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Aural symptoms in relation to the temporomandibular joint.

By

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That the occlusion of the teeth is intimately related to the physiology of the mandibular joint is now, one may venture to say, a well-established fact. It is recognized moreover that anomalies of the masticatory organ may give rise to arthrosis of the joint.

In 1929 VON STAPELMOHR pointed out that the physiologic joint mobility is dependent on the normal function of the dental organ and that malocclusion must consequently involve some changes in the mutual relation between the joint surfaces and the loading of the joint itself. BOMAN in a number of articles — and most recently in his dissertation — has further stressed this point. In an anatomic investigation, STEINHARDT has resolved the function of the mandibular joint in relation to the occlusion, and GOODFRIEND has performed similar studies. The Scandinavian literature contains papers by several odontologists who have dealt with this subject; among them THORLEIF, LINDBLOM, BRANDRUP-WOGNSEN, POSSELT, EKENSTEN and SUNDBERG. It is, however, chiefly in the American literature that one finds in recent years the greatest interest devoted to this problem.

Our knowledge of the symptomatology of arthrosis of the mandibular joint has gradually expanded and has become the subject of wide discussion. To the local symptoms in the form of crepitation, snapping, inhibited movement and pain in the joint itself have been added symptoms of a more peripheral nature. COSTEN has summarized these in his "syndrome of the temporo-

mandibular joint". He gives the following: "Pains in and about the ear, giddines, tinnitus, some degree of impairment of hearing, herpes in the external auditory canal, increased formation of wax, further symptoms in the form of pain — as in sinusitis, neuralgia of the temples, the crown of the head and the nape of the neck; glossodynia and disturbances of the taste and salivary secretion."

The etiology of these symptoms attributes them to mal-occlusion accompanied by lowering of the natural dentition and a consequent backward and upward movement of the condyles which would traumatize the organs in question. During the last few years the anatomy, physiology and pathology of the mandibular joint have been described in detail in the literature, so that it will suffice here to refer the reader to the relevant papers: for example GOODFRIEND, STEINHARDT, THOURÉN and BRANDRUP-WOGNSEN.

Of particular interest in this connection is the anatomy of the posterior limitation of the mandibular joint and its adjacent organs. The dorsal limitation of the glenoid fossa is constituted by the post-glenoid process. In some mammals, HUMPHREY asserts, this process is a prominent outgrowth which descends behind the condyle of the mandibular joint and prevents its backward displacement during mastication. The shape of the process varies in man and the variability in the glenoid fossa is, of course, related to the size of the two processes bounding it — the post-glenoid process and the tuberculum articulare. In some cases the post-glenoid process is strongly developed while in others it is absent altogether. In the bed of the fossa immediately in front of the post-glenoid process is attached the posterior limit of the joint capsule. The bone in the bed of the fossa is thin; frequently there is no more than a thin layer separating the fossa from the dura beneath. The glenoid fossa is divided into two sections — the anterior-ventral — consisting of the pars squamosa —, and the posterior-dorsal — of the pars tympanica. Between these bones is a fissure system; a plate from the pars petrosa is introduced to form two fissures — the petrosquamous and the petrotympanic fissure. The fissure system is practically parallel to the head of the condyle when the teeth are in the position of centric occlusion. The pars tympanica continues dorsally to the postglenoid process and forms the anterior wall of the external and middle ear. Between the joint capsule and the pars tympanica there is connective tissue — rich in vessels and nerves — and also part of the

parotid gland. Through this fissure system there is a communication to inner organs. The chorda tympani leaves the canal for the facial nerve through the canal for chorda tympani. The nerve passes in the middle ear between the malleus and the incus and on its exit from the middle ear passes in the petrotympanic fissure. It leaves this at the angular spine under which it passes, thereafter joining the lingual nerve. From the otic ganglion the chorda tympani receives sensory fibres to the tongue and the salivary glands in the floor of the mouth.

There is in this region a rich vascular supply to the mandibular joint, the retro-mandibular tissues and the middle ear. When the external carotid artery has given off the superficial temporal artery the vessel turns horizontally and leads medially along the lower part of the neck on the condyle, forming the pars mandibularis of the internal maxillary artery. This gives origin to the following branches:

(1) the deep auricular artery which passes in the retro-mandibular connective tissue, partly supplying the joint and tympanic bone and sending further vessels through the petrotympanic fissure to the middle ear;

(2) the anterior tympanic artery which supplies parts of the joint and passes through the petrotympanic fissure to the middle ear.

(3) the middle meningeal artery which passes through the foramen spinosum and branches into the dura mater. This vessel also gives off branches through the petrosquamous fissure to the middle ear, where it forms the branch superior tympanic artery.

In this connection attention should be drawn to the anastomosis of these vessels with the other vessels of the middle ear. These are:

(1) from the posterior auricular artery, the stylomastoid artery, which enters the stylomastoid foramen and passes along the canal for facial nerve. Through the canal for chorda tympani the vessel gives off the posterior tympanic artery to the middle ear.

