

SYNOVIAL MEMBRANE AND SYNOVIAL FLUID.

As was stated before the distribution of synovial membrane indicates the articular surface and adjacent non articular surface of a joint. (22) Three types of synovial membrane are usually recognised - an areolar type, an adipose type and a fibrous type. The areolar type is usually restricted to non articular surfaces. The adipose type is said to fill joint spaces during movement and the fibrous type lines the articular surfaces of the joint. The articular surfaces of the joint, the pars gracilis menisci, the pars posterior menisci, the articular surface of the condyle and the articular slopes of the articular fossa (39) are lined by flattened fibroblasts or chondrocytes spaced widely apart embedded in ground substance in which there are collagenous fibres. The adjacent non articular parts have synovial membrane of the areolar type and here the surface cells are 1 to 4 layers thick. The sub synovial connective tissue is loose in structure and contains blood vessels previously described. This type of synovial membrane is seen beneath the pes menisci the inferior stratum, also superior to the pes menisci and superior stratum, where it is reflected onto the periphery of the condyle and onto the boundaries of the articular fossa. At the reflection, a small amount of synovial membrane of the adipose type may be seen.

Synovial fluid consists principally of hyaluronic acid protein complex which contains very little chondroitin sulphuric acid and a dialysate which closely approximates blood plasma. (40, 41, 42) If this is so, then during joint movements a ready supply of blood should be available and this could be achieved by mechanisms such as arterio-venous-anastomoses occurring in parts of the temporomandibular joint not usually subject to compression during reflex masticatory shift and which also has a prominent layer of synovial membrane. (39, 43)

The probable elaboration of the synovial mucin has been reviewed by Leber and Ford (42) and their investigation suggests that it is the surface cells and the immediate sub adjacent cells that are responsible for the production of synovial mucin. The superficial cells are P.A.S. positive and show under electron microscopy a discontinuous plasma membrane which supports the belief that these cells are undergoing degenerative changes. They cannot be distinguished from fibroblasts and they have opaque granules similar to those observed in osteoblasts and fibroblasts. The deeper cells in the sub synovial connective tissue are fibroblasts and occasionally mast cells. The mast cells are believed by some authorities to produce the anti-coagulant heparin, which could be the precursor of hyaluronic acid. (45) The viscous polysaccharide of synovial fluid has been isolated as hyaluronic acid (40) but its origin is not known. It has been suggested that the fibroblast secretes this viscous polysaccharide. (42) It has also been suggested that mast cells secrete this substance. (45) However the evidence is far from conclusive.

The evidence regarding the origin of synovial mucin is as follows:-

A strong P.A.S. reaction is given by the cytoplasm of the cells lining the synovial membrane by the inter-cellular matrix between them. Davies (44) found similar layers which stained with mucicarmine. A thin film overlying the surface cells gives a faint positive reaction with alcian blue. Above pH 4 concentration, the nuclei of the synovial cells take up methylene blue, but the cytoplasm and the intercellular substance are relatively unstained. Below pH 4 there is no staining except of some granular cells which are probably mast cells. After staining with 1-1000 thionine, some cells are seen below the synovial surface which contain meta chromatic granules, and are thought to be mast cells. (44, 45)

It is notable that the P.A.S. reaction is not affected by hyaluronidase. It has been shown by Davies (44) however that the positive P.A.S. reaction of synovial fluid and even of some hyaluronic acids is unaffected by hyaluronidase. It is thought, therefore, the positive P.A.S. material concentrated in relation to the cells lining the synovial membrane is probably synovial mucin.

It has been noted by Griffin and Sharpe (9) that at the terminal part of the epithelioid cell type arterio-venous-anastomoses near the synovial surface there is an accretion of ground substance in their immediate vicinity, which is very suggestive of secretory function. It seems to indicate that at least some of the cells associated with the epithelioid cell types of arterio-venous-anastomoses has the property of secreting mucopolysaccharides intercellularly and peripherally into the lumens of the associated blood vessels. If such is the case, they could be responsible for the mucopolysaccharide content of synovial fluid. (43)

An interesting fact about hyaluronic acid is that it is a substance of high molecular weight and is not really diffusible through the synovial membrane. However solution and substances of low molecular weight readily pass through the synovial membrane in either direction, particularly through the capillary bed. On the other hand substances of high molecular weight are removed by the blood and lymph capillaries. In acute inflammation the rate of exchange across the synovial membrane increases and in chronic inflammation it seems variable. (41) The synovial membrane seems to subservise the following functions:- lubricatory; nutritional; protective; and maintenance of a constant fluid and chemical medium.

Mac Conati (47) has stated that there is a type of circulation of synovial fluid within the joint and corresponding with its movements, and this is the reason why moderate exercise of the joint is helpful during rehabilitation. On the other hand Griffin (43) suggests that movement of the

joint during rehabilitation facilitates the rate of exchange of substances across the synovial barrier and this is achieved by shutting down of the arterio-venous-anastomoses. Key (22) points out the cell content of the synovial fluid tends to increase after death. Different investigators have found cell counts of 80 to several thousand per cubic millimeter. A typical differential count by Key indicated 58% monocytes, 15% macrophages, 14% ill-defined types of phagocytes, 1% primitive cells, 3% synovial cells and 5% of other types of blood leucocytes. It would appear that the cell content of synovial fluid varies considerably from joint to joint and from species to species. As was stated there is very little synovial membrane of the adipose type lining the joint cavities and this is perhaps due to the fact that the spaces which would be created by movements of the condyle, are filled by neuro-vascular adipose tissue of the bilaminar zone. In thin faced persons, when they masticate a hollow can be seen in front of the tragus of the ear which indicates a medio-anterior movement of tissue posterior to the condyle during mastication.

Meniscus and age changes.

The human temporomandibular meniscus may be regarded as the persistent organised part of the embryonic meniscus which united during foetal life to the mandibular condyle and the temporal bone. The joint compartments are visible at about 57 m.m. C.R. stage of foetal life. (48) Moffett (49) states that the medial portion of the temporomandibular meniscus is derived from the posterior extension of the external pterygoid muscle which he refers to as a tendon and notes that Garden and Gray (50) described a similar occurrence in the human shoulder joint in which the tendon of the long end of the biceps brachii muscle develops in situ, in the extra blastemal tissue of the gleno-humeral joint. He found in a 75 m.m. C.R. foetus an inferior joint cavity and a superior joint cavity being formed.

Harpman and Woollard (51) confirmed Kjellberg's (52) findings that some of the fibre bundles of the external

pterygoid muscle pass into the meniscus of the temporo-mandibular joint and continue posteriorly between the squamous temporal and tympanic bones to be attached to the head of the malleus. Moffett (49) maintains that the meniscus is attached to the malleus until foetal development of the joint is complete. Symons (48) states that the connection of the meniscus to the malleus persists to at least the 180 m.m. C.R. stage. This may be the reason why part of the superior stratum passes through the squamo-tympanic fissure and why also medially the superior stratum is associated with the petro-tympanic fissure rather than the squamo-tympanic fissure.

The function of the menisci is somewhat obscure and the following suggestions have been made:-

(1) They compensate for the incongruity of the surfaces between which they are interposed;

(2) They serve the purpose of resilient buffer minimising the shock of impacts;

(3) They are related to the type of movement occurring at the joint.

