Changes in Internaxillary Dimension

Effect of Decrease in Vertical Dimension

Coulourites (1955) states that with an abnormally large freeway space the function, phonetics and aesthetics of the prosthesis will undoubtedly be affected and consequently cause hardship and suffering to both the patient and the dentist.

Intra-oral Effects

Intra-oraly the effects of an overclosed vertical dimension are evidenced by a decrease in the cubical space of the oral cavity. The tongue at rest normally completely fills the oral cavity and with over-closure there is a tendency for encroachment upon this space resulting in posterior displacement of the tongue. This as pointed out by Sholet in 1946 will cause difficulty in speech, in swallowing, and in mastication. This is the cause of much general discomfort. Graham (1950) has pointed out that under such circumstances, the tongue, over a period of time, changes shape, becoming flatter and broader. When new dentures are constructed at the correct vertical dimension, this abnormal tongue is now restricted, and is level with the cutting edges as it was with the natural teeth and old dentures before resorption. The restricted tongue tends to dislodge the new dentures and speech difficulties and tongue biting may be experienced. Most patients, however, with perseverance conquer these disabilities and the tongue returns to its normal shape.

Sears has pointed out in 1952 that an over-closure of the mandible will result in an upward positioning of the anterior part of the mandible with a consequent increase in force applied through the dentures to the anterior section of the alveolus, resulting in resorption of the bony process and flabbiness of the ridge.
Effects on the Joint

Trebitsch in 1950 points out that function within normal limits is necessary for the health of the joint, and that this function is fundamentally altered when, as a result of lack of molar occlusion, the joint is no longer relieved of disproportionate strain due to the mastigatory forces. Excessive strain is therefore transferred to the structures of the joint which will, due to the trophic plasticity of bone, readily accept the new strain and by so doing undergo tissue changes.

In a state of reduced vertical dimension the condyle goes deeper into the glenoid fossa towards the processes articularis posterior. The bony process is then an obstruction to any further retreat of the condyle and eventually may suffer trauma.

If the reduction progresses the condyle will be forced to rotate around its imaginary transverse axis, that is the hinge axis. As a result of this type of rotation the mandible can no longer be considered a spatial beam but becomes a third class lever with the fulcrum at the joint. Not only excessive pressure but what is worse - shearing stress - is now transferred to the joint. This latter is the most unfavourable kind of stress that such surfaces can experience. They become fringed and fimbriated and show villi formation.

In such circumstances the disc undergoes degenerative changes. In the initial stage it may calcify at the centre. At a later stage of progressive destruction it may become perforated or disappear altogether. The cartilage and subsequently the bone are split. Particles of various sizes are broken loose and, due to heavy pressure, are impacted into the clefts of underlying tissue. Reactive proliferation of blood vessels and of marrow spaces occur; bone and cartilage appear, with callus formation around tissue.
debris and fibrosis in the bone marrow bordering the affected area, the proliferations are usually found at this periphery, thereby producing irregularly shaped bulging protruberances which may simulate extension of the original articulating surfaces.

When the condyle passes more deeply into the glenoid fossa, the range of its movements becomes limited by the walls and borders of this cavity, thus when a patient tries to compensate by wide excursions for the poor masticatory effect of his bare ridges or unsatisfactory dentures additional damage between the condyle and the fossa is caused.

The pathological movement of the condyle especially during lateral excursions of the mandible may cause the grinding down of the entire tubercle and considerable deformity of the condyle.

A vicious circle has begun. Altered function has caused tissue changes with irregular attempts at repair. These tissue changes further cause aberrations from the previously healthy function. Finally the essential features of a chronic destructive inflammatory process, arthritis deformans, are established. In the advance stage the salient clinical signs of the disease are:

1. Reduction of Vertical Dimension
2. Rotation round the condyle in the first phase of opening movement.
3. Clicking and crepitation can frequently be heard and felt in the joint cavities.
4. The muscles and joint are stiff and initiation of movement is slow and difficult.

Gosten in 1934 has pointed out that an arthritic condition develops when there is loss of molar support or over-closure of the mandible, and a series of symptoms arising from this posterior superior dislocation of the condyle present these symptoms being
nearly all reflex in character. This will be dealt with more
fully under the heading "Costen's Syndrome".

Harris (1938) postulates the theory of bone deformation due
to abnormal muscle pressure and notes its effect on condylar
pathology. In cases which he classifies Class 2 condyle, where there
were several teeth lost and in which there was an apparent loss of
3 - 10 m.m. of vertical dimension, and in which there was deep
over-bites, he found that there was a bending forward of the
condyle at the neck indicating the backward thrust of the condyle
had been opposed by muscular force, tending to change the shape
of the bone in the region of the neck of the condyle. Likewise
it has been found that this same muscle - external pterygoid -
has pulled the meniscus forward so that the articulating surface
of the meniscus has approached almost a 45° angle anteriorly.
There was in those cases no evidence of the meniscus being worn.
In his Class 3 condyle, in which there has been an apparent loss
of from 10 m.m. to a maximum loss of vertical dimension, was found
the greatest destruction to the glenoid fossa. There were in many
cases tremendous changes, not only of the condyle itself but also
and particularly in the neck of the condyle. Several of those
cases showed the condyle forced upward and backward in the glenoid
fossa into the non-articulating portion of that fossa. The posterior
portion of the capsular ligament was worn through and the articular
membrane of the floor of the glenoid fossa has been worn so that
bone has been working against bone in various ranges of movement.
In one or two cases it was apparent that the meniscus itself had
been dragged completely off the head of the condyle and the major
portion of its body was lying on the upper head of the external
pterygoid muscle. In only two cases in the series of more than
100 was a perforation of the meniscus found. Harris noted no
perforations of the tympanic plate. Even in the few cases in which
it was obvious that bone was working against bone in various ranges of mandibular movement. He feels that this is due to the process being a slow one, caused by the continual breaking down of occlusion and because the bone at the floor of the glenoid fossa is adaptable to the pressure which it has to bear. Harris makes several conclusions,

(a) In no instance has a Class 2 or Class 3 over-closure case had a condyle and meniscus presenting a normal picture as found in Class 1 or normal cases.

(b) The degree of abnormality becomes progressively more severe as the occlusion continues to break down.

(c) Since 85% of the 100 cases studied are placed in the Class 2 and Class 3 it is evident that most restorative work is built to the recorded path of a deformed bone.

(d) Such restorative work merely perpetuates the existing deformity.

(e) Restoration of the vertical dimension relieves the pull of the external pterygoid muscle on the condyle and meniscus and allows the other muscles associated to recover.

According to Vaughan (1954) tenderness of the anterior wall of the external acoustic meatus is produced in all cases of temporomandibular joint involvement. This tenderness can be extreme, if deep seated. It occurs in varying degrees whether there is an anterior or posteriorly displaced condyle. This is an invaluable aid in diagnosing temporomandibular joint complaints.

**Facial Changes**

These are caused by an abnormal mandibular position, affecting the overall length of the face, together with its proportions, and due to a change of muscular tonus of the muscles of facial expression. Graham (1950) states that the facial changes that occur are shortening of the lower third of the face with a sharpend chin effect, loss of lip fullness, puckering of the lips, creasing of the corners of the mouth and flabbiness of the cheeks.
Many authors comment on these changes in facial appearance. Bolender (1956) points out that the chin becomes too prominent, the lips lose their fullness and the vermilion border area is reduced to approximately a line.

Shohet (1946) indicates that the mouth assumes a broadened proportion, there is an outward roll to the lips which appear pouchled, a prominent chin is effected and generally senile expression.

Rowell (1950) adds that the abnormally compressed lips give a fullness to the cheeks and cause crease tissue folds of the chin and upper lip. As a result of lack of tone of the orbicularis oris muscle it may no longer prevent the escape of saliva from the mouth at the commissures of the lip thus causing the development of chronic sores — angular cheilitis — in some cases.

Harris (1938) says facial evidence of this is shown by shortening of the lip, which means that the distance between the origin and insertion of the group of muscles known as the muscles of expression is shortened.

Burtenshaw (1948) offers a classification of facial changes or disfigurements caused by wearing the dentures too long. A These are patients who had normal or slightly receding lower jaws prior to alveolar resorption. They develop a characteristic weak expression after resorption and are marked by a shortened chin with the point in advance of the normal perpendicular, the convex portion of the chin being extremely large. Both lips are thin with the upper very much shortened and the lower slightly in advance of the upper. The mouth appears in width to equal twice the length of the chin.
B These are persons whose expressions become aggressive even brutally so sometimes after alveolar resorption. They are developed from those whose lower jaws always were protruding or from normal jaws where there was extreme alveolar resorption in the upper jaw. Type B differs from Type A in that the chin is not so short vertically, but that the point is a great deal more in advance of the perpendicular. Both concave and convex portions of the chin are poorly developed. The lower lip is large and protrusive, the upper regressed. The mouth does not give the effect of great width as in Type A. He also says abnormal muscular tonus of the orbicularis oris causes a falling in of the mouth and closer contact of the lips followed by the folding of the corners. These folds become moist and result in very painful cracks.

