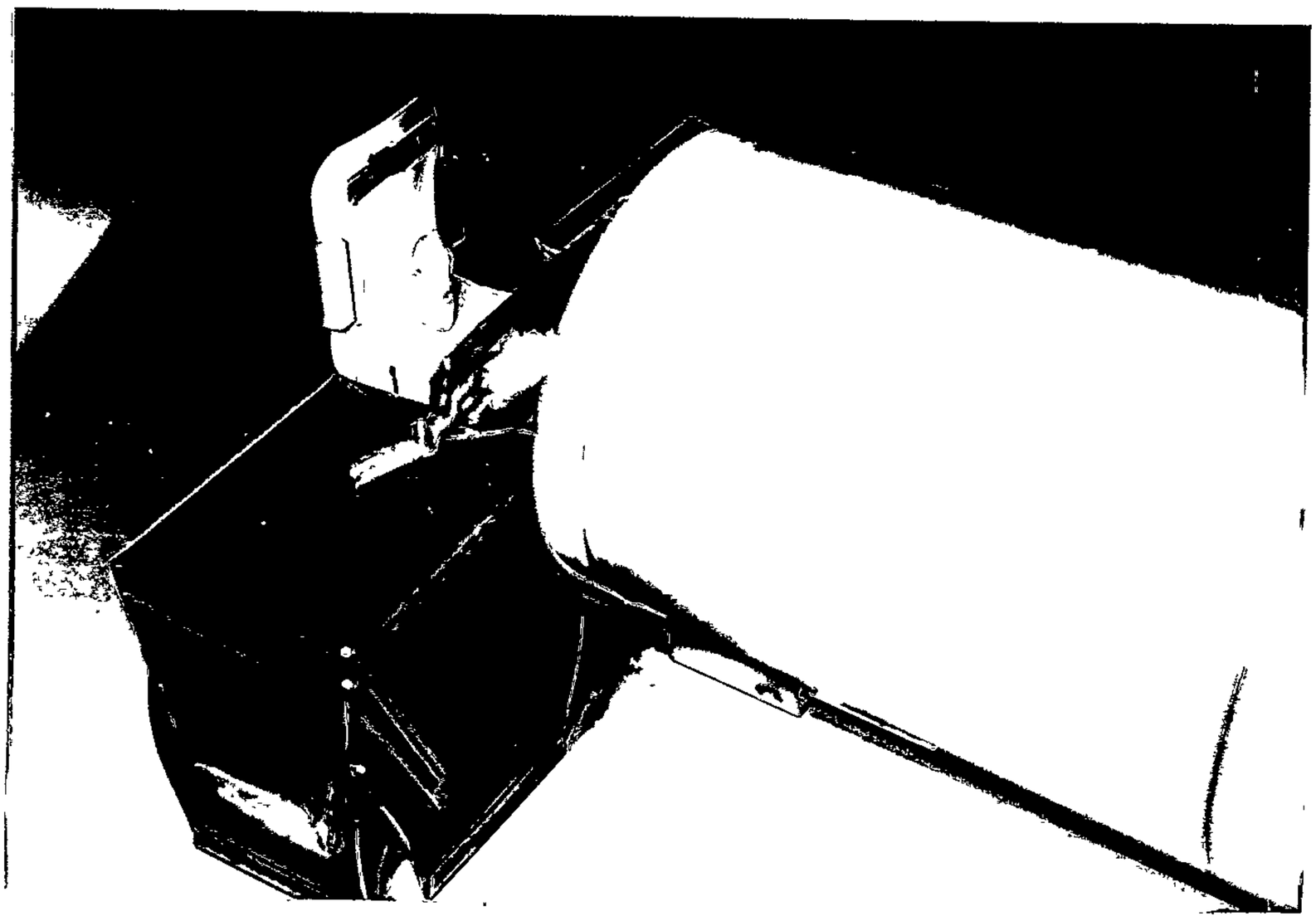

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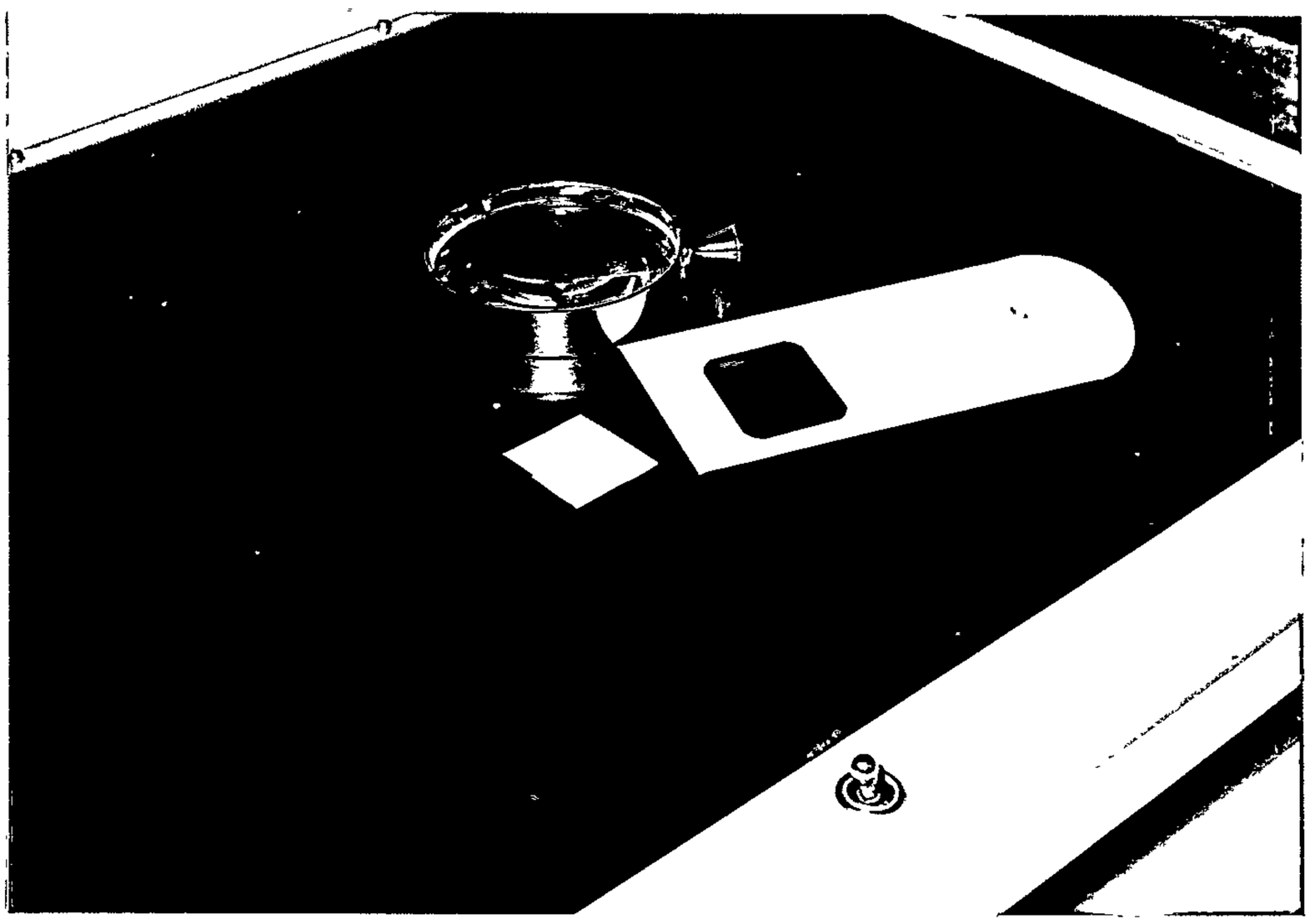

APPENDIX "C"

RADIOGRAPHIC ANALYSIS OF

OCCLUSAL CARIES IN DENTINE

1.

MOLARS

On clinical examination:
6 showed evidence of occlusal caries
1 was a control.

Radiographic Scoring System:

0	No evidence
1	Lesion limited to enamel
2	lesion extending to D.E.J.
3.	Lesion extending <½ into dentine
4	Lesion extending >½ into dentine

Observer 1 (B.L.) - Small numerals
Observer 2 (C.T.) - LARGE NUMERALS

a) 1 cm film/object, 20 cm focal/skin
* There were no false positive scores in the control tooth.