(2) from the ascending pharyngeal artery, the inferior tympanic artery branches off to the eustachian tube and the middle ear.

The anastomosis system thus formed supplied the middle ear and promontory, suggesting the existence of an anastomosis with the vessels of the capsule of the labyrinth.

The ligamentum mallei ant. proceeds from the angular spine of the sphenoid bone passing through the petrotympanic fissure.

It is attached to the collum mallei. The ligament is of great importance to the hearing mechanism, as together with ligamentum mallei post. it transmits the vibrations of the ear-drum to the malleus.

The auriculo-temporal nerve, which is a branch of the mandibular nerve, at first follows the middle meningeal artery; it then turns medially along the neck of the condyle, passing round this to appear over the zygomatic arch in the temporal region. It then follows the superior branches of the temporal artery, leading over the temple, the nape of the neck and the crown of the head. The nerve gives off secretory fibres, originating from the otic ganglion, to the parotic gland.

As may be seen from the above, the otic ganglion is intimately connected with this region. It is also connected with the sympathic through the meningeal plexus, and then with the glossopharyngeal nerve through the small superficial petrosal nerve. The otic ganglion is also responsible for the innervation of the musculus tensor tympani, which together with the musculus stapedius tenses the auditory bones.

Another organ of interest in this connection is the eustachian tube. When this has left the petrous bone it passes with the musculus levator and tensor veli palatini medially to the upper third of the internal pterygoid muscle.

In the case of a sunken bite it has been thought that the condyle is dislocated dorsally, thus, directly or indirectly, giving rise to the above-mentioned peripheral symptoms through pressure.

Attention will now be directed to ear-symptoms in connection with arthrosis of the mandibular joint. It is chiefly in the American literature that this problem has been discussed. The genesis varies and is apparently obscure.

In the 1920s MONSON and WRIGHT described cases of improvement in hearing through restoration of the normal vertical dimension. The etiological factors adduced are that a dorsally dislocated condyle might, by exerting pressure upon the tympanic bone, constrict the external auditory canal and prevent the transmission of sound to the middle ear. WRIGHT states that when the condyle is displaced backward and upward in cases of sunken bite not only is there resorption in the mandibular joint itself but the regions in the vicinity may also be affected. Resorption of that part of the tympanic bone that forms the wall of the external auditory canal might lead to a complete occlusion of this tube by

constriction. This process, together with continuous pressure upon and irritation of the tympanic bone to which the tympanum is attached, and, moreover, pressure upon the anterior tympanic artery and the chorda tympani, seem to give rise to partial or total loss av hearing. This condition he calls *traumatic deafness*. The fact, he says, that many people keep their mouths open when listening would suggest that the hearing is improved if the pressure on the condyle is removed. He also found from examinations of crania that perforation of the tympanic bone occurs more frequently in edentulous skulls where occlusal irregularities exist than in normal skulls.

COSTEN, BLOCK and HARRIS find a cause of ear symptoms in the fact that in cases of sunken bite a surplus of muscle tissue of the int. pterygoid muscle becomes bunched up and may exert pressure upon the soft part of the eustachian tube which it thus occludes.

For the same reason the function of the tensor veli palatini muscle in opening the tube on swallowing may be disturbed. This could interfere with the regular ventilation of the middle ear, giving rise to catarrh of this organ with consequent disturbances of the hearing, tinnitus and giddiness. BLOCK has further pointed out that the deepest parts of the fossa are very thin, and that the dura, which is very rich in nerves, might be affected traumatically.

GOODFRIEND draws attention to reflex effects from the auriculotemporal nerve and chorda tympani. The tensor tympani and stapedius muscles derive their innervation from the trigeminus and facialis, respectively, via the otic ganglion.

BLEICHER refers to the significance of the petrotympanic fissure. It is through this, he says, that the chorda tympani and the anterior tympanic artery pass. The latter supplies two-thirds of the ear-drum. He points out that sensory nerve fibres have been observed within the walls of the blood vessels, and that it is therefore reasonable to assume that such fibres occur in the tympanic artery. The irritation of these fibres alone may be sufficient to bring about disturbances in hearing. Moreover the chorda tympani contains both motor and sensory fibres, and an irritation of this nerve by the condyle may give rise to disturbances in the middle ear. The result would be the same whether this precipitating factor were a direct effect or reflex effect.