Muscular Changes

With mal-positioning of the mandible, obviously the muscles which are attached to it will be displaced, being either slackened and grossly inefficient or overtensed and capable of producing bone deformation.

Harris in 1938 points out that one of the most important physiologic characteristics of skeletal muscles is that they function properly when the normal distance is maintained between the origin and insertion of the fibres and that this type of muscle loses tone and strength when the distance between these two points is shortened. Conversely tone is increased beyond normal when the distance between origin and insertion is increased. He shows that loss of vertical dimension affects in a similar manner the anatomic structures known as the pendant muscles of the mandible. This group runs from the lower borders of the mandible to the hyoid bone and from the hyoid bone to the structures below. The mandible acts similarly to a sack
holder for these groups of muscles and their normal positions are altered by its displacement. Harris agrees that with a decrease of the vertical dimension of the face the masseter, temporalis and internal pterygoid muscles become shortened and therefore lose strength, but the external pterygoid is lengthened from its origin to its insertion and therefore gains in strength.

More recent evidence would seem to indicate that this latest statement is not essentially correct. The work of Boos has indicated that a muscle functions with maximum power at the specific length and above and below this length there is a loss of power. It would seem that increasing the length of the external pterygoid does not increase its strength, but there would be an increase in tone of the fibres, which would in itself provide sufficient force to mis-shape the bone to which it is attached.

Harris continues: It would follow that over-activity on the part of any muscle would change the bone to which it is attached and this is true in the case of the external pterygoid and its attachments to the condyle and meniscus. When the vertical dimension is shortened and the mandible is allowed to be thrust upward and backward the distance between the origin and insertion of the muscle is increased and a force is exerted on the condyle. If we recall also that the thinnest part of the mandible is just below the condyle in what is known as the neck of the condyle and applying Wolff's law that the external form and internal structure of living bone is determined by the muscular forces on it, we must expect changes in the position of the condyles and meniscus.

Standard and Lepley (1955) endorse the statements of Harris, describing how over-closure may produce a forward bend of the condyle due to increased muscular tension of the external pterygoid muscle.
Rogers and Applebaum (1941) comment on a case where the angle of the mandible was measured at 142° - the normal being in the region of 110°. There was marked closure of the bite which resulted in a decrease of inter-maxillary distance and also in the distance between the origin and insertion of the masseter, temporal, internal pterygoid muscles, and a loss of tension of these muscles followed. The regions which changed are those into which the abovementioned muscles were inserted. These same workers also show a mandible illustrating conditions resulting from neglect. The protractor recording an angle of 156°.

According to Rogers and Applebaum the normal angle of the mandible is due to maintenance of function. They feel that sagging muscles caused not only a marked facial deformity but likewise reduction in biting power and a certain degree of atrophy from lack of function following loss of teeth. In particular they suggest that a causal relation exists between changes in the angle of the ramus of the mandible and a decrease in the tension of the bite. Resorption of bone occurs into the region into which these sagging muscles are inserted. Widening of the angle of the mandible is caused by a decrease in tension of the sling formed by the internal pterygoid and masseter muscles. These authors also describe a correlation between the maintenance of correct vertical height and hence correct muscular function, with the process of changing of the angle of the mandible as age increases. They present a case whereby correct muscular function was maintained during life by means of "stop action" teeth which served to maintain the vertical dimension. Despite the absence of the majority of teeth the angle of the ramus did not change and this was ascribed to the maintenance of correct length of the muscle fibres.
Sears in 1952 also points out that bending of the bone or an increase in the obtuseness of the mandible is a result of over-closure or loss of vertical height of the face.

The muscular change due to over-closure of the mandible and associated with the decrease from the normal physiologic length of the muscles of mastication is the loss of power capable of being exerted by these muscles at the level of occlusion. This phenomenon is sometimes employed to reduce load on intra-oral tissues which may be incapable of withstanding the full force of the masticatory musculature. That is, in some cases it may be indicated to effect a slight over-closure of the mandible in the interests of comfort of the patient.

**Acoustic Defects**

These are described more fully in the Section on Costen's Syndrome. Some writers believe that in the case of over-closure of the mandible there may be decreased hearing ability due to closure of the Eustachian tubes caused by backward pressure of the tongue, thus causing a change in the auricular pressures with subsequent deafness. Others describe the deafness to a pressure on the anterior cartilaginous wall of the external auditory meatus caused by a posterior displacement of the condyle. Such an idea is expressed by Harris (1938) who points out that in some cases of partial or complete loss of teeth, pressure effects have been noted to have taken place upon the cartilaginous portion of the external auditory canals. MONSEN had drawn attention earlier to his conviction that this is the cause of a great deal of unnecessary deafness.

On the other hand Juneman (1941) feels there is no scientific proof to any statements regarding the improvement of hearing by increasing the inter-maxillary distance. Increasing this distance does not improve hearing ability as is shown by audiograms, however it
does relieve Tinnitus and pain about the temporal, parietal and masseteric areas in a great majority of cases. This author feels that the reason for this is unknown.

Restoration of Normal Dimension from a Decreased Position

Rowell (1950) points out that:

1. loss of vertical dimension in wearers of full dentures is usually a slow process and the resultant damage is of a degenerative type with anatomical changes which are often difficult to correct.

2. considerable loss of vertical dimension may occur without alarming symptoms other than a decrease in functional efficiency, and even this decrease may be so slow in its onset that the average patient is unaware of it until told about it.

3. The attempt to restore the correct vertical dimension is in a number of cases fraught with numerous difficulties and so I consider that thought should be given to the degree of initial restoration to be attempted prior to the commencement of such treatment.

Brown in 1936 has pointed out that in re-establishing the correct vertical dimension we

(a) restore the shortened facial muscles to their original length.
(b) remove unnatural strain on the adjacent muscles and
(c) allow space in which these muscles can function.

By increasing the vertical dimension we eliminate the line of the alae of the nose, we narrow the nostrils and turn the corners of the mouth upward. In this manner we re-shape the entire lip.

Concerning the changes in muscular function which are effected by a change of muscle length when an attempt is made to restore the vertical dimension to the norm, all writers are of the opinion that
care must be taken in effecting this change. This is because a change in the muscle tonus from that to which they are accustomed may cause gross discomfort and because of the possibility of fibrosis of the musculature at the shortened length, thus increase in muscle change will be effected only very slowly by natural processes. Another point which needs to be considered is that with the return to normal muscle length increased masticatory pressure is then applied to the supporting tissues of the denture which after years of misuse may not be in a position to withstand it.

Boos (1956) points out that if the change to be effected is greater than 5 to 6 m.m. it is best to proceed cautiously as considerable adjustment to the increased height would have to be made by the patient. Masticatory habits as well as adjustments by the lip and tongue may be difficult. If it is determined that the patient will adjust to the ideal vertical dimension gradually, it may be suggested that the patient have treatment dentures which would approach ideal half-way. Then a year or so later ideal occlusal vertical dimension can be built into new dentures. Consideration must be given to the ridges and the type of patient.

Howell in 1950 points out that it is dangerous to promise a patient that opening the bite some miraculous cure to either facial disturbances, muscular disturbances or acoustic disturbances will automatically result. He indicates that if the forward inclination of the neck of the condyle has been effected due to the hyper-tonicity of the external pterygoid muscle, it must be obvious that re-establishment of the normal overnight is only a pipe dream. He feels that with elderly patients only in special cases should complete recovery of normal vertical dimension be attempted immediately, as in most cases the patients muscles will not adjust themselves to their new circumstances. The desirability
of recovering the lost vertical dimension in successive stages should be considered. Never open the bite so much that freeway space is eliminated as this would cause excessive alveolar resorption until the freeway space is re-established.

The degree of recovery of lost vertical dimension may be determined by considering the age of the patient, the size and condition of the ridges, the size of the denture space, and the mental attitude of the patient to a marked change in bite and facial contour.

Brown again has pointed out that if the patient is well along in years and is wearing a fairly successful set of dentures, even though it may be lacking in every detail from an aesthetic standpoint, it is often wiser to let him alone or at best simply add a few millimetres in width and height rather than try to work for the ideal replacement and run the risk of causing unnecessary discomfort. The older the person is, the harder it is for him to adjust himself to a change. Occasionally we are confronted with cases which need extensive replacements owing to the fact that shortening of the vertical dimension has allowed the mandible to impinge on the anterior wall of the external auditory meatus possibly causing deafness, pain or Tinnitus. In these cases we simply must increase the vertical dimension if we are to achieve the desired results. We have no way of estimating the amount of bony tissue that has been lost, except possibly by inserting our fingers in the ears and feeling for the mandible as it comes back upon closing. No-one can be certain the dimensions are correct. We must simply set an opening which we think is correct, build a case and wait for time to tell us whether we have established the proper dimension.
Effects of an Over-Increased Vertical Dimension

These effects will be noticed intra-orally and musically.

Intra-Oral

These effects are noted intra-orally if the vertical dimension is increased.

Nature attempts to restore the correct dimension by effecting gross resorption of the ridges.

Smith in 1941 points out that it has been observed in dentulous as well as in edentulous patients that after a period of time the muscles will return to their normal length, but only after a sacrifice has been made through the development of detrimental changes in the oral cavity, it will be effected by a resorption of the underlying alveolus.