(4) That menisci bring about the formation of wedge-shaped films of synovial fluid in relation to the weight-transmitting parts of the joints during movement in accordance with what might be expected on the basis of physical theories of lubrication. (4) If this is true they should exist in joints where the articular surfaces have large radii of curvature. (47)

Menisci are capable of regeneration. Following the removal of a meniscus a new one usually forms growing in from the fibrous capsule of the joint. (53)

As mentioned before the central part of the meniscus is composed of fibro-cartilage whilst the peripheral portions are composed of fibrous tissue. The central portion of the meniscus at birth is very cellular and the intercellular substance is proportionally less. With age the intercellular substance increases in amount so that the structure is proportionally less cellular. It should be noted here that

elastic tissue distribution in the meniscus may increase with age. (30)

Fibrillation of the meniscus was also observed. This phenomenon is known to occur in articular cartilage and fibrocartilage. (38) It is essentially an unmasking of the collagenous fibres due to a dissolution or change in the ground substance. Associated with this change hypertrophic and degenerative chondrocytes were observed. It was suggested that swelling of the cytoplasm of the hypertrophic chondrocytes was associated with the dissolution of ground substance. (21) It is known that this phenomenon occurs in young individuals also, and when it happens, it is associated with malfunction of the joint and it occurs at the peripheral edges of the articular cartilage. (38) The peripheral edges of the articular cartilage are known as transition zones and here the articular cartilage is continuous with the fibrocartilage, which in turn becomes continuous with the synovial membrane of the joint. At these sites, in the case of the temporomandibular joint, a fold or fringe of synovial tissue may be seen to over lie the perichondrium for a short distance. This projection of synovial membrane of the arcolar type is continuous with the fibrous tissue of the perichondrium, and it underlies, in the case of the inferior joint cavity, and overlies, in the case of the superior joint cavity. If this region is exposed to stress the cartilage cells beneath the perichondrium may proliferate and be replaced by bone, so that a deformity of the head of the condyle results. Usually bone is laid down on the anterior articular slope of the condyle so that it becomes wedge shaped. This shape is also associated by a flattening of the anterior slope of the articular fossa and gross wear of the articular eminence.

It should be noted that Vaughan (54) observed changes in the condyle associated with age, the most common change being the wedge shaped anterior extension described above. He related it to maximum pressure being concentrated on the anterior surface of the condyle during mastication.

Griffin and Sharpe (55) have observed this condylar shape in roentgenograms of elderly people associated with a marked flattening of the articular eminence. An intermediate form would appear to exist in which the anterior articular slope is flattened associated with a moderate flattening of the articular eminence. It seems fair to infer that the bony changes are due to mastication in a forward or ventro position and that osseous activity occurs at the transitional zone where perichondrium of the condyle is continuous with the synovial membrane of the antero-inferior joint cavity, and it may be initially due to proliferation of fibro cartilage with subsequent calcification, whereby the wedge shape of the condyle results. It seems as if it is a resultant of mastication in ventro position and the bony changes occur slowly over a long period of time so that it is usually only observed in elderly people and may be diagnosed as an osteoarthritis of the temporomandibular joint due to mastication in ventro position.



FIG 243. A.



FIG. 243 B.

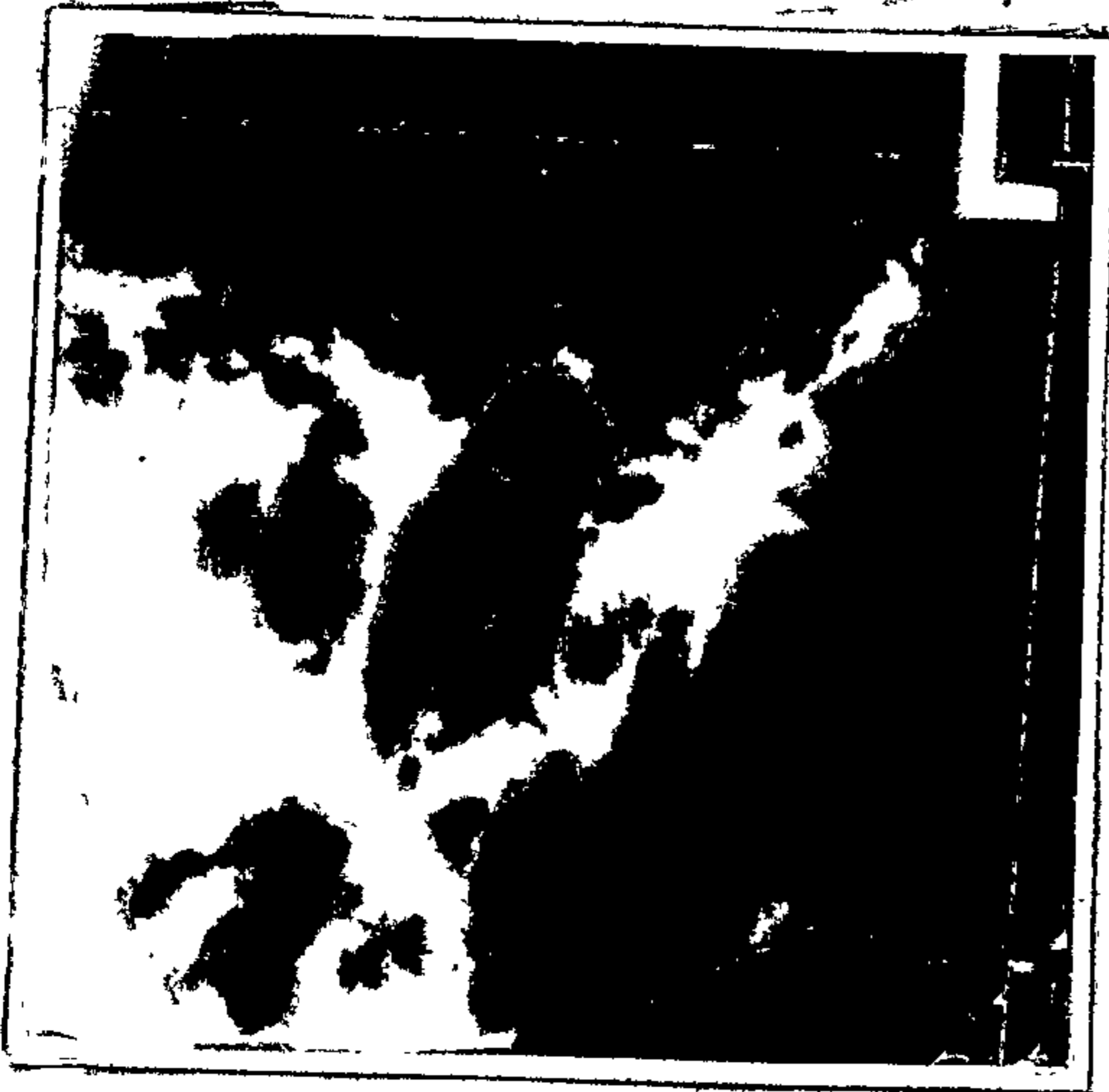


FIG. 243. C.



FIG. 243 D

Fig. 243.

Roentgenograms temporomandibular joint showing deformity of condyle with advancing age. Sagittal aspect.

243 A. Showing the normal appearance of a condyle and the normal temporo condylar relationship when the jaws are in normal or classical centric occlusion.

243 B. Showing an intermediate form of deformity of the condyle with the jaws open. Note: ~~Destruction~~ of articular eminence and flattening of the anterior articular slope of the head of the condyle.

243 C. Showing a more severe form of deformity of the condyle than in fig. 243 B. with jaws open. Note: Destruction of articular eminence.