SMITH points out that in cases of sunken bite the tongue is displaced backwards, forcing the surrounding tissues into abnormal positions. The soft palate would exert pressure upon the pharyngeal opening of the eustachian tube. A reduction of the lumen of the tube and even inflammatory conditions might result. It is undoubtedly an exaggeration to maintain that so-called "middle-age deafness" should rather be termed "deafness of the sunken bite"; on the other hand, to regret, as one author has, that "some men have gone so far as to assert that they can relieve deafness by opening the bite", is too sceptic an attitude. CHAPIRO and TRUEX and SICHER have advanced criticisms of the observations so far made. They consider it open to question that in the case of a dorsal dislocation the condyle might constrict the external auditory canal. It is pointed out that for anatomical reasons this movement would be much too extreme, and attention is also drawn to the protection of the retromandibular tissues (the postglenoid process, the capsule, the connective tissue between this and the tympanic bone and part of the parotid gland). The perforations that have been observed in the tympanic bone are explained more as anatomical. 20 % of all persons have small foramina in the tympanic bone resulting from arrested calcification. The traumatic effect of the condyle on the bed of the fossa is also considered problematical. CHAPIRO and TRUEX point out that in their investigations they have found no case of perforation in the fossa. It has always been possible to observe a layer of extra dural bone, even though it may be thin.

CHAPIRO and TRUEX are inclined to ascribe some importance to nervous influence. They point out the possibility of a reflex effect upon the tensor tympani and stapedius muscles. SICHER, however, considers that the chorda tympani lies so well protected in the petrotympanic fissure that a condylar effect upon the nerve seems unlikely. Neither, he says, does it contain fibres that can give rise to pain. The chorda tympani is composed of fibres for the innervation of the anterior part of the tongue and of pre-ganglionic fibres of a secretory nature for the salivary glands of the lower jaw. The auriculo-temporal nerve, SICHER adds, passes in such a position that no effect upon the nerve by the head of the condyle can be considered likely.

In this connection, however, one is struck by the fact that in many cases the neck of the condyle shows a dorsal bulge and, moreover, that the retromandibular space may show great varia-

tions. It is therefore quite possible that the nerve fibres lying between the tympanic bone and the condyle may, under certain conditions, be exposed to trauma in cases of a considerable dorsal dislocation of the condyle. There are also many reports in the literature of favourably treated cases in this "pain group". The present writer has himself observed this.

As regards the tubal occlusion, CHAPIRO and TRUEX admit that an anatomic connection between the tube and the adjacent muscles may very well exist, but they think it open to doubt whether a sufficiently great pressure may arise to effect complete tubal occlusion. SICHER points out here that the muscle tissue in the interior pterygoid occupies two-thirds of the middle length. The upper part that lies in the vicinity of the tube contains only tendinous tissues and does not change in volume during contraction.

It may also be pointed out in this connection that it should in this case be possible to differentiate the diagnosis, as catarrhal changes in the middle ear may certainly be observed otoscopically. If the air-passage to the middle ear is shut off the remaining air in the middle ear is rendered thinner by resorption. A negative pressure forms and the ear-drum is forced inward, resulting in disturbance of the normal function of the ear-drum. The otoscopic picture is changed, a diagnostically important point; catarrh resulting from these disturbances in ventilation is described as characteristic. In those cases treated by the present author the otoscopic picture has not, according to reports from the otologist, given any indication of disturbances in the middle ear. The ear-drum has been entirely normal.

SICHER also emphasizes that the impairment of hearing reported in this connection seems more to indicate disturbances in the inner ear, but excludes the possibility that the inner ear may have any connection with the displacement of the condyle.

As regards the above-mentioned vascular supply to the middle ear, CHAPIRO and TRUEX are of the opinion that a traumatic effect may quite possibly be exercised by the condyle upon anterior tympanic artery and branches of middle meningeal artery, but with frequent anastomosis with other vessels in the middle ear they consider it doubtful whether trauma of a single vessel can bring about such nutritional disturbances in that region as to cause any considerable loss of hearing. The solution of this problem will they feel, be revealed by continued research into the etiology of otosclerosis and arteriosclerosis.

Views on the etiology of ear symptoms in connection with arthrosis of the mandibular joint are thus many and varied. It has been categorically stated that these symptoms might derive from the external or the middle ear. The inner ear has been entirely left out of account in the discussion. CHAPIRO and TRUEX, who, like the majority of other writers, emphasize the importance of a detailed anatomical study, have asserted that the membranous cochlea and the auditory nerve are in no respect dependent upon the arteries surrounding the condyle for their blood supply. They have mainly based their conclusions upon the investigations of the supply of the inner ear carried out by SIEBENMANN at the end of the last century, where it is concluded that the membranous tissues of the inner ear derive their blood supply exclusively from internal auditory artery.

The problems with which the present investigation have been concerted are: Is it correct to assume that the internal auditory artery alone supplies the membranous organs of the inner ear? If there are other sources of supply, in what relation do these stand to the mandibular joint?