4												1
3	3 1	4 2	4 4	3 2	3 3	4 3	4 2	4 2	4 3	4 3	4 2	3 4
2	1 2	1 3	1	1 1	1 2	1 1	2 3	2 3	2	1 1	2 3	1 1
1	1	1	1	1	2	1				1		
0	2 4	1 2	1 3	2 4	1 2	1 3	1 2	1 2	1 4	1 3	1 2	2 2
Exposure Time (sec.)	1.0	1.25	1.5	0.6	0.8	1.0	0.6	0.8	1.0	0.4	0.5	0.6
	60 kVp			70 kVp			80 kVp			90 kVp		

CEJ

b) 2 cm film/object, 20 cm focal/skin
* There were no false positives in the Control Tooth

4												
3	4 5	4 4	4 3	4 5	4 3	5 2	4 2	4 5	4 4	4 2	4 3	4 2
2	1	1	2		1	1 2	2 2	1 1	1	1 2	2 2	1 2
1	1	1		1						1		1
0	2 1	1 3	1 4	2 2	2 4	1 3	1 3	2 1	2 3	1 3	1 2	1 3
Exposure Time (s)	1.0	1.25	1.5	0.6	0.8	1.0	0.6	0.8	1.0	0.4	0.5	0.6
	60 kVp			70 kVp			80 kVp			90 kVp		

c) 1 cm film/object, 30 cm. focal/skin

* There were no false positives in the Control Tooth

4								
3		3 3	4 2	4 2		4 4	5 5	4 3
2		1 1	1 3	1 3				2
1		1		1		1		1
0		2 3	2 2	1 2		2 3	2 2	2 2
Exposure Time (s)		1.25	1.5	1.75		0.8	1.0	1.25
		70 kVp				90 kVp		

11. Premolars

On clinical examination:

3 showed evidence of occlusal caries.

1 was a control

3 showed no evidence.

Radiographic Scoring System:

0 No evidence

1 Lesion limited to enamel

2 Lesion extending to D.E.J.

3 Lesion extending $<\frac{1}{2}$ into dentine

4 Lesion extending $>\frac{1}{2}$ into dentine

Observer 1 (B.L.) - Small numerals

Observer 2 (C.T.) - LARGE NUMERALS

a) 1 cm film/object, 20 cm focal/skin

* There were no false positives in the Control Tooth
* Bracketted numbers denote positive readings in those teeth which showed no clinical evidence of caries.

4																		
3	(1) 3			(1) 2	1	(2) 4	(1) 1	(1) 2	(1) 2	(1) 3	1	(1) 3	1	(1) 3	(1) 2	(1) 3		(1) 2	1
2		(1) 1		(1) 1			(1) 2	1		(1) 1	(1) 1		(1) 1				(1) 2		
1																			
0	4	6		4	6	3	4	4	5	3	5	4	5	4	5	4	5	5	6
Exposure Time(s)	0.6			0.8		1.0		0.4		0.5		0.6		0.4		0.5		0.6	
	70 kVp							80 kVp						90 kVp					

b) 2 cm. film/object, 20 cm focal/skin

* There were no false positives in control tooth
* Bracketted numbers denote positive readings in those teeth which showed no clinical evidence of caries.

4									
3	2 (1) 2	3 (1) 1	(1) 4	(1) 3	(1) 3 (1) 2	2 (1) 1	2 (1) 1	2 (1) 2	1 (1) 1
2	1	(1) 1	(1) 1	(1) 1	(1) 1		1	(2) 2	1
1									(1) 1
0	5 4	4 5	3 6	4 6	4 4	5 6	5 5	3 5	4 6
Exp. Time(s)	0.6	0.8	1.0	0.4	0.5	0.6	0.4	0.5	0.6
	70 kVp			80 kVp			90 kVp		

c) 1 cm film /object, 30 cm focal/skin

* There were no false positives in Control tooth
* Bracketted numbers denote positive readings in those teeth which showed no clinical evidence of caries.