243 D. Showing a very severe form of deformity of the condyle with the jaws open. Note: Erosion of articular eminence.

Probably it can be inferred that a change of this nature in the head of the condyle is associated with mastication in ventro position. (8)

Whilst deformity of the mandibular condyle can result from subluxation also fibrillation can occur in the meniscus, and the writer has observed this phenomenon in the pars gracilis menisci. (21) The specimen in this case was from a 72 year old cadaver. Here undoubtedly the change was associated with age, although there was thrombosis of an articular branch of the pterygoid artery.

The dissolution of intercellular substance is a reverse process of the compensatory mechanism by which fibre-cartilage is alleged to compensate for wear. So that inevitably, thinness of the meniscus results and decrease in radiolucent areas occupied by the meniscus in roentgenograms of these people is undoubtedly due to fibrillation caused by pressure over a number of years. This indeed supports the contention that the temporomandibular joint is a stress bearing joint and not non-stress bearing as alleged by Robinson. (55)

With wear of the meniscus, the soft tissue areas related to the meniscus, that is, the bilaminar zone and the pterygo-condylar area must eventually be involved in articulation.

Symons (20) states that in those muscles with tendinous attachments such as the lateral pterygoid muscles, the suprahyoid muscles, the attachment of the tendon fibres to the bone must continually undergo a process of breaking down and restoration. Although this may be compensated in part by the inferior re-attachment of the lateral pterygoid muscle.

Symons (20) further states that during growth of the mandibular condyle there is a constant breaking down and restoration of the tendons of the lateral pterygoid muscle and a similar phenomenon could conceivably compensate to certain extent a retro-position, ventro-position and superior position of the condyle associated with age and malfunction.

The writer et al (6, 7, 8, 21) has noted pathological changes in the blood vessels in these areas and agrees that

pressure plays a part in the pathogenesis of these conditions. The most susceptible blood vessels are the anterior tympanic artery and its articular branches. This artery is relatively fixed since it passes through the squamo-tympanic fissure on its way to supply the tympanic membrane. In the retro position of the condyle, usually associated with class II malocclusion or loss of teeth the condyle approximates the squamo-tympanic fissure and these blood vessels must inevitably be compressed. This compression may play a part in the pathogenesis of atherosclerosis since this condition was observed in arteries in this region of the bilaminar zone in the menisci obtained from cadavers aged 28, 58 and 73 years. Similarly in ventro-position of the condyle, masticatory compression of blood vessels would occur in the anterior extremity of the joint and in the pterygo-condylar area. In one specimen thrombosis of a muscular artery was observed. (21) Some authorities (57, 58, 59, 60, 61, 62, 63, 64, 65.) state that compression at least of some degree is concerned in the aetiology of atherosclerosis. The occurrence of this condition in these strategically placed blood vessels, since they are relatively fixed, supports this contention. However the abundance of nerve fibres in this region leads to the consideration of possible effects if blood vessels were compressed. It is probable the effects are concerned with proprioception, deep sensibility, vaso-constriction and the possibility of neuro-vascular reflexes originating from this source.

Innervation.

When the blood vessels become involved local and reflex pain may occur. In this respect it is important to note the innervation of the blood vessels involved. It is known that (66) arteries, capillaries and veins are innervated to a variable degree, the most profuse innervation is of arteries with well developed muscular coats and vasa vasorum, whilst thick walled veins have a less profuse innervation. It is stated that

cutaneous arteries have a richer innervation than others. (67) King (68) noted very fine nerve fibres around capillaries which ended in Rouget cells. Kramer and Toda (69) stated that the larger vessels within the body cavities receive their innervation from the sympathetic trunks, prevertebral plexuses and cerebrospinal nerves. The exact mode of termination of these vascular fibres is controversial, some claiming it is a terminal reticulum, whilst others maintain that free loop and bouton endings occur in or on muscle fibres. Also corpuscular and other endings of a sensory type may be found in relationship to the adventitia.

The majority of the nerve fibres are said to be unmyelinated although larger myelinated fibres may be present. It is thought that sympathetic fibres usually produce vasoconstriction and para sympathetic fibres vaso-dilation and furthermore they maintain nutrition and integrity of the parts supplied. (66) Kellegren and Samuel (70) proved that the fibrous articular capsule and synovial membrane have a sympathetic and somatic innervation and Rossi (71) has revealed nerve fibres in the articular cartilage.

The reflex pain associated with temporomandibular joint dysfunction is probably of sympathetic origin, the afferent pathway being the cephalic extension of the upper four thoracic spinal nerves. The evidence for this is after total or subtotal resection of the sensory root of the trigeminal nerve for the relief of trigeminal neuralgia clinical observations led Fraser (72) to believe that fibres extending from the cervical sympathetic into the head played a role in certain sensory phenomenon in the area of distribution of the fifth cranial or trigeminal nerve. Helson (73) examined Fraser's patients and found different forms of sensibility in the area of distribution of the 5th nerve following sectioning of its sensory root. According to his findings, sensibility to light, touch and ordinary painful stimuli is lost, and sensibility to deep pressure and the ability to localise touch are greatly reduced. After the

operation, but later they are gradually restored to an appreciable degree. Temperature stimuli between 15° C. and 45° C. evoke no sensations, but hot stimuli (60° C. - 75° C.) give rise to stinging or pricking sensations. It is significant that if a cervical sympathectomy is done at the same time as resection of the sensory root of the 5th nerve hot stimuli evoke no response. The evidence was unequivocal, that in order to obtain the absolute zero of cutaneous sensibility in the area of the 5th nerve that cervical sympathectomy has to be done as well as the sensory root resection of the 5th nerve.

Fay (74) advanced the opinion that afferent components both of the upper thoracic and vagus nerves extend in the cephalic region along the carotid arteries. Kuntz (75, 76) and Christensen (77) produced experimental proof of afferent fibres of the four upper thoracic spinal nerves extending along the arteries extending into the cephalic region. They felt that these fibres were not primary pain conducting fibres because they were of a large calibre. Electrical stimulation of the nerve plexuses of the carotid arteries did not elicit pain reaction but resulted in reflex responses in the lower cervical and upper thoracic segments and particularly in the forelimb. Clinical evidence (78, 79, 80, 81, 82.) as well as experimental evidence (83, 84) supports the contention that the initial effect of stimulation of these efferent nerve fibres is vaso constriction and that other clinical phenomenon are secondary to this primary vaso constriction. The efferent pathway would seem to involve the hypothalamo-reticulo spinal tract. (85)

Experiments have shown that stimulation of certain areas of the bulbar reticular formation (86, 87) elicits the patellar reflex and inhibits the jaw reflex; and, conversely, when the jaw reflex is elicited, it is not possible to produce the patellar reflex. It is also known that sustained compression of the femoral artery will cause renal ischaemia, and, in certain cases, necrosis of the renal cortex. This

will not occur if either the sciatic or lower-splanchnic nerves are sectioned prior to compression of the femoral artery. (38) Almost nothing is known as regards the effects of sustained cephalic vascular compression. It is probable, except by tumor or infection, that the only blood vessels in the cephalic regions which are susceptible to sustained compression are the blood vessels of the temporomandibular articulation.

The evidence clinical, experimental and morphological indicates that the reflex symptoms associated with temporomandibular dysfunction are due to intermittent compression of neuro-vascular connective tissue relative to the articular surfaces of the joint, and that the basis of these symptoms is neuro-vascular reflexes, which in the afferent side enter the spinal cord via the dorsal root ganglia of thoracic 1 to thoracic 4 and on the efferent side is disposed by the ganglionated sympathetic trunk undoubtedly higher centers being also involved. It is probably initially of a vascular spinal reflex nature. With this reflex pain limitation of movement of other joints and muscular rigidity have been noted. (8) It is probable that neuro-vascular reflexes are the basis of these conditions.