To consider now the embryological development of the inner ear, attention has already been drawn by RAUBER to "Die Eigentümlichkeiten des häutigen Labyrintes welche an seine Abkunft von der äusseren Körperhülle erinnern". More recent embryological research also shows that the epithelial elements of the labyrinth are formed from labyrinth bladder or sac developed from the ectoderm. This sac is gradually closed off and is filled with endolymph. The sac sinks towards the rhombencephalon and receives innervation from the acoustic nerve and nutrition from the internal auditory artery. Before this connection has taken place, however, the labyrinth sac with its endolymph must obtain its supply from the internal carotid artery through the stapedia artery. This latter vessel is described as passing through the place where later the fissure system in the glenoid fossa is formed. The stapedia artery divides into the ramus supraorbitalis, maxillaris and mandibularis. Between the ramus mandibularis and the external carotid artery an anastomosis is gradually formed. The connection between the internal carotid artery and the stapedia artery atrophies. The supply, formerly effected from the internal carotid artery is now taken over by the external carotid artery through the internal maxillar artery.

As a consequence, the present writer considered it worth while to investigate the existence of a vascular supply to the inner ear from the internal maxillary artery which might contribute to the supply of certain of the tissues of the inner ear — and especially the endolymph-producing cells in the stria vascularis, as these must of course already exist before the labyrinth sac has communicated with the internal auditory artery. If such a supply does exist, it ought, from the above argument, to pass through the fissure system in the glenoid fossa. It would then be possible in cases of sunken bite for the condyle to exercise a traumatic effect upon this supply.

An investigation was pursued the object of which was to follow the supply of the middle ear, and possibly the inner ear, from the internal maxillary artery with the help of contrast injections in this vessel. It was hoped to reveal the anastomotic conditions in the middle ear and to follow the supply of the promontory and possibly also the inner ear by this anastomotic system. It was also intended to bring to light any existing direct supply to the inner ear from the internal maxillary artery.

Contrast injections were performed on cadavers of full-time foetuses and of adults, using a cinnabar gelatine solution, prepared according to the following prescription: 1 part gelatine, 2.5 parts cinnabar, 4 parts water, heated on water bath and filtered through flannel. A screw injection syringe was used.

The external carotid artery was exposed, and the occipital, the auricular posterior and the superficial temporal arteries were ligatured. The injection cannula was introduced in the case of the embryonic preparations about a centimetre above the origin of the external carotid artery from the communis, or higher if conditions allowed. On cadavers of older subjects it was of course possible to introduce the cannula directly into the internal maxillary artery. The preparations were warmed well and the injection performed under gentle pressure. The amount of injected solution was tested from case to case, to ensure that it reached the capillaries. The capillaries of the mucous membrane of the lip were observed, and when they had been filled the injection was discontinued. The preparations were then fixed in formalin solution for 24 hours. In some cases it was possible to observe that the injection material filled the posterior auricular artery to the point of ligature. In this way the anastomotic relationship between the stylomastoid and the tympanic arteries was clarified. When

the glenoid fossa had been exposed it was possible to observe that this was well supplied. Along the fissure system in the glenoid fossa streaks of contrast running into the tympanic bone and the fissure system were observed. After exposure of the middle ear it was possible to follow the injection over the paries jugularis and paries tegmentalis. Further, in connection with these a fine-meshed system filled with contrast medium extended over the promontory. The temporal bone was then sawn out. On the medial side one could see that the middle meningeal artery, after passing the foramen spinosum, was partly distributed over portions of bone corresponding to the glenoid fossa.

On all the foetal preparations these observations were well marked. In one of these cases one could distinguish a vessel with contrast medium passing through the porus acusticus int. The injection had evidently here been complete. From this one may venture to assume that an anastomosis between vessels from the internal maxillary artery and the internal auditory artery may exist, at least at an early age. In a preparation of a 60-year-old man, on the other hand, no anastomosis was found between the internal maxillary artery and the other vessels of the middle ear. Thus stylomastoid artery was not filled with contrast medium. On the promontory, however, it was possible to observe a clear supply even if it was not so marked as in the case of the foetus. As a supply to the inner ear might also have existed in this latter adult case, despite the above-mentioned observation, this preparation was made the object of special examination during which the radiograph and microscopic sections shown below were made.

The continuous phases in the course of the work have been photographed both in colour and in black and white with various enlargements. Furthermore, stereoscopic radiographs have in a number of cases been taken. Some of the preparations were then treated for transparency by the method described by Dr. W. SPALTEHOLZ. The preparations were so transparent that it was possible in parts to follow the ramifications of the contrast medium into the inner ear. Stereoscopic photo-micrographs were taken, and it was possible to observe ramifications from the promontory into the inner ear. It seemed as if anastomotic bunchings extend over the promontory and then sent ramifications to the inner ear. In some preparations the petrosal bone was sawn out and radiographed, and then cut into sections for microscopic examinations.

It was possible to follow the contrast into the inner ear in the radiographed and transparent preparations and in the sections. The injection was observable in the capsule of the labyrinth and in part in the cochlear canal.