Mershon in 1937, has indicated that during the process of resorption of the alveolus to accommodate the increased vertical dimension the patient experiences indescribable discomfort, until the bone beneath the dentures resorbs and the muscles are relieved of all stress. Prior to this loss of bone there will of course be considerable tissue soreness; this has been indicated by Kimball in 1954 who lists an increased vertical dimension as a mechanised factor which will induce trauma and is quite likely to result in a considerable degree of tissue soreness. The tissue soreness is due to an overloading of the soft protecting mucosa with resultant inflammation, pressure necrosis and ulceration. It may be this surface tissue reaction which induces the underlying bony changes or it may be simply a case of direct pressure.
Muscular Changes

In discussing musculature Tench (1938) indicated that nature may shorten muscles but she rarely if ever increases their functional length. Increasing the elevation of the occlusal surfaces of human teeth for the purpose of increasing the vertical dimension of the human face would require that nature increase the functional length of the muscles of the masticatory apparatus. Hershon in 1937 has pointed out that nothing known to science except accident, death or surgery can increase the length of muscle after it has reached the fullness of its growth. Moreover, he indicates that muscles are always dominant over the bone. To this end the prosodontist must determine the length of the posterior teeth which will be in conformity with the length of the patients muscles of mastication.

Tench quotes muscles do not increase in length to accommodate dentists. More recently in 1953 Gurian also comments: "It seems that muscle fibres have a pre-ordained length. The state of physiologic rest demands that they be not stretched nor contracted. The determination of this factor is exceedingly important — it may often spell the difference between success and failure."

This summarises the approach of most workers to the problem of over-extension of the muscles.

Bolender points out that the terrific strain will cause fatigue and strain in the muscle controlling the mandible.

Monson (1932) feels that the excessive muscular forces will carry the mandible forward.

Another sign of over-opening of the bite is the loss of ability to open the mouth sufficiently wide to incise food. This is effected because of the large bulk intra-orally and because of the hyper-tonicity of the controlling musculature.
Burtenshaw feels that this will cause a discomfort which is both physical and psychological. It may also be evidenced by a clicking of the teeth due to the interference that they provide during swallowing, phonetics and attempts at mastication. Innie et al (1958a) also comment on this discomfort. Refer Fig.1 Page 23.

Schoet in 1946 summarises the symptoms and effects of abnormally open bite.
1. A stiff expression about the mouth
2. An abnormally long look of the lower third of the profile.
3. Loss of the natural curve of the upper lip which appears flat and stretched.
4. Difficulty in taking large bites and fatigue of the muscles of mastication.
5. Inability to get the lower jaw into the physiologic rest position.
6. Speaking with clenched teeth which will shatter if the occlusal surfaces are porcelain.
7. Infra occlusion of the posterior teeth.
8. Absorption of alveolar bone.

It will be noted that no reference is made to temporomandibular disturbances caused by an opening which is excessive of the vertical dimension. This in fact is due to the resorption of the underlying tissues of the denture which will occur in favour of pathology of the temporomandibular joint.

Decrease of Vertical Dimension of Normal

Usually this will occur pathologically. Burtenshaw in 1948 points out that sometimes a decrease in vertical dimension is purposely effected to reduce mastigatory force from a patient's dentures, as for example when underlying ridges are of unsatisfactory dimensions, or lack of satisfactory tissue coverage. On such occasion
it is important to provide a balanced occlusion. If the decrease in vertical dimension is brought about by lack of compensation for the resorptive processes then the change of position of the mandible will reduce two contact areas and cause cuspal interference in functions leading to rocking or even displacement of the dentures.

Due to the rapid deleterious effects experienced when the denture space is impinged upon when the vertical dimension is excessively opened most authors advise that in cases of uncertainty of correct vertical dimension the error should be made favouring a slight decrease in the dimension that a more comfortable result may be attained for the patient.
Costen's Syndrome

In 1934, Costen expounded his theory to the medico-scientific world, in an article which he published in "The American Journal of Otolology, Rhinology and Laryngology".

As a result of the interest this article created, many medical practitioners were referring their patients to dentists, expecting cures of deafness, and many dentists were expecting to achieve just such results by radically opening the bite, that is increasing the vertical dimension; sometimes even beyond the level of rest position.

Experience later demonstrated that success was obtained only in a minority of cases.

Costen outlined a group of symptoms which he believed were directly caused by loss of vertical dimension. Among these symptoms included:
1. Pain - dull within and about the ears.
2. Tinnitus - a low buss in the ears.
3. Stopping or stuffy sensation in the ears, marked about meal time.
4. Mild catarrhal deafness which is improved by inflation of the Eustachian tubes.
5. Dizzy spells relieved by inflation of the Eustachian Tubes.
6. Tenderness to palpation of the joints.
7. Typical sinus headaches after sinus or eye involvement has been corrected or typical headaches when the sinus or eyes have been found negative.
8. Traumatic, noisy, painful, limited or excessive movement of the temporo-mandibular articulation.

10. Burning or prickling sensation of the tongue, throat and side of nose. Accompanying this sensation a slight herpes of the affected part may be present.

Block (1947) an advocate of Costen's theories, grouped these symptoms into three catalogues:

1. Acute local pain around and within the temporo-mandibular joint.
2. Headaches and neuralgia on the side of the head, face or in the mastoid region.
3. Eustachian symptoms, such as stuffy feeling in the ears, vertigo, Tinnitus and simulated catarrhal deafness.

Sinclair in 1959 adds to these symptoms a disturbance of the salivary glands, and all clinical manifestations attributable to direct extension or inflammatory process from the injured temporo-mandibular joint to the adjacent anatomical structures.

Cause

Costen in 1934 originally stated that the initial cause was due to a loss of vertical dimension, in this he is well supported by many authors.

Block (1947) indicates that some cases are so definitely the dentist's own problem for both diagnosis and treatment, that consultation with a physician is not necessary. These are the cases visibly resulting from:

1. Loss of vertical dimension owing to loss of and unreplaced posterior teeth.
2. Excessive wear of posterior teeth.
3. Dentures allowing over-closure.

In 1948 Walsh reports that there is an overwhelming weight of evidence that loss of vertical dimension or an alteration in the
functional position of the mandible causes changes in the temporo-
mandibular joint, and these changes lead to the onset of many symptoms
which can cause considerable disability to the patient. Not all patients
present with all the symptoms, in fact an isolated symptom may appear
and not be recognised as a manifestation of the syndrome. The response
to treatment varies according to the degree of existent abnormality
and nerve trauma. The average patient reports relief from pain within
48 hours of joint rehabilitation. Followed by complete relief
from all symptoms two or three weeks after treatment. Walsh
emphasises that bite rehabilitation is necessary and this may
not involve only bite raising.

Agreeing with Walsh, Rowell in 1950 points out that associated
with the other signs of loss of vertical dimension may be these
symptoms of Costen's Syndrome. The signs, however, may be entirely
lacking in cases with gross loss of vertical dimension. On the other
hand symptoms similar to those of Costen's Syndrome may exist where
a careful clinical examination indicates that there has been no apparent
loss of vertical dimension so that some other possible cause for the
symptoms must be found.

Symptoms

The symptoms of pain are, according to Costen and his supporters,
for example Goodfriend and Block, directly related to anatomical
involvement of nervous tissue. The ear symptoms depend on actual
involvement of the Eustachian Tube and tympanic structures. They feel
that the sinus symptoms are more apparent than real. The actual source
of this group of complaints was confirmed by marked improvement which
followed correction of the over-bite, renewal of molar support to
take pressure off the condyle and establishment of proper articulation
of the condyle within the fossa. This original concept is now somewhat in doubt as the forthcoming paragraphs will indicate.

Pain of the Temporo-mandibular Joint

This seems to be the least contentious phase of the symptomatology. Most authors believe that the joint pain is due to a direct inflammatory process within the joint. Amongst these are Graham (1950) and Block (1947) who feel that the inflammation is due to traumatic injury within the joint resulting from displacement or trauma within the glenoid fossa.

Sinclair in 1959 says that in these cases of arthritis, movement of the affected joint may be limited and the joint is tender to digital pressure in comparison with the normal joint.

Sicher in 1951 attaches great importance to the impingement of the condyle on the muscular and well innervated connective tissue behind the meniscus. The impingement of this tissue can taken place when there has arisen disorders of the joint. Sicher regards this as the main source of pain within the joint itself and the source of some pain by reference to adjacent structures.

Ear Symptoms

Early writers, for example Batson (1938), Harris (1938), ascribed the cause to partial or complete blockage of the external auditory meatus by the retruded condyle.