Specialised Blood Vessels.

In this respect specialised type of blood vessels have been noted in the bilaminar zone notably the genu vasculosis menisci, (9) sub synovial connective tissue and pterygo-condylar area. These vessels are of epithelioid type arterio-venous-anastomoses. Similar arterio-venous-anastomoses have been described in joints. For example:-

(1) Epithelioid cells at the terminal portion of cushioned arteries terminating in veins of synovial membrane, and the human pulvinar acetabulum has been described by Muratori. (89)

(2) Arterio-venous-anastomoses in the interphalangeal joints of the big toe have been shown by the injection method by Lang (90)

(3) Arterio-venous-anastomoses in the human knee joint have been described by Luna (91)

(4) Arterio-venous-anastomoses have been demonstrated in the human temporomandibular joint by Griffin and Sharpe (9,92.)

Glomus bodies have been observed by the writer in various parts of the human temporomandibular meniscus. A neuro myo-arterial glomus in the human temporomandibular meniscus in the vicinity of the squamo-tympanic fissure has been described by Griffin. (92) In that instance it consisted of a cushioned artery, a Sucquet-Hoyer canal, a receptaculum, a periglomerular nerve, arterioles and capillaries.

The literature on blood vessels which have been described by Clara (93, 94) Clark (95) Boyd (96) as arterio-venous-anastomoses has been reviewed. The sites where arterio-venous-anastomoses of characteristic morphology have been found are; the external ear of the rabbit; the calf and man; the human finger and toe; the bird's foot; the coccygeal body of man and the caudal glomeruli of various animals; and the tongue of the dog, sheep and goat. (97, 98) A monograph on their distribution has been written by Clara. (99) Their existence in the mucous membrane of the gastro-intestinal tract, the thyroid gland, the carotid body and the sympathetic ganglia of man has been reported by Le Gros Clark. (1)

The innervation of arterio-venous-anastomoses has been described by Brown (100) as follows: "It seems reasonable to conclude that the elaborate arborizations of thick fibres are receptor mechanisms primarily concerned with vascular reflexes in which the activity of the arterio-venous-anastomoses is involved. On the other hand, the much thinner probably unmyelinated fibres may be concerned with vaso-constriction since they end on the surface of the modified media of the Sucquet-Hoyer canal."

Arterio-venous-anastomoses possess the following properties:-

(1) They exhibit spontaneous rhythmic change in calibre (101,102)

(2) They react to temperature changes. (101, 102, 103.)

(3) They keep exposed parts warm by allowing a greater amount of arterial blood to flow through the part. (104)

(4) They constrict in response to adrenalin and faradic stimulation of the sympathetic nerves and dilate in response to acetylcholine and histamine. (101, 105.)

Epithelioid type arterio-venous-anastomoses.

The origin of epithelioid cells is controversial. Some investigators (106, 107, 108, 109, 99.) state that the epithelioid cells and smooth muscle cells originate from the same type of mesenchymal cell, others (110, 111) state that they differentiate from a mesenchymal syncytium and one recognisable in 27 to 30 m.m. fetuses. In regard to the pathogenesis of the glomus tumors King (112) stated it is important to realise that the development of any of these cells is dependent on the particular stimuli which are acting on the tissues and is not an inherent function of the cells themselves. According to Griffin and Sharpe's (9) observation, epithelioid cells are very common in the human temporomandibular meniscus at 19 months of age, but are rare at term. However it may not be necessary to assume that glomus cells arise de novo and glomus tumors described in the knee joints, (113, 114, 115, 116) wrist joints, (113, 116) and bone of the foot (117) arise from proliferating pre-existing epithelioid cells.

Popoff (118) described a rich innervation of the blood vessels constituting the glomus body and referred to the external coat of the Sucquet-Hoyer canal as a neuro-collagenous reticulum. According to Masson (119) the innervation of glomus bodies subserves vasomotion, he states that the vascular dilations perceived by the extra vascular sensory fibres can be the origin of a vasomotor reflex. The extreme dilation perceived by the nerve termination inside the vessel's walls can be the origin of an inhibitory reflex. Thus one can understand the alternate rythmical systoles and diastoles of the glomus. One can also think that the

pressures perceived by the tactile dermal corpuscles can be also the origin of contractile reflexes inhibiting the glomus. Popoff (118) states that the neuro muscular structure of the glomus indicates that it has two functions: local and general. It would seem therefore, they have a local mechanical function and also a secretory function. (105) Moreover the mechanical function may be secondary to the secretory function, since, when the cell is swollen it obstructs the lumen of the blood vessel and when empty i.e. after secretion the lumen becomes patent. It is difficult to decide whether the secretion is intercellular or intravascular but the concentration of ground substance in their immediate vicinity indicates the possibility of intercellular secretion of muco-polysaccharides. If such is the case and this has already been discussed under synovial fluid, they could be responsible for the muco-polysaccharide content of the synovial fluid. (9, 46) The local mechanical function would also regulate the blood flow to the synovial membrane and thus play a role in the maintenance of blood pressure by effecting a reasonable constant peripheral resistance. Furthermore, since they have the ability to greatly increase the blood flow through the part, it is possible that they have yet another function of temperature control near the surface of the joint.

It is well known that the epithelioid cell type arterio-venous-anastomoses is well innervated (100) both by myelinated and amyelinated nerve fibres and it has been suggested (100) that neurovascular reflexes are mediated via the myelinated nerve fibres associated with epithelioid cell type arterio-venous-anastomoses. The possibility of glomus tumor arising in the temporomandibular meniscus has to be considered. It seems that certain types of a typical facial neuralgia could be due to a glomus tumor in the temporomandibular meniscus. One has to think also of the exceedingly diverse symptoms associated with glomus tumors and the frequent occurrence of a diagnosis of neuroesthesia

when actually a glomus tumor in the body was responsible for the symptoms. ^[112] These symptoms are explicable on the basis of neurovascular reflexes, which may cause intractable and, at times, paroxysmal and excruciating local pain. Referred pain elsewhere in the body can be caused by glomus tumors. For instance, it has been observed that referred pain from a subungual glomus tumor (120) was relieved when blood was squeezed out from the tumor which indicated that pressure is an important factor in initiating reflex symptoms. Therefore the existence of a glomus tumor in the temporomandibular meniscus could be diagnosed by depression of the mandibular condyle with subsequent relief of symptoms. (92) The probable pathway of these neuro-vascular reflexes and pain mechanisms have been described. (75, 76, 77, 78)

Epithelioid cell could not be observed in the meniscus of 58 years but were readily observed in the menisci from the 19 months and 28 year specimens. Apart from this Griffin (92) noted a glomus body in the temporomandibular meniscus of a male cadaver aged 58 years. It has been suggested by some authorities (118) that epithelioid cells disappear with advancing age, and this may be the case as far as our limited observations to date.

Popoff (118) remarked that the disappearance of these cells might be associated with a decrease of thermal regulatory ability of the aged, and if this is the case, it could be inferred that disappearance of these cells in the joints could impair their function.

Proprioception.

Joints, tendons and the muscles associated with them have a sensory proprioception innervation. Proprioception may be defined as muscle sensibility and controls muscle contraction in association with the function and pressure required. Receptors concerned with proprioception have been found in muscle tendons, periosteum and synovial membranes associated with joints. These nerve endings may be of the free ending type, or of the encapsulated type, these latter

are pacinian corpuscles, neuromuscular spindles and the Golgi tendon organs.