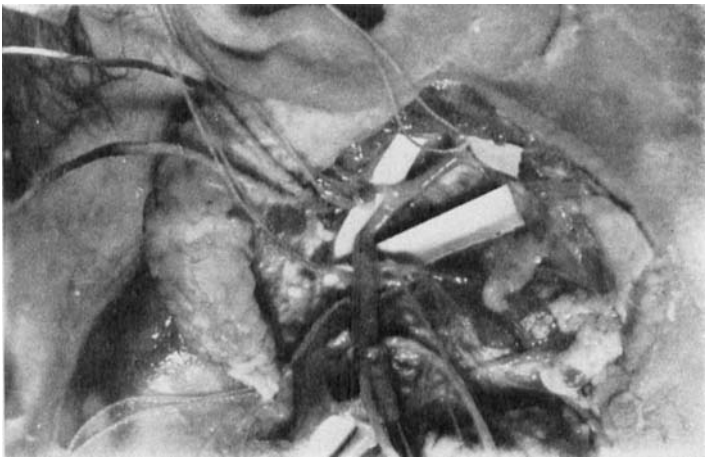
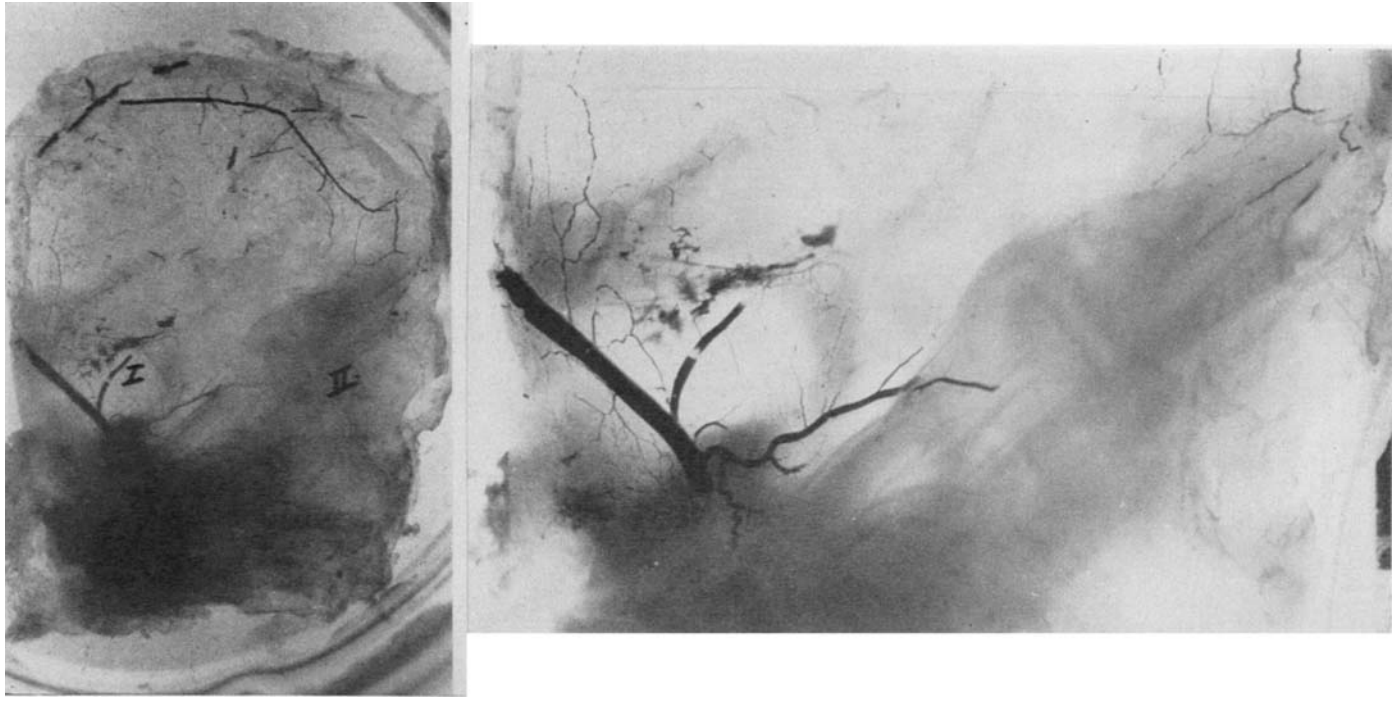


Fig. 1. Dissection of the ext. carotid artery with the injection cannula inserted. Excluded: the occipital, post. auricular and superf. temporal arteries.



A B

Fig. 2. Transparency preparation of the temporal bone after injection.
A. I. glenoid fossa. II. contour of the innner ear.
B. An enlargement of A.