Costen (1934) himself ascribed the deafness and dizziness to compression of the Eustachian Tubes. He explains it thus: "With the joint in normal position the external pterygoid muscle is taut and the tensor veli palatini borders the tube exteriorly on an almost straight line. Between these, lie only connective tissue and adipose tissue,"
and posteriorly, close to the tube are the auricula-temporal nerve and inferior alveolar nerve. If the jaw is brought upward in a position of marked over-bite, the upper head of the internal pterygoid muscle is relaxed and a bundle of soft tissue piles against the tube. The tensor veli palatini muscle appears loose, preventing its function in tightening the soft palate and opening the Eustachian tube during deglutition. Especially during the act of swallowing when the tensor palatini muscle usually opens the Eustachian tubes, the compressing effect of the tissues on the tube, from the over-bite is present and prevents the opening. The very looseness of the capsule of the mandibular joint and its restraining ligaments now works to further exaggerate the pushing of the tissues towards the tube. With each overacting closure of the mandible by the masseter and internal pterygoid and temporal muscles, the condyle is shoved upward against or through the atrophic or perforated meniscus or it moves backward to the tympanic plate and pushes medially on one side or the other through the loose capsule.

Harris in 1938 is in agreement stating that when the mandible is over-closed the tongue is moved posteriorly together with its associated structures impairing the activity of the tensor veli palatini and the levator palatini. As these muscles have their origin on the side of the Eustachian tube and from the cartilaginous part of the tube any condition which interferes with their physiologic action interferes with the drainage through this tube.

Haves also of 1938 has pointed out that a high percentage, 15 - 25% of all deafness results from advanced catarhal deafness and somewhat less from senile nerve deafness. The actual cause as far as can be determined is, according to Wright and Prentice, attributed to one of two conditions. The first of these is a reduction of the normal vertical dimension in the molar region, causing a relaxation
of tissues and ligaments crowding against the Eustachian tube and maintaining a partial or complete closure. This prevents proper drainage of the posterior nasal and Eustachian passageways. Thus there is a disturbance of the allocation or pressure within these cavities.

Shambough as quoted by Batson (1933) says: "While unusually severe cases of malocclusion might conceivably cause direct temporary pressure on the Eustachian tube, we have yet to see any case of deafness in which the defect could be reasonably ascribed to temporomandibular joint malocclusion, though we have been on the lookout for these cases since Costen's original article. Deafness due to this condition must be very uncommon, and we suspect that its importance is being greatly over emphasised".

Batson continues that there are many cases of auriculo-temporal neuralgia and of paresthesia of the tongue and pharynx that can be relieved by opening of the bite. He does not agree with Costen's explanation involving the idea of a passive obstruction to the Eustachian tube. This statement is based on studies made by a nasopharyngoscope. It seems to him that whenever improvement in hearing is found in closed bite cases, after re-opening of the bite, it is due to the restoration of the normal function of the salpingopalato-pharyngeal kinetic chain of muscles.

Tench (1933) states that there is no conclusive proof that impaired hearing has ever been partly restored to normal by opening the bite, and he states that there has been no scientific evidence that hearing can be impaired by a closed bite condition. However, as Graham (1950) says, deafness is a symptom frequently found but sometimes difficult to explain. It is possible that the general disturbance in the joint may directly interfere with the intratympanic pressure.
Sicher (1943) points out emphatically that the explanation of the symptoms as described by Block and Costen is incorrect. He states that the anatomic rationalisation of Costen's Syndrome is based in part on interpretation and elaboration of statements found in one of the other text book of anatomy, and in part it seems to be derived from inspection of anatomic specimens without any consideration for the different behaviour of tissue, especially, muscles, in the cadaver and the living person. Defects in the floor of the bony acoustic meatus are found in about 20% of all persons. Defects of the tympanic bones which are situated further laterally and superiorly are encountered rarely. They are found only in old edentulous skulls and appear to be associated more with senile osteoporosis than with the pressure exerted by a posteriorly displaced condyle. In an evaluation of the possibility of tympanic erosion the presence of the post-glenoid or post-articular process of the temporal bone, should not be forgotten. Although somewhat variable in size it is absent in only a few skulls. Any posterior displacement will be directed first against the post-glenoid process and only the destruction of this process could open the way for the condyle to impact and erode the tympanic plate.

Sicher admits the possibility of a link between referred ear pain and mandibular over-closure. This may be due to a stretching of cramped muscles or vigorous nervous contraction of the muscles. The Eustachian tubes, normally closed, are reflexly opened by the action of the tensor palatini muscle and this is active during phonation and swallowing. Thus muscle extends in its fleshy part between the cranial base and thepterygoid hamulus and its tendon extends into the palate, no change in the position of the lower jaw can influence this muscle to any measurable degree. A piling of soft tissue against the tube by the relaxation of the internal pterygoid muscle is wholly impossible. In life the jaws are actively closed while the muscle contracts, also the upper part of this muscle is mainly tendinous and although in close relation to the Eustachian tube does not change its volume during contraction. Thus we have to realise that
disturbances of the middle ear if really related to closed bite, can be explained only as resulting from an obstruction of the Eustachian tube itself, so that the tensor tympani although contracting normally, is not able to open the tubal lumen.

Symptoms of the Tongue

Costen reports that in this type of pathological joint in which the condyle snaps backwards over the articular disc impacting and eroding the tympanic plate, the chorda tympani nerve passes this spot through the inter-chordae anterior, at the medial end of the petrotympanic fissure. He points out the majority of the fibres of the chorda tympani are sensory and are continued onward through the muscular substance of the tongue to the mucous membrane covering its anterior two thirds. He thus ascribes the pain and burning sensation of the tongue to mechanical interference of the chorda tympani nerve in the joint cavity itself. In this theory he is supported by Block (1917).

Sicher (1948) points out, however, that it is anatomically impossible for this nerve to be directly involved by an averted condyle. The chorda tympani nerve leaves the tympanic cavity through a narrow canal which is situated in the petro-tympanic fissure. The outlet is found behind the posterior attachment of the capsule approximately between its middle and medial third, from this point it continues in the depth of the petro-tympanic fissure to its medial and is well protected from any external pressure. It then turns anteriorly around the downward projecting inner end of the articular fossa, which leans on and is jointed to the angular spine of the sphenoid bone. The nerve passes the angular spine often in a narrow horizontal groove on the medial surface of this structure. Sicher after pointing out that this nerve is out of reach of the condyle, further states that the structure of the nerve is such that mechanical pressure upon it would not cause the symptoms of pain.
The nerve structure lacks the specialised anatomy necessary for the transference of pain stimuli.

Graham (1950) feels that the chorda tympani nerve is probably involved in the general traumatic inflammatory situation which exists in the glenoid fossa in such a case of mandibular displacement.

**Temporal Pain**

Costen (1934) felt that with some of the chewing movements and closures of the jaw the condyle exerts pressure on or near the auriculo-temporal nerve which passes immediately to the medial side of the capsule and between the condyle and tympanic plate to distribute over the temporal region. Costen published further reports in 1936 and 1944. In the former he assumed that the temporo-mandibular articulation was weak medially. He said that on this poorly supported side, the joint would be destroyed by the impact of chewing movements, the condyle would slip medially on closure impacting the nerves and causing pain.

In 1944 Costen again emphasised the impingement of the condyle on the chorda tympani and of the auriculo-temporal nerve. He also makes a reference to the possibility of pain being reflex when sufficient destruction of the joint structure occurs to allow the eroded face of the condyle to rest against the tubercle of the glenoid fossa. Block (1947) also indicates that during the chewing or swallowing movements, in cases of decreased vertical dimension, the condyle may exert pressure on or near the auriculo-temporal nerve which passes close the the medial side of the capsule and between the condyle and tympanic plate to distribute over the temporal region.

However, Sicher (1948) does not agree that the auriculo-temporal nerve passes between the mandibular condyle and the tympanic plate. He places the nerve as running down from the foramen ovale posteriorly on the inner surface of the external pterygoid muscle and reaching.
the mandibular neck below the insertion of this muscle. It then
crosses the posterior border of the mandible at a level below the
insertion of the capsule and also below the level of the tympanic
bone and here it divides into its external ramifications. This
nerve can thus not be impinged upon by any mal-position of the
mandibular condyle.

Graham (1950) indicates that the auriculo-temporal nerve is
in close proximity to the head of the condyle and irritation to this
nerve would explain pain the the temporal region, outer ear and
parotid gland. Such irritation could hardly be caused by direct
interference from the head of the condyle but the general
inflammatory reaction in the region might reasonably be said to
effect the nerve.

Sinclair in 1959 points out that the anatomical structures
adjacent to the joint may become involved in consequent of direct
extension of an inflammatory process from the joint cavity.

**Vertex Pain**

Costen originally ascribed this pain to injury of the dura after
perforation of the tympanic plate had occurred, as a result of
condylar trauma. The anatomic explanation for pain is simple.
Deep erosion of the bone of the glenoid cavity leaves only a thin
plate between the dura and the condyles. Each closure of the jaw
impacts this evacuated area with the condyle which thus rocks in
the glenoid fossa barely separated by the remaining thin bone from
the dura and its rich nerve supply. He is again supported by Block
in 1947.
Sicher (1948) refutes the ideas of these previous authors. He points out that the bone separating the articular fossa from the middle cranial fossa is normally quite thin – this region is evidently never under pressure. In the occlusal position the thickest part of the articular disc fills the deepest part of the articular fossa and the resultant force of the mastigatory muscles is directed upward and forward so that the condyle presses the thinnest central part of the disc against the posterior slope of the articular tubercle.