Sherrington (121) found at least 40% of the fibres innervating a given muscle subserved the sensory rather than the motor end organs.

Fulton (122) states " The evidence points to the muscle spindles as being the stretch receptors and the findings of Cooper et al (123) on spindles of eye and jaw supports this contention. "

Granit (124) in his Silliman Lectures for 1954 summed up the evidence indicating that muscle spindles are the stretch afferents.

Also Granit and Kasda (125) have shown that stimulation of the facilitatory part of the reticular formation gives rise to an increase in the rate of afferent discharge from single muscle spindles. Similar stimulation of the inhibitory area of the ^{reticular.} reticulater formation may cause inhibition of spindle discharge.

In this respect, the writer has referred to inhibition of stretch reflexes associated with the jaws when the knee jerk is elicited, and, similarly, when the jaw jerk is elicited, the knee jerk is inhibited.

Again in this respect it is interesting to note, that the Golgi tendon organs inhibit contractile responses evoked by muscle spindles. (122) McCouch et al (126) found that when tension developed in a tendon of dangerous magnitude, active contraction is automatically inhibited. This is the splinting phenomenon of Hagoun and Rhines. (127) Murphy (128) states basically jaw movements are activated by a muscle mass in relation to the temporomandibular joint. At rest motor units throughout this mass are contracting (activated) asynchronously to maintain a state of tone. Every nuance of movement from this position has its own pattern of contracted (excited) and inhibited motor units throughout the muscles of mastication; and, for that matter, throughout the hyoid musculature and the neck muscles which fix the head as well.

The evidence for jaw opening and jaw closing reflexes has been found in the decerebrate animal. (121, 129, 130, 131)

The jaw opening reflex may be evoked by blunt pressure on the gum bordering the crown of a tooth and on the tooth crown. It is more easily elicited in the bicuspid region, but most difficult to elicit in the region of the cuspids. It also may be elicited by faradism in the anterior part of the hard palate. The reflex is unilateral. The diaphragm muscle contracts in opening of the mouth and there is reflex central inhibition of the elevator muscles (temporalis, masseter and medial pterygoid) when the jaw opens.

Sherrington (132) says the reflex therefore strikingly exhibits reciprocal innervation. The reflex also exhibits the phenomenon of rebound. (133, 134, 135) That is to say it is a diphasic reflex, one phase of the reflex inducing the other phase. In the cerebral cortex there is a large representation of the jaw opening reflex. (131)

The first order cytons subserving proprioceptive sensibility for the masticatory muscles are located in the mesencephalic nucleus of the 5th trigeminal nerve. It is also probable that proprioceptors concerned with eye, musculature are located in this nucleus. (136, 137, 138) It is probable that proprioceptive nerve endings are located in the periodontal membrane since chromatolysis was found in this nucleus in the cat after re-section of the maxillary nerve ventral to the sphenopalatine ganglion. (139)

In the frog the cytons of the mesencephalic nucleus are small and insignificant in the larval stage and only mature when the animal possesses its masticatory apparatus. (140) It would seem that maturation of the cells in this case is functional. Evidence presented by Corbin and Gardiner (141, 142) and Brody (143) indicated that 52% of myelinated nerve fibres in spinal nerves disappear from the third to the end of the ninth decade. This suggests that there is a progressive loss of muscle sensibility with age. It is probable that this is the reason why oral rehabilitation is more difficult in the

old person and also the possibility of a temporomandibular joint dysfunction is more likely to occur with loss of proprioception. The purpose of proprioception is to regulate muscle contraction with voluntary effort. For instance, Mitchell (66) suggests that there is no reason why all the nerve elements concerned with proprioception should not be regarded as autonomic and points out there is much evidence indicating that sympathetic fibres, some of which end in close relationship to striated muscle, play some part in non-volitional activity. He also points out that the autonomic afferent fibres which carry interoceptive impulses from viscera and blood vessels are only a variety of proprioception.

There seems no doubt that vasomotion plays a large part in muscle efficiency. Apart from the controversial experiments of Hunter (144) Kuntz and Kerper (145) Coates and Tiege, (146.) Van Dijk (147, 148) indicated that sympathetic nerves are directly concerned with plastic tonus. It seems fairly certain that the brake phenomenon of Rieger (149) is associated with the sympathetic nervous system since it disappears after sectioning of sympathetic innervation. (150, 151) Again the evidence is conflicting as regards the role, the sympathetic nervous system plays in sustained muscle activity, (152, 153, 154, 155, 156, 157) Although it seems fairly certain that sympathetic stimulation enhances the restitution of fatigue muscle. The basis of this finding may be that arterio-venous-anastomoses are associated with the metabolism of skeletal muscle and sympathetic stimulation would constrict these arterio-venous-anastomoses and thus increase their blood supply and thereby presumably facilitate the removal of metabolites. (43. 92) Thus it would seem that the effect of sympathetic nerves on muscle function would be indirect and not direct. Also that muscle sensibility is to a large extent dependent on an adequate blood supply. The reflex musculature symptoms observed in certain cases of temporomandibular joint dysfunction therefore seems explicable on the basis of neurovascular reflexes interfering with their

metabolic requirements and it may be also that the facilitatory inhibiting properties of the bulbar reticular formation may have to do with the indirect effect of vaso motor pathways rather than direct effect via motor efferent nerves.

BIBLIOGRAPHY.

1. Le Gros Clark, W.E. The Tissues of the Body: Oxford, Clarendon Press, 9th. Ed., 1958.
2. Salzman, J. A. Principles of Orthodontics: Philadelphia. J.P. Lippincott Co., 2nd Ed., 1950.
3. House, E.L. & Pansky, B. A Functional Approach to Neuroanatomy: McGraw-Hill Book Co., New York, 1960.
4. Rees, L.A. Structure and function of the mandibular joint: Brit. D. J., 96: 125-133, 1954.
5. Orban, B. Oral histology and Embryology: St. Louis, The C.V. Mosby Co., 2nd. Ed., 1949.
6. Griffin, C.J. & Barnett, A.V. Plastic thrombophlebitis of the pterygocondylar area. Oral Surg., Oral Med. & Oral Path., 11 : 12, 1323-1336, 1958.
7. Griffin, C.J. & Barnett, A.V. Atherosclerosis in the temporomandibular joint and its clinical and pathological significance. Aust. D.J. 3 : 5, 293 - 297, 1958.
8. Griffin, C.J. & Sharpe, C.J. The structure of the adult human temporomandibular meniscus. Aust. D.J., 5 : 4, 190-195, 1960.
9. Griffin, C.J. & Sharpe, C.J. The Genu Vasculosis Menisci of the human temporomandibular meniscus. Aust. D.J. (in press.)
10. Sicher, H. Oral anatomy: 2nd. Ed., C.V. Mosby Co., St. Louis, 1952.
1. Steinhardt, G. Die Beanspruchung der Gelenkflächen bei verschiedenen: Bissarten. Dtsch. Zahnheilk. Vortr. 1 : 91, 9. 1934.
2. Langer. Das Kiefergelenk des Menschen: Sitzungsbericht K. Akad. Math.naturw. Klasse, Bd. 39. (quot from Lubosch).
3. Kjellberg, K. Bidrag till kakledens utvecklingshistoria: Diss., Stockholm, 1901.
4. Lubosch, W. Über Variationen am Tuberculum articulare des Kiefergelenkes des Menschen und ihre morphologische Bedeutung: Gegenbaurs Morphol. Jahrb. 35 : 1 - 2, 322, 1906.
- Fabian, H. Studien zur Kaufunktion. Ein Beitrag zur Frage nach der Ursache der Spee'schen Kurve und des Tuberculum articulare: Dtsch. Zahnheilk. Vortr. 65 : 6, 1925.
- Humphreys, H. Age changes in the temporomandibular joint and their importance in orthodontics: Int. J. Orthod. 18 : 8, 809, 1932.
- Lindblom, G. On the anatomy and function of the temporomandibular joint: Acta Odont. Scand. 17 : supplement 28, 1960.
- Pritchard, J.J. A cytological and histochemical study of bone and cartilage formation in the rat: J. Anat. Lond., 86 : 3, 259 - 277, 1952.
- Hunter, W. Philos. Trans., 42 : 514, 1743.
- Symons, N.B. The attachment of the muscles of mastication: Brit. Dent. Journ. 96 : 76 - 81 1954.
- Griffin, C.J. & Sharpe, C. J. Pressure atrophy of the human temporomandibular meniscus associated with thrombosis of an articular branch of the pterygoid arteries: Aust. Dent. J. (in press.)