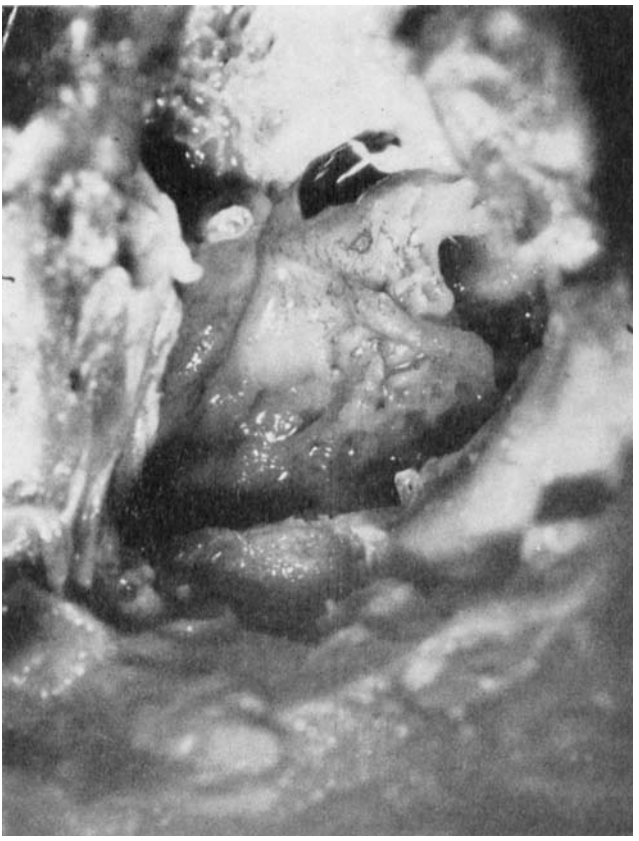


Fig. 3. The promontory after injection into the int. maxillary artery. Observe the injection striae scattered over the promontory.

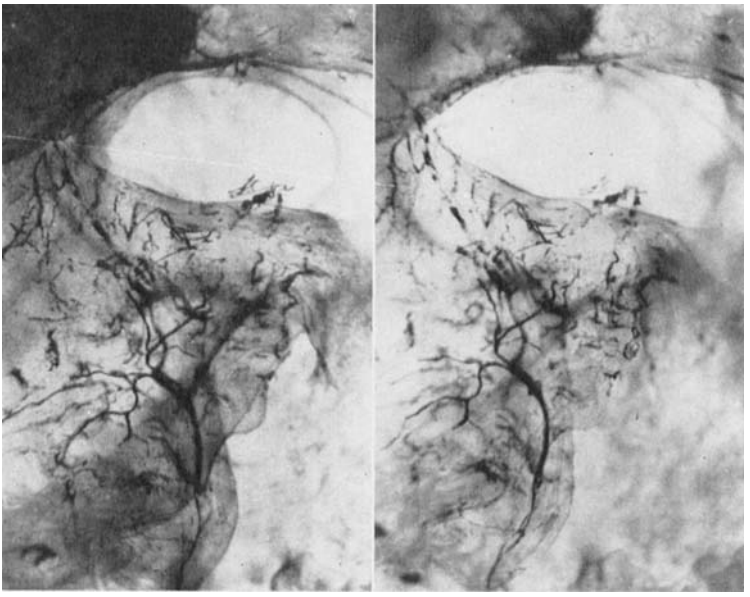


Fig. 4 (a). Stereoscopic photo-micrographs of transparency preparations of the inner ear. The aggregations of the anastomoses on the promotory are seen to pass to the inner ear.

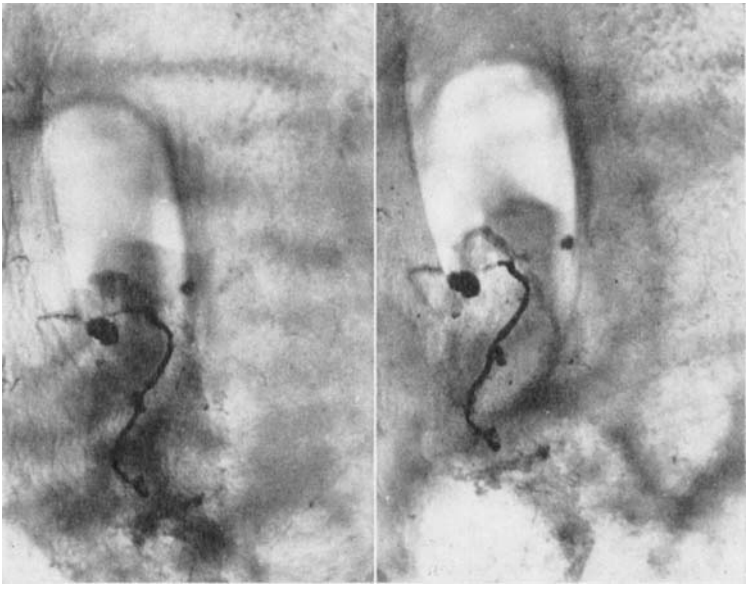


Fig 4 (b). As above but taken from the internal auditory meatus, through which passes an injection stria.