Zimmerman (1951) points out that vertex pain could not be of dural origin. He indicates that pain fibres in the dura are known to exist only along the meningeal blood vessels. As has been pointed out previously we would expect the deposition of new bone on the brain side of the roof of the glenoid fossa if there was undue pressure on this bone.

**Glossopharyngeal Neuralgia**

This may result, according to Costen, from pathology in the temporo-mandibular joint as a result of the association of the chordae tympani and auricula-temporal nerve with the mouth. This association occurs via a sensory connection to the otic ganglion.

Sicher has pointed out that the auriculo-temporal nerve is well protected from any infringement by a displaced condyle. He further points out in 1948/1951 that the chorda tympani nerve contains no pain fibres. This is in agreement with other workers. He also points out that the otic ganglion has no sensory connections and hence eliminates the possibility of a glossopharyngeal neuralgia as explained by Costen.

Antoni (1955) reports that recent investigation by Costen's workers has shown that there are pain fibres in the chordae tympani. He does not, however, quote a source of his information.
One must not overlook the concept of Batson and Shamaugh (1938) who refer to a disturbance in the salpingo palato-pharyngeal kinetic chain of muscles, which one could easily expect to occur in cases of lost vertical dimension. This may produce pharyngeal symptoms without the involvement of trigeminal nerve pathways.

**Referred Pain**

Referred pain was mentioned by Costen (1936) as being a possible source emanating from the temporo-mandibular joint. Since the articulator capsule is supplied by branches of the auriculo-temporal nerve there may be referred pain from this nerve source. Zimmerman (1951), Grewcock (1955) Sicher (1948). This pain may be referred to any of the structures supplied by the trigeminal nerve. Thus there may be pain referred via the lingual nerve to the tongue.

More recently however, Sicher (1955) regards most of the extra articular pain as being caused by muscle spasms. This is in accordance with the work of Scharants (1955) who regards muscles as being the main factor in the production of pain. He says temporo-mandibular joint pain may be due to a painful self perpetuating spasm of the mastigatory muscles.

Dechaume and Al (1955) suggested another cause of the development of pain as well as other symptoms in the joint. They consider that irritation caused by disturbances of the articulation spreads along sympathetic fibres which are found periarterially in proximity to the articulating surfaces, as described earlier, to give symptoms of the most varied types. They say the temporo-mandibular joint pain is accompanied by certain secondary signs, namely facial oedema, red patch areas, articular or salivary parathesis, as well as crackling, crepitation and trismus.
Sinclair (1959) points out that as the auriculo temporal nerve is a branch of the third division of the trigeminal nerve, any combination of referred pain is possible throughout the distribution of the fifth cranial nerve, including the tracks of the fifth nerve in the central nervous system and its ramifications to the sensory cortex and also the cervical segments related to the fifth nucleus as far as the third cervical nerve.

It would seem to us now that the original concept of Costen's Syndrome does not as such exist. The symptoms may or may not appear collectively, and the cause may not be solely loss of vertical dimension but any type of occlusal arrangement.

Thus the condition is one of an arthritis caused by trauma in the glenoid fossa, and it is this arthritis which apparently causes the symptoms, either by involvement of the associated nerves and structures in the area of inflammation or by reference of this disturbance of the nerve through the pathways of the trigeminal nerve.

If in a particular case this arthritis is caused by loss of vertical dimension and the symptoms concur with those postulated by Costen in 1934 then this case may be correctly termed Costen's Syndrome. But it must be realised that the syndrome is only part of a temporo-mandibular arthritis.

Cheraskin and Langley in 1956 say whether or not there is a specific syndrome as described by Costen, is being debated. However at least it is unarguable that derangements in the oral cavity, especially in occlusion, may lead to mechanical and neurologic signs. It is the recognition of this relationship, rather than the specific terminology which is of cardinal importance.
Muscle Physiology and Neuromuscular Data

Structure of Skeletal Muscle

Even the smallest skeletal muscle seen in anatomical preparation is made up of very large numbers of muscle cells, sometimes called muscle fibres. The individual cells which are from one to forty millimetres long, occasionally 120 mm., and from 10 to 100 μ in diameter, have a tough outer membrane, the sarcolemma, and each possesses numerous nuclei which lie immediately below this covering. The contractile cells are directly connected to the tendon. The cell as seen in a histological preparation is filled with numerous myofibrils each approximately one millimetre in diameter embedded in sarcoplasm. When examined in the ordinary light microscope the fresh fibrils appear to be transversely striated due to the alternation of zones of higher and lower refractive index. The "A" bands are of high refractive index and are birefringent, while the "I" bands are of lower refractive index and isotropic. The centre of the "I" band is crossed by the "Z" line, or Dobie's line of high refractive index; the centre of the "A" band has a region "H" of low refractive index (Hensen's line). Huxley finds that during passive stretch and in active contraction the "A" band remains approximately constant in length, the change in length occurring mainly by a change in the length of the "I" band. This is because the fibril is made up of two sets of overlapping, interpenetrating filaments. (See Figure 2). The region between the filaments is in life filled with a solution of salt and soluble proteins. Skeletal muscle cells are innervated by myelinated nerve fibres, 8 to 20 μ in diameter, derived from the large motor cells of the anterior horn of the grey matter of the spinal cord. When a nerve fibre reaches the muscle its neurilemma merges with the sarcolemma under which the axon spreads out to form the motor end-plate. Each muscle cell may have one, two, or more end-plates.
these may be supplied by branches of a large nerve fibre or by branches derived from several nerve fibres.

Emig (1951) explains the mechanical changes when a muscle is effectively stimulated and a number of almost instantaneous changes takes place. It shortens or attempts to shorten, and at the same time electrical, structural, chemical and thermal changes occur. These changes are reversible. They can repeat themselves after very brief intervals and result in the production of tension which is the fundamental property of muscles.

**Structural Changes**

Evidence indicates that the complex hydrophobic colloid, actomyosin, is the substance that endows muscle with the property of contractility. Muscle contains about 10% actomyosin which is located in the myofibrils. These are built of bundles of small filaments extending continuously in straight lines with the fibril axis. They shorten but do not coil in contraction. Contraction and length changes are the result essentially of changes within filaments consisting possibly of a folding of the molecular chains from which the filaments are built, rather than disorientation of the filaments.

**Mechanical Changes**

The basic mechanical changes associated with contraction are the development of tension, and shortening of the muscle. One of these factors that determines the tension produced at any given time is the number of fibres or motor units activated by a given stimulus. Gradation of contraction and movement is possible, and it is based on the number of units responding. The total amount of tension exerted by the muscle represents the sum of tensions of the individual active motor units. The parallel and pennate arrangements of muscle fibres permit the mechanical additive effect of increasing the number of units that are recruited into activity.
Relation between Length, Tension and Shortening

Emig (1951) states that skeletal muscle is extensible. The tension varies directly as the lengthening of the fibre.

Mershon (1939) feels, however, that muscles lack elasticity but have the power to contract and relax. He states that muscles are only inert masses of tissue until they are made to function by the nervous system.

Atwood (1957) disagrees and points out that muscle is not merely an elastic band but has both physical elasticity and physiologic elasticity. This latter is defined as "the ability of the muscle to surrender contraction, to discontract, to relax".

This is an active living response involving neurologic activity, and Wright (1956) has shown if a paralysed muscle is stretched a very small increase in tension results, that is elasticity.

This elasticity of unstimulated stretched muscle is due to the sarcolemma as at rest the acto-myocin acts in a plastic manner.

During contraction, the long polypeptide chains actively exert tension and resist stretch. The relation of the tension developed to the length of a contracting muscle is important because it represents a dynamic characteristic of muscle. The relation is a fundamental one in relation to studies of dynamics of motion in man. The rest position of muscle is close to the maximum extended length. From the standpoint of exerting tension the muscles function most efficiently at rest lengths. (Bowditch). The tension of a contracting muscle increases with length over the limits of physiologic utility.
Contractions can also be graded in frequency of stimuli. Motor nerves may be stimulated simultaneously; however, the motor impulses are highly asynchronous. This fact is evident in the smoothness in the grading of muscular responses. The smooth chewing action in the mastication of food and the rhythmic movements in speech indicate the smoothness of muscular responses.

The nervous system takes over the function of integration of muscular activity, that results in posture and purposeful movements.

Thompson in 1941 discussing the antagonism of muscles points out that they are controlled through a mechanism of reciprocal innervation and reciprocal inhibition. All the voluntary reflexes have antagonists whether they are the other muscle, gravity, or a combination of the two.

Sherrington's investigations conclusively proved that as one muscle group contracts the antagonist relaxes. In opening the mouth the stimulus that causes the upper head of the external pterygoid muscle to contract, causes the antagonist, namely the temporal, masseter and internal pterygoid to relax at the same time. The muscles of mastication are highly co-ordinated through their innervation so as to permit complex jaw movements. They function not only in mastication but also in deglutition and speech and in the maintenance of posture.