22. Key, J.A. The synovial membrane of joints and bursae: In Special Cytology, Ed. by E.V. Cowdry, Paul B. Hoeber, Inc., New York, 1928.
23. Perry, H.T. & Harris, S.C. Role of the neuromuscular system in functional activity of the mandible: J. Amer. Dent. Assoc., 48 : 665 - 673, 1954.
24. Last, R.J. The muscles of the mandible: Proc. Roy. Soc. Med., 47 : 571 - 578, 1954.
25. Pleasure, M.A. Prosthetic occlusion, a problem in mechanics: J. Amer. Dent. Assoc., 24 : 1303 - 1318, 1937.
26. Angel, J.L. Factors in temporomandibular joint form: Amer. J. Anst., 83 : 2, 223 - 246, 1948.
27. Tristram, G.R. The proteins: New York, Academic Press Inc. Ed. Neurath and Bailey, 1, part A, 181, 1953.
28. Bowes, J.H. & Kenton, R.H. Biochem. F. 45, 281, 1949.
29. Neuberger, A. Fibrous proteins and their biological significance: Symposia of the Society for Exp. Biol. No. IX, Cambridge. At the University Press, 73, 1955.
30. Griffin, C.J. & Sharpe, C.J. Distribution of elastic tissue in the human temporomandibular meniscus especially in respect to "compression" areas: Aust. Dent. J. (in press.)
31. Berry, D.C. The movement of the meniscus of the temporomandibular joint: Preliminary Communication, The Lancet, 82 - 83, 1952.
32. Kellegren, J.H. Collagen diseases: S.E.B. Symposium IX. The Fibrous Proteins. Cambridge. At the University Press, 164, 1955.
33. Lansing, A.L. Ageing of elastic fibres: J. Nat. Cancer. Inst. 12 : 217, 1951. - Chemical Morphology of elastic fibres in connective tissues: New York Ed. C. Ragan, Macy, 45, 1951.
34. Ireland, V.E. The problem of the clicking jaw: Proc. Roy. Soc. Med., 44 : 363, 1951.
35. Elliott, H.C. Studies on articular cartilages: 1. Growth mechanisms: Amer. J. Anat. 58 : 127, 1936.
36. Bywaters, E.G.L. The metabolism of joint tissues: J. Path. and Bact. 44 : 247, 1937.
37. Rosenthal, O., Bowie, M.A. & Wagoner, G. Studies in the metabolism of articular cartilage: 1. Respiration and glycolysis of cartilage in relation to its age: J. Cell. and Comp. Physiol. 17: 221, 1941.
38. Ham, A.W. Histology: Philad. J.B. Lippincott Co., 1950.
39. Griffin, C.J. & Sharpe, C.J. The distribution of the synovial membrane and mechanism of its blood supply in the adult human temporomandibular joint: Aust. Dent. J. (in press)
40. Meyer, K., Smyth, E.M. & Dawson, M.H. Isolation of mucopolysaccharide from synovial fluid: J. Biol. Chem., 128 : 319 - 327, 1939.

41. Bauer, W., Bennett, G.A., Marble, A., & Claflin, D. Observations on normal synovial fluid of cattle: cellular constituents and nitrogen content. *J. Exper. Med.*, 52 : 835 - 848, 1930.
42. Lever, J.D. & Ford, E.H.R. Histological, Histochemical and electron microscopic observation on the synovial membrane: *Anat. Rec.*, 123 : 4 525 - 539, 1958.
43. Griffin, G.J. The mechanism of the blood supply to synovial membrane: *Aust. Dent. J.*, 4 : 6, 379 - 384, 1959.
44. Davies, D.V. Synovial membrane and synovial fluid of joints. *Lancet*, ii : 815 - 819, 1946.
45. Osboe-Hansen, G. The mast cell: In *Inter. Rev. Cytol.*, Ed. iii New York, Acad. Press, 1954.
46. Griffin, G.J. & Sharpe, G.J. Concerning the origin of synovial mucin: (in preparation.)
47. Mac Conail, M.A. The movements of bones and joints: 3. The synovial fluid and its assistants: *J. Bone & Joint Surg.*, 32 - B: 244 - 252, 1950.
48. Symons, N.B.B. The development of the human mandibular joint: *J. Anat. Lond.*, 86 : 3, 326 - 332, 1952.
49. Moffett, B.C. The prenatal development of the human temporomandibular joint: *Contributions to Embryology*, vol. 36., Carnegie. Inst. Wash. Pub. 611. (242 - 251), 1957.
50. Gardener, E. & Gray, D.J. Prenatal development of the human shoulder and acromioclavicular joints: *Amer. J. Anat.*, Vol. 92, 219 - 276, 1953.
51. Harpman, J.A. & Woollard, H.H. The tendon of the lateral pterygoid muscle: *Journ. Anat.*, vol. 73, 112 - 115, 1938.
52. Kjellberg, K. Beitrage zur Entwicklungsgeschichte des Kiefergelenks: *Gegenbaurs Morphol. Jahrb.* vol. 32, 159 - 186, 1904.
53. Smillie, I.S. Injuries of the knee: Edinburgh, Livingstone, 1946.
54. Vaughan, H.C. A study of the temporomandibular articulation: *Journ. Amer. Dent. Assoc.* 30 : 19, 1943.
55. Griffin, G.J. & Sharpe, G.J. The mechanism of osteoarthritis in the human temporomandibular joint. (in preparation.)
56. Robinson, M. Temporomandibular joint : theory of reflex controlled non lever action of mandible: *J.A.D.A.*, 33 : 1260 - 1271, 1946.
57. Duguid, J.B. Thrombosis as a factor in the pathogenesis of coronary atherosclerosis: *J. Path. Bact.*, 58 : 207 - 212, 1946.
58. Duguid, J.B. Thrombosis as a factor in the pathogenesis of aortic atherosclerosis: *J. Path. Bact.*, 60 : 57 - 61, 1948.
59. Duguid, J.B. Diet and coronary disease: *Lancet*, i : 891, 1954.