4							
3	1 (1) 1	1 (1) 2	3 (1)		3 (1)	2 (1) 1	2
2	1	1	(1) 1		(1) 1		(1) 1 (1) 1
1							
0	5 6	5 5	4 6		4 6	5 6	4 6
Exp. Time(s)	1.25	1.5	1.75		0.8	1.0	1.25
	70 kVp				90 kVp		

lll. Incisors/Canines

Obviously, occlusal caries is very unlikely in these teeth unless pits are present. As to be expected, not one positive reading was evaluated by either observer at any altered variable, i.e. kVp, exposure time, F/S and F/O distance..

Appendix D.

RADIOGRAPHIC ANALYSIS OF

PROXIMAL CARIES IN DENTINE

1 Molars

Of the 7 molar teeth (i.e. 14 proximal surfaces), the following clinical findings were found:

2 control surfaces

4 showing no clinical evidence

8 (6 showing subsurface demineralisation
8 (2 showing cavitation

Radiographic Scoring System:

0 No evidence

1 Lesion limited to enamel

2 Lesion extending to D.E.J.

3 Lesion extending $<\frac{1}{2}$ into dentine

4 Lesion extending $>\frac{1}{2}$ into dentine

Observer 1 (B.L.) - Small numerals

Observer 2 (C.T.) - LARGE NUMERALS

a) 1 cm film/object, 20 cm focal/skin

* No false positives in the Control Teeth.

4												
3	1 2	2 2	1 1	1 2	1	3 1	2 2	3 1	2 1	1 1	1	1 2
2		1	1		1 2	2		2	1 1	2 1	1 3	2
1	6 2	4 3	6 2	4 1	4 1	5 1	5 4	4 2	3 2	3 1	3 2	4 1
0	7 10	7 9	7 10	9 11	8 11	6 10	7 8	7 9	8 10	8 11	9 9	9 9
Exp. Time (s)	1.0	1.25	.5	0.6	0.8	1.0	0.6	0.8	1.0	0.4	0.5	0.6
	60 kVp			70 kVp			80 kVp			90 kVp		

b) 2 cm film/object, 20 cm focal/skin

* No false positives in Control Teeth

4																	
3	1	1	1		1	1	1	1	1	2	3	1	3	1	1	3	1	2	1		2	1	1	2
2	1	1	1	3	1	2	1	1	1	1		2	1	2	2		1			2		2		1
1	4	2	5	2	5	3	5	2	3	1	1	2	2	3	1	3	1	2	4	3	2	3	4	2
0	8	10	7	9	7	8	7	10	9	10	10	9	8	8	10	8	11	10	9	9	10	8	9	9
Exp. Time(s)	1.0		1.25		1.5		0.6		0.8		1.0		0.6		0.8		1.0		0.4		0.5		0.6	
	60 kVp						70 kVp						80 kVp						90 kVp					

c) 1 cm film/object, 30 cm focal/skin

* No false positives in Control Teeth

4																	
3			1		1	1	2	2					3	1	2	2	2	2
2			1	2		2	1							2		1		
1			5	3	5	2	3	3					5	2	5	2	6	2
0			7	9	8	9	8	9					6	9	7	9	6	10
Exp. Time(s)			1.25		1.5		1.75						0.8		1.0		1.25	
			70 kVp										90 kVp					

11. Premolars

Of the 7 premolar teeth (14 proximal surfaces), the following clinical evidence was found:

2 control surfaces
2 surface with no evidence of decay
5 surfaces showing subsurface demineralisation
5 surfaces showing cavitation

Radiographic Scoring System:

0 No evidence
1 Lesion limited to enamel
2 Lesion extending to D.E.J.
3 Lesion extending $\leq$½ into dentine
4 Lesion extending $>$½ into dentine

Observer 1 (B.L.) - Small numerals
Observer 2 (C.T.) - LARGE NUMERALS

a) 1 cm film/object, 20 cm focal/skin

*Bracketted numbers are those false positives in the control tooth surfaces.

4						1			1
3	3 2	4 2	5 2	5 3	4 3	3 1	4 2	4 2	5 1
2	1 1	2 3	1 3	1 1	1	2 3	1 1	2	1 2
1	2 1	2 2	2 (1) 3	2 2	(1) 4 1	3 1	3 4	2 4	(1) 3 (1) 3
0	8 10	6 7	6 6	6 8	6 9	6 8	6 7	6 8	5 7
Exp. Time (s)	0.6	0.8	1.0	0.4	0.5	0.6	0.4	0.5	0.6
	70 kVp			80 kVp			90 kVp		

b) 2 cm. film/object, 20 cm focal/skin

*Bracketted numbers denote those false positives in control tooth surfaces.