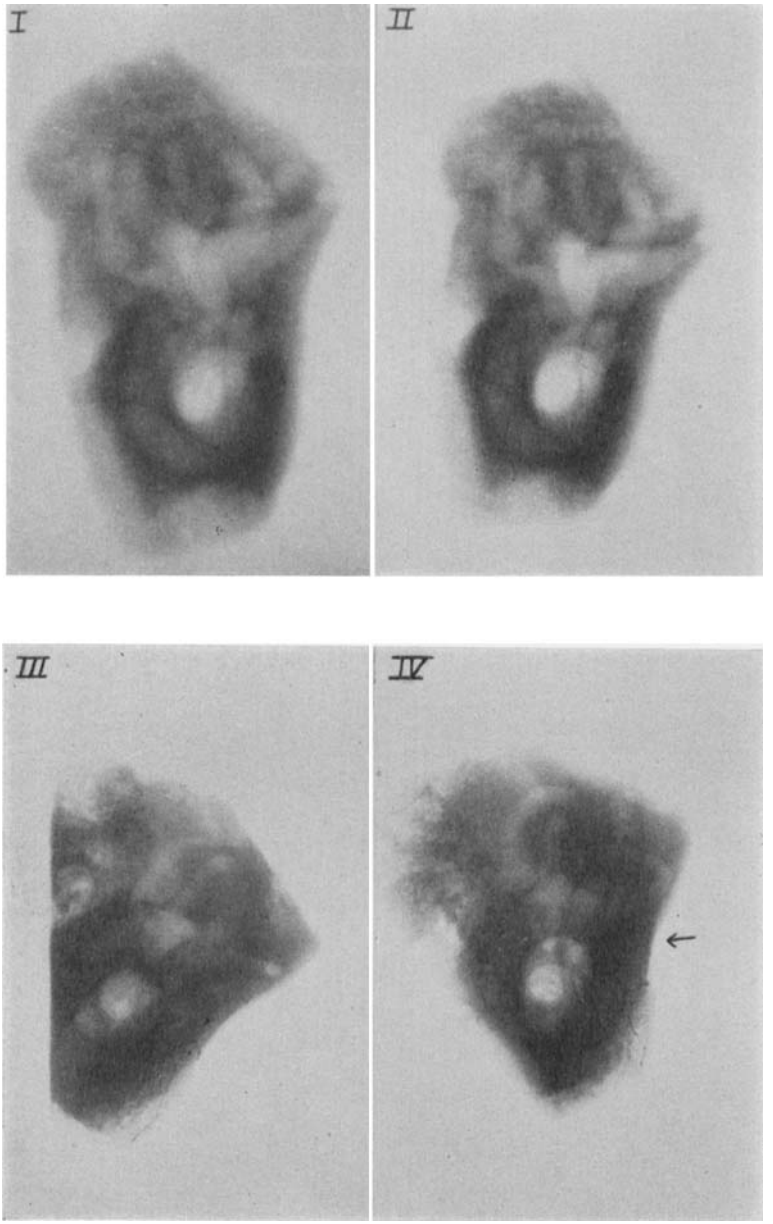


Fig. 5. The petrous part sawn from both fœtuses and cadavers in radiographs from opposite sides. Note injection passing into the cochlea. In IV observe the leak from the injection in the cochlea.