60. Harrison, C.V. Experimental pulmonary arteriosclerosis: J. Path. Bact., 60 : 289 - 293, 1948.
61. Geiringer, E. Intimal vascularization and atherosclerosis: J. Path. Bact., 63 : 201 - 211, 1951.
62. Crawford, T. & Levene, C.I. The incorporation of fibrin in the aortic intima: J. Path. Bact., 54 : 523 - 528, 1952.
63. Dunlop, G.R. & Santos, R. Adductor-canal thrombosis: New England J. Med., 256 : 577 - 580, 1957.
64. Palma, E.C. Arteriopatias estenosantes del miembro inferior Síndrome del canal de Hunter Y anillo del tercer adductor: Bol. Y. Trab. Acad. argent cir., 34 : 771 - 787, 1950.
65. Hall, G.V. A clinician's view of the pathogenesis of atherosclerosis: M.J. Aust., 26 : 1053 - 1056, 1955.
66. Mitchell, G.A.G. Anatomy of the autonomic nervous system: E. & S. Livingstone. Edin. & Lond., 1953.
67. Le gros Clark, W.E. The tissues of the body: an introduction to the study of anatomy: 2nd. Ed. (reprint) Oxford Clarendon Press, 1946.
68. King, A.B. Nerve endings in the cardiac muscle of the rat: John Hopk. Hosp. Bull. 65 : 489 - 499, 1939.
69. Kramer, J.G. & Todd, T.W. The distribution of nerves to the arteries of the arm. With a discussion of the clinical value of results : Anat. Rec. 8 : 243 - 255, 1914.
70. Kellegren, J.H. & Samuel, E.P. The sensitivity and innervation of the articular capsule : J. Bone Jt. Surg., 32-B : 84 - 92, 1950.
1. Rossi, F. "Sur l'innervation fine de la capsule articulaire : Acta anat., 10 : 161 - 232, 1950.
2. Fraser, J. The autonomic nervous system in relation to surgery: Edin. Med. J., 38 : 189 - 214, 1931.
3. Helsen, H. The part played by the sympathetic system as an afferent mechanism in the region of the trigeminus: Brain, 55 : 114 - 121, 1932.
4. Fay, T. Atypical facial neuralgia, a syndrome of vascular pain: Ann. Otol. Rhinol. and Laryngol., 41 : 1030 - 1062, 1932.
5. Kuntz, A. Nerve fibres of spinal and vagus origin associated with the cephalic sympathetic nerves : Ann. Otol. Rhinol. and Laryngol., 43 : 50 - 67, 1934.
6. Kuntz, A. Pathways involved in pains of nasal and paranasal origin referred to the lower cervical and thoracic segments and the upper extremity: Ann. Otol. Rhinol. and Laryngol., 45 : 394 - 400, 1936.
7. Christensen, K. The innervation of the nasal mucosa with special reference to its afferent supply: Ann. Otol. Rhinol. and Laryngol., 43 : 1066 - 1084, 1934.

78. Kuntz, A. & Main, L.R. The neural basis of certain syndromes associated with dental lesions: Arch. Clin. Oral. Path., 4 : 333 - 344, 1940.
79. Haggett, E.W. Dental infection: a factor in disturbing the sympathetic-parasympathetic balance of the autonomic nervous system: Aust. D.J. 20 : 147 - 151, 1948.
80. Francis, E. Unrecognised sequelae of dental imbalance: Med. J. Aust. 4, 1955.
81. Griffin, C.J. Odonto-neuralgia-sympatheticus: Aust. D.J. 2 : 6, 339 - 352, 1957.
82. Griffin, C.J. Odonto-neuralgia-sympatheticus: Case report. Aust. D.J., 3 : 4, 262 - 264, 1958.
83. Pollock, L.F. & Davis, L. Visceral and referred pain: Arch. Neurol. Psychiat., 24 : 1041 - 1054, 1935.
84. Phillips, G. Recent observations on referred pain: Aust. & N.Z. J. Surg., 6 : 350 - 357, 1937.
85. Le Gros Clark, W.E., Beattie, J., Riddoch, G. and Dott, N.M. The hypothalamus: London, Oliver & Boyd, 1938.
86. Magoun, H.W. & Rhines, R. An inhibitory mechanism in the bulbar reticular formation: Anat. Rec., 91 : 289, 1945.
87. Magoun, H.W. & Rhines, R. An inhibitory mechanism in the bulbar reticular formation: J. Neurophysiol., 9 : 165 - 171, 1946.
88. Trueta, J., Barclay, A.E., Daniel, P.M., Franklin, K.J., & Prichard, M.J. Studies of the renal circulation: Oxford, Blackwell Scientific Publications, 1947.
89. Muratori, G. Anastomosi arterovenose e dispositivi vascolari di blocco nel pulvinar acetabuli dell'uomo: La Chirurgia 30 : 117, 1946.
90. Lang, J. Beitrag zur Gefaßversorgung der Gelenkinnenhaut Z. mikrosk: Anat. Torsch. 60 : 503, 1954.
91. Luna, G. Studies sulla vascolarizzazione della sinoviale: Quod. Anat. prat. 6 : 1, 1951.
92. Griffin, C.J. A neuro myo-arterial glomus in the temporomandibular meniscus: Aust. Med. J. (in press.)
93. Clara, M. "Die arterio-venösen Anastomosen der vogel und saugtiere" : Ergebn. Anat. Entw. Gesch. 27 : 246, 1927.
94. Clara, M. "Die arterio-venösen anastomosen": Barth. Leipzig, 1939.
95. Clark, E.R. Arterio-venous anastomoses : Physiol. Rev. 18 : 229, 1938.
96. Boyd, J.D. General survey of visceral vascular structures: Ciba Foundation Symposium on Visceral Circulation: Churchill, London, 1952.
97. Daniel, P.M. & Prichard, M.M.L. Arterio-venous anastomoses in the external ear : Quart. J. exp. Physiol. 41 : 107, 1956.
98. Prichard, M.M.L. & Daniel, P.M. Arterio-venous anastomoses in the tongue of the sheep and the goat: Amer. J. Anat., 95 : 203, 1934.

99. Clara, M. Die arterio-venösen Anastomosen : Springer Verlag, Wien., 1956.
100. Brown, M.E. The occurrence of arterio-venous anastomoses in the tongue of the dog: Anat. Rec., 69 : 287, 1937.
101. Grant, R.T. Observations on direct communications between arteries and veins in the rabbit's ear : Heart, 15 : 281, 1930.
102. Clark, E.R. & Clark, E.L. Observations on living arterio-venous anastomoses as seen in transparent chambers introduced into rabbit's ear: Amer. J. Anat., 54 : 229, 1934.
103. Grant, R.T., Bland, E.F. & Camp, P.D. Observations on the vessels and nerves of the rabbit's ear with special reference to the reaction to cold: Heart, 16 : 69, 1932.
104. Grant, R.T. & Bland, E.F. Observations on arterio-venous anastomoses in human skin and in the bird's foot with special reference to the reaction to cold: Heart, 15 : 385, 1931.
105. Tischendorf, F. Experimentelle Untersuchungen zur histobiologie der arterio-venösen Anastomosen: Z. mikr. Anat. Forsch., 43 : 153, 1938.
106. Schumacher, S. v. Über die Bedeutung der arterio-venösen Anastomosen und der epitheloiden Muskelzellen: (Quellzellen.) Z. mikrosk. Anat. Forsch., 43 : 107, 1938.
107. Krompecher, St. Histologische und entwicklungs geschichtliche Untersuchungen über das Glomus coccygium des Menschen: Anat. Anz. 75 : (Erg. Bd.) 176, 1932.
108. Mathis, J. Die Regulierung des arteriellen Blutstromes in der Nierenrinde: Wien klin. Wschr., 11 : 1444, 1934.
109. Gasparini, F. & Bucciante, G. Sulla morfogenesi delle anastomosi artero-venose delle dita dell'uomo: Atti Soc. Med. Chir. Padova, 28 : 198, 1950.
110. Von Rotter, W. Zur Orthologie und Pathologie der peripheren Regulations Systeme des Kreislaufes: Dtsch. Zahnärztl. Ztg., 5 : 813, 1950.
111. Von Rotter, W. & Wagner, L. Über die Entwicklung der subunguealen Glomera (sog. arterio-venöse Anastomosen) der Zehen. Arch. Kreisf. Forsch. 18 : 68, 1952.
112. King, E.S.J. Glomus tumor: Aust. & N.Z. J. of Surg. 23 : 280, 1955.
113. Butz, A. Über Erscheinungsformen des Glomus tumors: Chirurg. 12 : 97, 1940.
114. Hoffman, H.O.E. & Ghormley, R.K. Glomus tumor and intramuscular lipoma: Report of two cases. Proc. Mayo. Clin., 16 : 13, 1941.
115. Mackey, W.A. & Lendrum, A.C. Three cases of glomangioma or angioneuromyoma (painful subcutaneous tubercle) : Brit. J. Surg. 24 : 208, 1936.
116. Lewis, D. & Geschickter, C. Glomus tumors (arterial angioneuromyoma of Masson) : J. Amer. Med. Assoc. 105 : 775, 1935.