4			1		1			1	1					1		1	
3	4	2	4	2	4	2	4	2	4	1	6	3	2	2	5	2	6	2
2	1	1	1	2	1	3	1	1		2		1	3	1	1	2	1	3
1	3		2	3	(2) 3	2	3	3	3	2	2	2	3	2	2	3	2	1
0	6	11	6	7	5	7	6	8	6	8	6	8	6	9	6	6	5	7
Exp. Time(s)	0.6		0.8		1.0		0.4		0.5		0.6		0.4		0.5		0.6	
	70 kVp						80 kVp						90 kVp					

c) 1 cm. film/object, 30 cm focal/skin

*Bracketted numbers denote those false positives in control tooth surfaces.

4									1			
3	3	2	3	2	5	3		5	2	6	2	5	5
2	3	1	3	2	1				2		4		2
1	2	3	2	2	2	3		2	3	(1) 3	1	2	
0	6	8	6	8	6	8		7	7	5	7	6	7
Exp. Time(s)	1.25		1.5		1.75			0.8		1.0		1.25	
	70 kVp							90 kVp					

111. Canines/Incisors

Of the 4 teeth in this category (8 proximal surfaces), the following clinical observations were made:

2 control surfaces
1 showing no evidence of decay
1 showing subsurface demineralisation
4 showing cavitation

Radiographic Scoring System:

0 No evidence
1 Lesion limited to enamel
2 Lesion extending to D.E.J.
3 Lesion extending $< \frac{1}{2}$ into dentine
4 Lesion extending $> \frac{1}{2}$ into dentine

Observer 1 (B.L.) - Small numerals
Observer 2 (C.T.) - LARGE NUMERALS

a) 1 cm film/object, 20 cm focal/skin

*Bracketted numbers denote false positives in control tooth surfaces.

4	1	1 1	2 1	1 1	3 1	1 1	1 2	1 2	1 1	1 1	1 2	1 1
3	3 3	3 2	3 2	2 2	1 2	3 3	3 1	3 1	3 2	3 2	3 1	3 3
2	1 1	1	1	1	1		1	1	1	1	1	
1	(1) :2		(1) 1	1	2 1	2	1 2		1	1 1	1 1	2
0	1 4	4 4	2 4	4 4	2 3	2 4	3 2	4 4	3 4	3 3	3 3	2 4
Exp. Time(s)	0.6	0.8	1.0	0.6	0.8	1.0	0.4	0.5	0.6	0.3	0.35	0.4
	60 kVp			70 kVp			80 kVp			90 kVp		

b) 2 cm film/object, 20 cm focal/skin

*Bracketted numbers denote false positives in control tooth surfaces.

4	1 1	1	1 1	2	3 3	2 2	2 1	1 1	2 3	1 2	1 1	2 3
3	3 2	2 3	3 2	2 4	1 1	2 1	2 2	3 3	2	2 1	3 2	2
2	1		1			1			1	1 1	1	1
1		3 1	2 2	(1) 2 1	(1) 1	2 2	1		(1) 3			2 1
0	4 4	2 4	2 2	2 3	3 4	2 2	4 4	4 4	1 4	4 4	4 4	2 3
Exp. Time (s)	0.6	0.8	1.0	0.6	0.8	1.0	0.4	0.5	0.6	0.3	0.35	0.4
	60 kVp			70 kVp			0 kVp			90 kVp		

c) 1 cm film/object , 30 cm focal/skin

* Bracketted numbers denote false positives in control tooth surfaces.

4		3 2	1 2	2		2 2	2 2	1 1
3		1 1	3 1	2 3		2 2	2 1	3 2
2		1	1	1			1	1
1		(1) (1) 3 1		(1) 3 2		(1) 2		
0		1 3	4 4	1 2		2 4	4 4	4 4
Exp. Time (s)		1.25	1.5	1.75		0.5	0.6	0.8
		70 kVp				90 kVp		

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