Hershon (1939) agrees that muscles work in groups or pairs, and Kazis (1946) has stated that each mandibular movement is produced by a contraction of one group of muscles and a proportional relaxation of an opposing group of muscles.
A more recent and more correct approach is expounded by the Shpuntoffs in 1956 who consider the muscles attached to the mandible act as a myostatic unit. Such a unit is defined by Szentagothai as consisting of the muscles of the mandible that are mutually dependant, together with their reflex connections excitatory and inhibitory. Thus they point out that the muscles of mastication act as a unit, rather than a series of units each with its own individual antagonist. Necessarily the different parts of this myostatic unit are co-ordinated by the central nervous system.

Perry and Harris (1954) have indicated that all the muscles attached to the mandible have some active part in mastication, either in contracting or relaxing. The contribution of muscle to the oral cavity is governed by the architecture of the jaw and muscle attachment, by occlusal relations of the teeth, by demands of various types of food and by the innate or acquired functional pattern of the individual. He indicates that the mandible is moved by the action or inter-action of groups of muscles as follows: the initial movement of depression is started by the depressor portion of the group, but the other portions do not remain quiescent for they are acting as a resistance. The more the mandible approaches a position from which it will be elevated the more do these portions of the group tend to contract so that when this position is reached the muscle groups are in a state of equilibrium. The depressor portion now tends to relax and the elevator portion tends to contract more strongly. Elevation is opposed therefore by the gradually increased resistance of the depressor portion of the group until the teeth are firmly occluded, the two portions again being in equilibrium.

To do work a muscle must shorten, and the work done by a muscle is equal to the tension developed times the distance through which it shortens. The realisable work is dependant upon the fibre length. When each fibre is stimulated it must contract maximally or not at all. The elongation of an anti-gravity muscle by less than 1% of its length stimulates the muscle spindles and a sustained reflex
contraction results. (Best and Taylor).

The movements produced by muscles which are under voluntary control is dependent upon the number of fibres active, which is a function of the number of motor units which are active at any one time. The action of each motor unit is one an all or none basis and the mode of action of the moving parts is determined by the co-ordinating activity of the nervous system.

Wright in 1956 has demonstrated that muscles are unable to relax. All efferent fibres passing to skeletal muscles are excitatory, that is they produce contraction of the muscle fibres. There are no efferent fibres which on stimulation produce relaxation or elongation of the muscles, that is there are no inhibitory efferents. Skeletal muscle contraction under natural conditions always results from discharge of the motor neurones; muscular relaxation is the result of a decrease or cessation of discharge of the motor neurones.

Motor Unit

Skeletal muscles receive their motor nerve supply from the ventral horn cells of the spinal cord and the corresponding cells in the motor cranial nuclei. Each ventral horn cell (or cranial equivalent) supplies a considerable number of muscle fibres varying with the individual muscle from 5 to 150.

A ventral horn cell and its efferent fibre is termed a motor neurone.

A motor neurone together with the group of muscle fibres which it innervates is called a motor unit. The smallest group of muscle fibres that can ever be employed naturally in the body, either in reflex or voluntary activity is obviously that supplied by a single motor neurone.
Pruzansky (1952) indicates that a muscle comprises an assembly of motor units. Not all of these units are of the same size and the number of muscle fibres innervated by a single motor neuron is referred to as the inervation ratios. Smaller inervation ratios make possible better nervous control for delicacy of movement.

Emig in 1951 points out that available evidence seems to indicate that motor units are spread through the muscle, in fractional small bundles rather than in one single bundle.

The gradation of muscular responses is due, in part, to the number and frequency of motor unit discharges. The smoothness of the response is in the main a consequence of the asynchronous activity of the motor units.

**Action Potential.**

Pruzansky (1952) points out the formation of action potentials is apparently due to polarization changes accompanying the almost synchronous contraction of the muscle fibres comprising one motor unit. The individual action potential as recorded on the cathode ray oscilloscope possesses the following characteristics:

1. Wave Form
2. Amplitude
3. Duration
4. Frequency

During a normal contraction increased effort will elicit a greater number of motor units discharging asynchronously and in greater frequency. The additive and overlapping forms will mask the form of the individual action potential and produce an interference pattern.
Jarabak (1957) indicates that muscle fibres are banded together to make motor units, each of which has its own blood and dual nerve supplies, and in order to have the myriad of complex movements associated with chewing and swallowing, rhythmic and smooth, it is necessary to have muscle activity precisely timed and synchronously carried out by some muscles which are contracting, by others which are relaxing, and by still others which are holding, guiding or poising the mandible. It is the integrated action of several muscles or muscle groups acting in unison through a precise timing of nerve impulses from the central nervous system which makes co-ordinated muscle activity possible.

Earlier Perry and Harris (1954) have pointed out that the neuro-muscular system provides all the power and dominates all the changes in the mechanical functions of the mouth. Hickey in 1957 feels that such integration of activity requires special overall centres which are found in the mesencephalic root of the fifth cranial nerve.

Jarabak (1957) claims that the reflex arc involved is as follows. Afferent impulses are sent by individual sensory nerves to the mesencephalic root of the trigeminal nerve. From here the messages are transferred to the motor nucleus of the trigeminal nerve which in turn sends motor impulses to the muscles causing them to contract in various degrees in response to the sensory stimuli from the proprioceptors. These are the simplest type of reflexes, commonly referred to as monosynaptic reflex arcs. It is believed these two neuron reflex arcs are almost wholly responsible for reflex mandibular posture.
At birth proprioceptors are found in the temporo-mandibular joints and in the muscles of mandibular posture. These record the local tension during both rest position and phasic movements of the mandible. The same neural mechanism which maintains mandibular posture in the infant before teeth erupt is called upon to maintain it in the adult when the teeth are lost.

Atwood (1953) claims that when occlusal contacts are removed not only are the mechanical stops removed but also the neuro-muscular stops, that is reflex receptors which stimulate reflex inhibition. It may be postulated that if some of the reflex inhibition is removed, the closing muscles may tend to shorten until sufficient other stops are called into play whether they be neuro-muscular or mechanical. Perhaps the contact of the tongue with the maxillae is such a neuro-muscular stop and in certain patients with large tongues, even a mechanical stop. Lips may also be a neuro-muscular stop.

Brill (1957) has also emphasised the afferent importance of the exteroceptors in the oral mucosa, including the tongue.

Pruzansky in 1952 has pointed out that afferent impulses are also introduced from peripheral stimuli from the labyrinth or vestibular apparatus, from the eyes as well as from proprioceptive nerve endings in muscles, tendons, and joints. These converge centrally and act to modify muscle movement.

Jarabak in 1957 has indicated that while teeth are present stimuli from periodontal proprioceptors are much more selective than those which arise from the more primitive proprioceptors of the joint and musculature. Because of this greater selectivity, periodontal stimuli take over dominance of many neuro-muscular reflexes which govern temporo-mandibular joint activity. It is assumed that the length
of time these periodontally dictated stimuli remain in the brain memory depends upon two factors:
(a) How long were the stimuli imposed and
(b) How far from normal was muscular activity when these stimuli were imposed.

Lamque, Perry and Crumain 1959 have also discussed this aspect. They consider that in the case of the vital periodontal membrane and its mesencephalic proprioceptive component, the attainment of the engram is recognised and its position reinforced by the firings of the periodontal membrane. After the loss of natural teeth and their accompanying proprioceptors and acknowledgement and reinforcement of a terminal intercuspal position is dependent upon muscle, mucosa, and joint health, as well as mechanical denture construction. With either one or all of these features varied from the original dentition a new pattern or engram must be established within the cortex and its acceptance will be dependent upon its effect upon health of all the structures concerned.

Levels of Neuro-Muscular Control

The neurological control of mandibular position, that is position both at rest and during function, can be controlled or interfered with at various levels, that is mastication in the human being when carried on as a learned habitual muscle pattern must also be partly controlled by proprioceptive impulses from the periodontal membrane of the teeth.

Hickey et al (1958) points out there are two general levels of nervous activity. One is the reflex level which is considered inert; the second is consciousness itself, and has its origin in nerve cells within the cortex. The cortex will utilise proprioceptive and tactile signals to recognise and localise, spatial relation and postural position. For the proper perception of these, the cortex must integrate all of the sensory information from the exteroceptive,
interoceptive and proprioceptive systems.

According to Windars, Corbin and Harrison, have shown that the mesencephalic nucleus is responsible for the reflex control of mastication. Therefore the muscles of mastication, they conclude, are not necessarily controlled by impulses arising from the conscious level of the brain, but rather from sensory impulses emanating from proprioceptive receptors found within the periodontal membranes of the teeth.

Atwood (1959) however, feels that there are five main levels of neuro-muscular control. At the periphery there is the simple reflex arc. This may be augmented or inhibited at the second, or spinal level. The cerebellum and mid-brain with their integrative functions constitute the third level. The fourth level is in the cerebral cortex, that is voluntary control level. The fifth level is the level of social or emotional factors and is contained in the association areas of the fore-brain.

**Electrical Potential Associated with Neuro-muscular Action**

Wright (1956), has indicated that when a nervous impulse is travelling in an efferent peripheral nerve, reaches the terminals of its fibre, it produces a characteristic response in the effector tissue. The question arises: what is happening at the junctional tissue between the nerve terminals and the effector tissue, that is at the motor end plate, the synapses in autonomic ganglia or the autonomic post-ganglionic end organs in smooth muscles and glands.