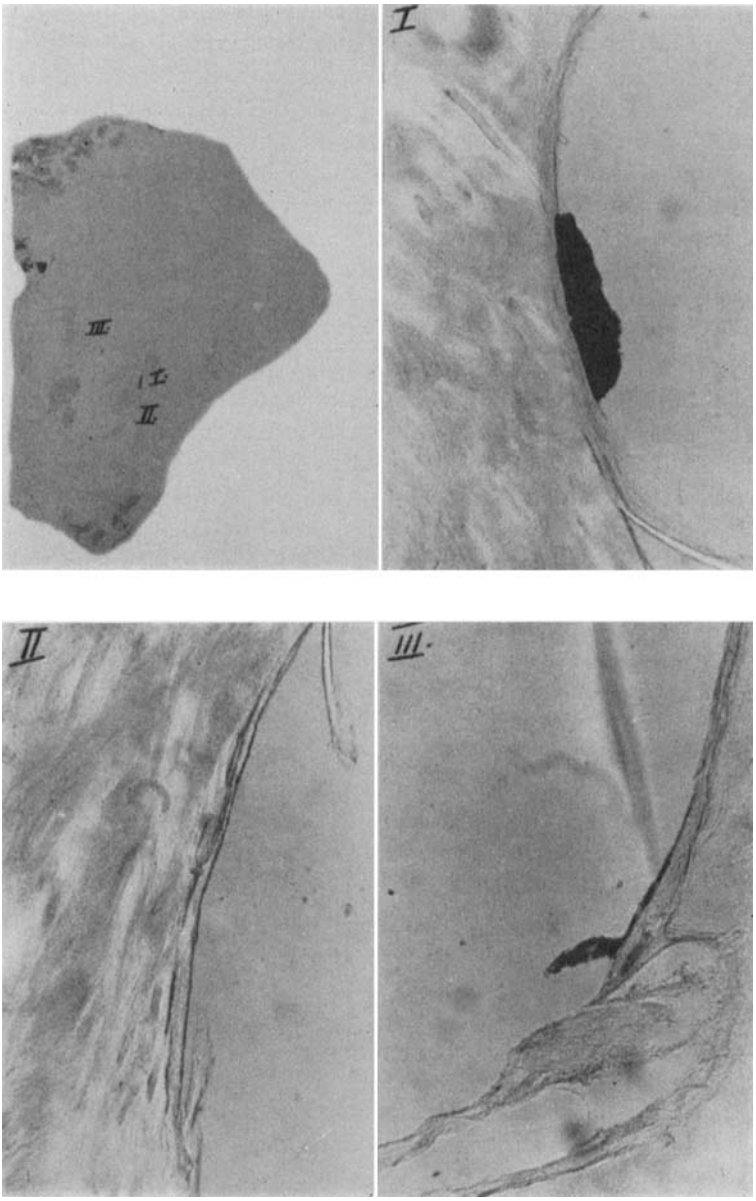


Fig. 6. A central section from preparation of the inner ear of the cadaver after injection into the int. maxillary artery. Note the injection in the cochlea. a) general aspect; note the same leak as in the radiograph I, II and III are enlargements.

A vascular supply that has not earlier been taken into consideration seems to exist from the internal maxillary artery to the inner ear and to pass through the fissure system in the glenoid fossa. There is embryological support for this assertion, which is also indicated by these original investigations. That the condyle may in certain cases disturb this supply also seems likely.

Reports of symptoms in the aural region in connection with arthrosis of the mandibular joint have latterly become rather numerous. Reference may be made to WRIGHT, COSTEN, BLOCK, HARRIS, BLEICHER, GOODFRIEND et al. The cases treated by the author, most of which were referred to by Dr. A. HALL, the Head of the Ear, Nose and Throat Department at the Norrköping Hospital, are admittedly as yet not so numerous. They do, however, seem to indicate a connection between arthrosis of the mandibular joint and aural symptoms.

That this problem has been the subject of differing views and much discussion, especially between otologists and odontologists, is probably to be explained by the fact that it has not been possible to find acceptable etiological factors. Aural symptoms with normal conditions in the outer and middle ear would appear of course to indicate disturbances in the inner ear. These original investigations have shown a connection between the mandibular joint and the inner ear and may consequently throw some light upon the etiology.

Further etiological conclusions fall outside the scope of the author's jugdment. The problem is this: if the nutrition to the capsule of the labyrinth and certain membranous organs in the cochlear canal is disturbed, can such disturbances give rise to the symptoms that have been described in connection with arthrosis of the mandibular joint such as tinnitus, impaired hearing and giddiness?

Summary.

A description is given of the posterior limits of the mandibular joint and the surrounding structures. There follows a discussion of those etiological factors given in the literature that bear on the peripheral symptoms in arthrosis of the joint — and especially symptoms of the ear.

As a contribution to this etiology an anatomic investigation has been performed, the results of which show that a hitherto unrepor-

ted vascular supply exists from the internal maxillary artery to the inner ear, passing through the fissure system in the glenoid fossa. Support is lent to this assertion by the presence of an embryonic basis.

Zusammenfassung.

In dieser Schrift beschreibt der Verfasser teils die Anatomie von der hinteren Begrenzung des Kiefergelenkes und dieser naheliegenden Organ, teils die Diskussion in der Literatur von den etiologischen Faktoren der peripherischen Symptome bei Kiefergelenksarthros, insbesondere der Ohrensymptome.

Als ein Beitrag zu dieser Etiologie wird eine anatomische Untersuchung beschrieben, welche weist dass eine früher nicht erkannte Blutversorgung von der Arteria maxillaris interna nach dem inneren Ohr besteht. Der Verfasser gibt an dass dieser Blutbahn durch dem Fissursystem in die Fossa glenoidalis passiert. Als Stütze für diese Thesis wird auch auf eine embryonische Unterlage hingewiesen.

Résumé.

L'article commence avec une description des limites postérieures et des structures contiguës de l'articulation temporo-maxillaire. L'auteur discute les facteurs étiologiques, donnés dans la littérature, qui concernent les symptomes périphériques d'arthrose de l'articulation, en particulier les symptomes de l'oreille.

Comme une contribution à cette étiologie l'auteur a entrepris l'examination d'un système vasculaire pas encore rapporté dans la littérature. Ce système aurait son origine de l'artère maxillaire interne à l'oreille intériure, passant par le système de scissure dans la cavité glenoïde du temporal. L'existence d'une fondation embryonique sert à soutenir cette thèse.

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