117. Bergstrand. Multiple glomic tumors. Amer. J. Cancer, 29 : 470, 1937.
118. Popoff, N.W. Recherches sur l'histologie des anastomoses arterio-veineuses des extremités et sur leur rôle en pathologie vasculaires: Bull. Histol. Appl., 12 : 156, 1935.
119. Masson, P. Le glomus neuro-myo-arteriel des régions tactiles et ses tumeurs: Lyon chir. 21 : 257, 1924.
120. Mason, M.L. & Weil, A. Tumor of a sub cutaneous glomus: Surg. Gynec. Obstet., Vol. 58 : 807, 1934.
121. Sherrington, C.S. J. Physiol., 17 : 211 - 258, 1894.
122. Fulton, J.F. A text book of Physiology: W.B. Saunders & Co., Philad. 17th. Ed., 215 - 216, 1955.
123. Cooper, S., Daniel, P.M. & Whitteridge, D. J. Physiol., 120 : 471 - 490, 1953.
124. Granit, R. Receptors and sensory perception : (Silliman Lectures, 1954.) New Haven, Yale University Press, 1954.
125. Granit, R. & Kaada, B.R. Acta physiol. Scand. 27 : 130 - 160, 1952.
126. McGouch, G.P., Deering, I.D., & Stewart, W.B. J. Neurophysiol., 13 : 343 - 350, 1950.
127. Magoun, H.W. & Rhines, R. Spasticity : The stretch reflex and extrapyramidal systems: Springfield, Ill., Charles P. Thomas, 1947.
128. Murphy, T. Control of the pressure strokes at the temporomandibular joint: Aust. D.J. 1 : 276 - 287, 1956.
129. Miller, F.R. & Sherrington, C.S. Some observations on the bucco-pharyngeal stage of reflex deglutition in the cat: Quart. J. Exp. Physiol., 9 : 147 - 186, 1915.
130. Sherrington, C.S. Integration action of the nervous system: New Haven and London. Yale Uni. Press, 16 : 411, 1906.
131. Sherrington, C.S. Flexion-reflex of the limb, crossed extension-reflex, and reflex stepping and standing: J. Physiol. 40 : 28 - 121, 1910
132. Sherrington, C.S. Selected writings of Sir Charles Sherrington Hamish Hamilton medical Books, London, 1939.
133. Sherrington, C.S. Strychnine and reflex inhibition of skeletal muscle : J. Physiol. 36 : 185 - 204, 1907.
134. Sherrington, C.S. On reciprocal innervation of antagonistic muscles: Eleventh note, Further observations on successive induction: Proc. Roy. Soc. 80 B : 53 - 71, 1908.
135. Forbes, A.A. Reflex inhibition of skeletal muscle; Quart. J. Exp. Physiol. 5 : 149 - 187, 1912.
136. Freeman, W. The columnar arrangement of the primary afferent centres in the brain stem of man: J. Nerv. & Ment. Disease, 65 : 378, 1927.
137. Woollard, H.H. The innervation of the ocular muscles: J. Anat. London. 65 : 215 - 223, 1931.
138. Sheinin, J. J. Studies on the mesencephalic nucleus in the normal and experimental cat: Anat. Rec. 55 : 36, 1933.

139. Griffin, C.J. Chromatolysis in the mesencephalic nucleus of the 5th cranial nerve after section of the maxillary nerve ventral to the spheno-palatine ganglion. (in preparation.)
140. Kollros, J.J. & McMurray, V.M. The mesencephalic V nucleus in anurans: *J. Comp. Neur.* 102 : 47 - 63, 1955.
141. Corbin, K.B. & Gardner, E.D. Decrease in the number of myelinated fibres in the human spinal roots with age: *Anat. Rec.* 68 : 529 - 536, 1937.
142. Gardner, E.D. Decrease in human neurones with age: *Anat. Rec.* 77 : 529 - 536, 1937.
143. Brody, H. Organisation of the cerebral cortex: A study of ageing in the human cerebral cortex: *J. Comp. Neur.* 102 : 511 - 556, 1955.
144. Hunter, J.I. The influence of the sympathetic nervous system in the genesis of the rigidity of striated muscle in spastic paralysis: *Surg. Gynaec. & Obst.* 39 : 721 - 743, 1924.
145. Kuntz, A. & Kerper, A.H. The sympathetic innervation of voluntary muscle: *Proc. Soc. Exp. Biol. & Med.* 22 : 23 - 24, 1924.
146. Coates, A.E. & Tiegs, O.W. The influence of the sympathetic nerves on skeletal muscle: *Aust. J. Exp. Biol. & Med.* 5 : 9 - 46, 1928.
147. Van Dijk, J.A. The part played by the sympathetic innervation in producing postural tone in the wing of the pidgeon: *Arch. Neerl. de Physiol.* 15 : 114 - 125, 1930.
148. Van Dijk, J.A. The effect of stimulation of the cervical sympathetic cord upon the function of cross striated muscle in the pedgeon. 15 : 126 - 137, 1930.
149. Spiegel, E.A. Zur physiologie und pathologie des skelett muskeltonus: Berlin, 1923.
150. Kuntz, A. & Kerper, A.H. Experimental observations on the functional significance of the sympathetic innervation of voluntary muscles: (*Proc. Soc. Exp. Biol. and Med.*,) 22 : 25 - 28, 1924.
151. Kuntz, A. & Kerper, A.H. An experimental study of tonus in skeletal muscles as related to the sympathetic nervous system: *Amer. J. Physiol.* 76 : 121 - 144, 1926.
152. Hunter, J.I. The postural influence of the sympathetic nervous system: *Brain*, 47 : 261 - 292, 1924.
153. Tower, S.S. A study of the sympathetic innervation of skeletal muscle: *Amer. J. Physiol.* 78 : 462 - 493, 1926.
154. Coates, A.E. & Tiegs, O.W. Are the skeletal muscles of the extremities directly innervated by sympathetic nerves? ; *Aust. J. Exp. Biol. & Med.* 8 : 99 - 106, 1931.
155. Buttner, H.E. & Heimbrecht, B. Ueber den einfluss des sympathicus auf den verkurzungsdruckstand des muskels: *Pfluger's Arch.* 221: 93 - 103, 1928.
156. Schneider, K. Der einfluss des sympathicus auf die quergestreifte muskulatur: *Pfluger's Arch.* 222 : 415 - 419, 1929.

157. Schneider. K.

Der einfluss des sympathicus auf die
quergestreifte muskulatur: Pfluger's Arch.
227 : 293 - 300, 1931.