Two main views have been advanced.

1. The transmission process at nerve ends is essentially electrical when the spike potential reaches the nerve ends it directly sets up a localised catelectropotential; when the latter reaches a critical
magnitude it depolarises the surface membrane of the adjacent tissue and thus stimulates it. Such a catlectrotonus has been demonstrated at the motor end plate.

The alternative view is that a chemical intermediary, or chemical transmitter intervenes between the spike potential and the processes which stimulate the effector tissue. These transmitters have been demonstrated intervening between the spike potential and the end plate potential at motor end plates.

In either case there is a release of potential which is directly proportional to the functional activity of the muscle.

Electrical Activity in Muscle

The Shpuntoffs (1956) state that when a muscle contracts, mechanical energy, heat and electrical activity are produced. The electrical activity is minute and must be amplified $10^5$ to $10^2$ times for perception. The Shpuntoffs used an electron beam for recording the muscular electrical activity.

The recording of this electrical current is usually as a graph and is described by Moyers in 1950. If this recording is done on a moving surface the potentials are recorded as a wave, that is diphasic in character. The initial and main deflection is termed a spike. The continued contraction of a muscle is recorded as a series of spikes as different muscle units enter the contraction. As indication of the entrance of additional units into the contraction is seen as an increase in frequency or number of spikes per second. Simultaneous contraction of several motor units may produce a single spike which is of greater amplitude than that of a single motor unit. This phenomena results from summation. Most physiologists agree that the amplitude of the spikes is proportional to the strength of the contraction. It is also generally held that the increases in frequency occur with increasing effort.
The advantage of these electro-myographical recordings is that muscular function as a whole at the level of a single muscle can be studied. Moyer reports that work on cadavers suffers from two shortcomings:

(a) It is not always possible to deduce from sheer observation the action of a muscle.
(b) The very nature of dissecting, that is one muscle at a time, causes one to lose sight of the synergistic interplay of muscles around any joint.

Greenfield and Wykes (1956) report that it is impossible to decide whether or not a muscle is in fact participating in any particular movement, merely from consideration of its origin and insertion: Visual examination fails to reveal what proportion of motor units in a muscle is involved, the distribution of active units within a muscle, or the manner in which changing activity in one muscle is related to simultaneous contraction or relaxation of related muscles.

Electromyographic Analysis

For the electromyographic analysis the various authors have used various techniques. Some, such as Greenfield and Wykes (1956) used surface electrodes and others used needle electrodes.

The Shpuntoffs recorded the wave pictures on a cathode ray tube and others used a graphical recording method.

Some techniques caused erroneous conclusions as shown by Hickey et al. (1957) because electrical activity thought to be originating from certain muscles or areas may actually be overlapping electrical fields from other muscles. This overlapping electrical activity has been recorded with both needles and surface electrodes and appears to be more intense with surface electrodes.
These same authors feel that surface electrodes should be used for dental electromyography. They are not the best for clarity and freedom from ambiguity, but the co-axial needle technique is not altogether satisfactory since the activity recorded is necessarily that of one or at most a few motor units, which may not be representative of overall activity of the muscle. In the case of surface electrodes, potential in deep area, for example the external pterygoid, may be shielded from an ipsilateral temporal electrode by the remus of the mandible or a part of the maxilla. In such cases the use of single electrodes as references is fraught with much error. In fact the greater number of electrodes included in the reference combination, the more nearly valid are the tracings from an active electrode.

Electromyographic Analysis of Rest Position

The Shumptoms (1956) state that in rest position the number of motor units firing are practically negligible, and the patient may retain his jaw in rest position by maintaining a specific picture on the cathode ray tube while the regulation is made. When electrical silence was found in one muscle to indicate physiologic rest position our tests show that all other units of this mystatic unit were also electrically silent.

Lamie et al (1958) also felt that at rest position minimum activity was recorded. These authors considered this to be indicative of a healthy muscle pattern. Jarabek (1956) however, feels that at rest there may be some discernable activity of the temporal muscle. Since the task of elevating the mandible in forceful biting falls principally on this group of muscle fibres, it would seem logical to assume that here should lie the greatest number of motor units and from here the great neuro-muscular activity would be recorded. In such a normal patient, after any activity, the temporal muscles come to rest in a very short time.
He does, however, feel that physical rest position of the mandible is not always synonymous with electromyographic silence in the muscles, for the excessive distances through which the mandibular muscles contract during the phasic movements, in chewing, in speech and in swallowing, frequently influence rest behaviour of these muscles.

The electromyographic behaviour of rest in the temporal and digastric muscles of the subject with short dentures indicates the posterior and anterior fibres of the temporal muscle and to some degree the digastric muscle were in a hypertonic state after this spontaneous hyperactivity. When the movement was completed and the mandible returned to rest the temporal muscle returned to its former state of hyperactivity. From this we may conclude that when milled movements were initiated, hyperactivity which had been present at rest, ceased, only to return when voluntary movements were completed.

If dentures are being worn, which function with interocclusal distance which is too great, there will be found at rest position to be hyperactivity in the temporal and digastric muscles. This disappeared although not immediately, upon the insertion of dentures which function at the correct vertical dimension.

Jarabak feels this is due to the presence of a tremor or unsteady muscle response. This tremor will definitely preclude the establishment of physiologic rest position at a level which can be recorded electromyographically, however, if the cephalometric method of appraisal is used the physiological rest position will be deemed to be sufficiently accurate.

Lamie et al (1958) also found that when an open denture was placed, a great deal of electrical discharge was noted in the temporal muscles, and a small amount in the masseters. It is probable that this is a stretch reflex type firing of the various motor units.
initiated by the increased vertical dimension.

Similar discharges, not of such great magnitude, however, have been noted in rest position studies immediately after plaster has been introduced about the mucosa and teeth, to record mandibular position. This is as indicated in a thesis submitted by R.P. Mullen 1956.

Jarabak in 1957 feels that there is no practical technique for locating rest position electromyographically. Since slowly performed mandibular movements from 1 to 3 mm. on either side of rest position are difficult to identify by changes of action potentials. He feels that the use of the electromyograph for establishing maxillo-mandibular relation has practical limitations.

**Habitual Movements**

Pruzansky (1952) points out that intact muscle is extremely sensitive to the demands of external forces. Thus in describing a particular motion, care must be taken to qualify the motion in terms of (1) speed of motion (2) resistance to the motion (3) posture of the limits in relation to gravity during the motion.

Sven Carlsoe in 1952 feels that the primary muscle involved in habitual closing movement appears to be the temporal muscle. It also seems to be the mandibular postural muscle in habitual rest position.

Protrusion of the mandible leads to a decrease in the activity of the temporal muscle and activation of the masseter and medial pterygoid. Thus these latter two are considerably more active and the temporal muscle is notably less active in the cutting movement than in the habitual closing movement. During transverse rotation of the mandible towards habitual rest position, activity is demonstrated in all the subjects in the temporal and deep masseter portions.
Hoyers in 1950 points out that the internal pterygoid, masseter and temporal muscles all display high amplitude spiking during elevation. Brill in 1957 says in closing of the mouth there is an intensification of the contraction of the elevator muscles. This increased contraction is reciprocated with a corresponding gradual alteration of tonus of the opening muscles through a so-called reciprocal innervation of these muscles. If movement is performed with the mandible in its most retruded position, other groups of musculature come into play which serve the purpose of stabilising the condyle in their retruded positions. When the teeth come into contact the pattern of innervation is reversed to serve a contraction of the openers and an alteration in tonus of the closing muscles. These integrated actions of muscles are timed with great accuracy. Also in the case of right side activity there must be co-ordination with the action of the opposite side.

Hickey in 1957 reports the closing record shows that the increased activity is primarily observed in the masseter and temporal muscles, with the least activity from the external pterygoids. The authors believe that once teeth are lost closure always becomes random in nature. Greenfield and Wyke (1956) show that with the accompanying figure, on slow closure the almost simultaneous onset of discharge from both parts of the temporal and masseter muscles is seen in various recording combinations. The synchronous cessation of discharge in all leads should also be noted. In the upper four tracings of the figure, of the same movement in a patient with slight deviation of the jaw to the right during biting, it can be seen that firing develops less rapidly on the left in the posterior temporal and posterior masseter regions than in the anterior parts of these muscles. The appearance on the right side during the same movements are seen in the second set of four tracings while bilateral comparison of posterior temporal and posterior masseter activity is made on the third set of tracings.
(a) The anterior part of the temporal muscle shows the greatest number of active motor units during biting in habitual, retruded and ipsilateral molar occlusion. Fewer motor units are active in protruded occlusion. There are very few active motor units during incisor and contra lateral molar biting. In simple protrusion this part of the muscle is relaxed.

(b) The posterior part of the temporal muscle shows the greatest number of active motor units in habitual, retruded and ipsilateral molar biting. Some motor units are active in retrusion and ipsilateral deviation of the mandible. This part of the muscle is relaxed in incisor and protruded biting, and during protrusion and contra lateral deviation of the mandible.

(c) The lower anterior part of the masseter muscle shows the greatest number of active motor units in habitual, protrusive, contra lateral molar and incisor biting and during protrusion of the mandible without occlusion.

(d) The upper posterior part of the masseter shows the greatest number of active motor units in retrusive and ipsilateral molar biting.

Brill (1957) feels there must be some differentiation made between active and passive muscular movements. In the former the mandible is moved by the muscle and it is believed that at some juncture in dorsal displacement a certain pattern of innervation is being induced which causes an interruption of the muscular activity necessary for the continuation of movement. This happens in a split second just before the ligaments of the joint capsules will quite mechanically take up and prevent the movement, and by this mechanism the occurrence of pathological disorders in the nature of peritendinosis and the like, in and around the tempromandibular joint capsule is prevented. This protective action is reflex.
INTRODUCTION AND DEFINITION.

The importance of establishing correct centric relation in any field of restorative work cannot be over-emphasised. Centric relation is indeed, as Block (1953) pointed out, the heart of prosthetic procedure.

Furness in 1935 states it has been variously estimated that from 50 - 80% of the denture cases which must be reconstructed, are failures because the operator did not establish the centric jaw relation.

Simpson (1939) has stated that centric relation of the mandible to the maxilla is the key to all functional positions and relations. More recently Shore (1959) said: "The accurate use of any restoration, that is made on an instrument outside the mouth itself, will depend basically upon the accuracy of the centric relation wax bite."

Meyers in 1956 points out that there are two problems in centric relation, defining it and registering it. It is with the first of these problems that we are about to deal.

Robinson in 1951 has pointed out that some vagueness and ambiguity exists concerning the concept of centric relation. This is due, in the main, to a confusion of terms and a looseness of terminology used in the literature.

Kurth (1949) feels that centric relation consists of two separate and equally important components, the vertical and horizontal position of the mandible to the maxilla. I personally do not feel that this is correct. The use of the word "centric" in the title limits the relationship to that of two planes; that is the horizontal and the sagittal. It
would seem that the term centric relation refers to mandibular position in these planes only and the broader concept of maxillo-mandibular relationship involves the mandibular position in all planes: vertical, horizontal and sagittal. I do not, however, wish to imply that centric relation is not dependent upon vertical position of the mandible to the maxilla.

According to Sears (1960) the first pronouncement by the National Society of Denture Prosthetists in 1925 said: "The mandible is in a central relation to the maxilla when in its most retruded position from which the incisor point can make lateral movements." The same Society gave the following definition in 1930: "the mandible is in centric relation when the heads of the condyles are in the most retruded position from which the jaw can make lateral movements."

Finally in 1956 the Glossary of Prostodontic Terms reads: "Centric Relation — the most retruded relation of the mandible to the maxillae when the condyles are in the most posterior unstrained position in the glenoid fossa from which lateral movements can be made at any given degree of jaw separation."

Various authors working in different fields of dental research have based their concept of centric relation according to the structures or techniques with which they are working. That is, the problem has been viewed from the angle of occlusal integration, from a condylar position and from the point of view of muscular balance.

Robinson (1951) points out: "Centric position is not only the mandibular-maxillary relation where the teeth should occlude in the normal or good functioning situation, but also where the condyle of the mandible is in a balanced and unstrained position in the mandibular fossa. — This position exists when the antero-superior surface of the condyle is in close
approximation with the postero-inferior surface of the articular eminence."

As quoted by Sears, the Glossary of Prostodontic Terms in 1956 defines centric relation according to condylar position.

They were preceded in 1956 by Nagle who also determined centric relation according to condylar position and describes it as the relationship existing when the condyle has a physiologic relationship to the glenoid fossa. Kazis in 1954, however, has pointed out that this condylar balance is a result of proper muscular co-ordination.

Swenson in 1963 has also defined the centric relation according to condylar position indicating it is the most retruded unstrained position of the condyles in the glenoid fossa at a given degree of jaw opening. He emphasizes that the word "unstrained" refers to stress in a superior-inferior direction not in an antero-posterior direction.

Lucia (1953) bases his definition on muscular action; "For all practical purposes centric relation is the most retruded position into which the muscles of mastication can pull the mandible." This I feel, is taking into consideration the research of Posselt who found that it was only a very small percentage of patients that are unable to retrude their mandible horizontally past "centric relation."

Moyers (1956) working on neuromuscular research defines centric relation as the position of the mandible as determined by the neuromuscular reflex, first learned for controlling the mandibular position when the primary teeth were in occlusion.

Another group of workers base their definitions on the axial relations of the condyle.

Granger (1952) feels that the usual definition of centric relation as the most retruded position of the mandible from which lateral motion
can take place, is inadequate, because it fails to consider the axial relationship which is the only reason for the importance of centric relation. He claims that any given point on the surface of the head of the condyle does not remain in fixed relation to the meniscus. In every position of the condyle, however, the hinge axis does remain in the same relation to the meniscus. He defines centric relation thus: "There is only one position in which the hinge axis is constant to both mandible and maxilla. Since the hinge axis is established at the point in each condyle where the horizontal axis and centre of rotation meet, the centre of rotation in this position is also constant to both the mandible and the maxilla. This is centric relation."

Shore (1959) is of the same school of thought and defines centric relation as a skull to mandible relationship. The mandible is in centric relation when the heads of the condyle exhibit pure rotary motion around the hinge axis while the mandible traverses an arc before translatory movement of the head of the condyle occurs.

These theories while they may be academically correct, do not assist the operating prosthetist in establishing the centric relation of the patient; as Kingery (1959) points out there are many definitions that are basically correct, but do they mean anything to the operator who is about to locate the position. Thus the definition "centric relation is the position of the mandible as determined by the neuromuscular reflex first learned for controlling the mandibular position when the primary teeth are in occlusion" is correct, but not clinically informative.

All of the factors, that is neuromuscular, condylar and occlusal, which influence the position of the mandible at centric relation, must be considered, but not necessarily have to be included in the definition.

As pointed out by Sears an acceptable definition of centric relation is with reference to the maxillae. "The mandible is in centric
relation when in a position from which any horizontal translation produces an eccentric relation. This he feels infers physiological, muscular balance and unstrained position of the condyles.
CENTRIC OCCLUSION:

As Kingery (1959) points out centric occlusion and centric relation are so inter-related that the two go hand in hand in any discussion. Apparently some confusion exists between the two entities and in the literature one often finds the terms being used loosely and incorrectly.

Right in 1936 has pointed out that the wording "occlusal" automatically refers to "tooth", so that centric occlusion is a tooth position as contrasted with centric relation which is a jaw position.

The tooth position referred to by centric occlusion is one of maximum contact. Furnas in 1938 had defined it as that relation of the mandible to the maxillae in which the functioning planes of the teeth are in maximum contact.

J Thompson indicating that this position is a tooth position said in 1954 that centric occlusion is not established until teeth erupt into occlusion; it varies as the occlusion is altered and is lost when the teeth are extracted.

Standard and Lepley (1955) define centric occlusion as a tooth contact position in which the head of the condyle is balanced in the glenoid fossa within the range of vertical displacement. This is the position of the mandible such that the teeth are in contact. In this position the head of the condyle is never in the deepest part of the fossa, but is opposite the posterior slope of the articular tubercle. This seemingly labial equilibrium is maintained by the interlocking cusps of the occluding teeth which prevent further movement of the mandible upward and backward.

Kazis in 1954 has pointed out that centric occlusion is the midway point in the centric cycle, which consists of a movement of the mandible from physiologic rest position to the tooth contact position and the
return of the mandible to the physiologic rest position. Therefore, he defines centric occlusion as the occlusal inter-relationship of the mandible to the maxillary teeth when the mandible is in its utmost state of closure.

Shanahan in 1956 has also pointed out that centric occlusion is the principal occlusal position and is the terminal position for both lateral excursions and for the Boswell cycles.

In the dentulous case we have a definite fixed position, one that is easily identified by both patient and operator and one to which the patient can return repeatedly at will.

This position is not necessarily identical with the habitual biting position, but may become so after a few years of abrasion. Thus we find centric relation to be a fixed positional relation, while centric occlusion becomes a constantly changing relation influenced by age, abrasion, extraction and habit.

The edentulous patient, however, has no centric occlusion or occlusal position until dentures are constructed.

Anthony in 1942 indicated that centric occlusion of complete dentures is dependant upon centric jaw relation, and Kingery (1952) feels that centric occlusion should be established to co-incide with the recorded centric relation position.

Kazis (1954) points out a state of harmony must exist between the centric relation of the mandible and the centric occlusion of the teeth. Deviations from this functional pattern will result in disharmonious relationships of the support musculature. An abnormal path of closure will be in operation as a result of this malfunctioning relationship.

Boos (1959) has also indicated that centric occlusion is established in conjunction with centric jaw relation. The objectives are:
1. An occlusion in harmony with the musculature and the temporo-mandibular joint function.

2. A centric occlusion to which the mandible is directed in mastication.

3. A centric occlusion providing the proper relation in swallowing and in closing from physiologic rest position.