PARTIAL PARALYSIS OF THE FACIAL NERVE IN CHRONIC SUPPURATIVE OTITIS MEDIA

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The otologists's interest in facial paralysis is centered in his need of being able to diagnose the site and nature of the lesion causing paralysis. In order to be able to make such a diagnosis the otologist must know the origin, course, branches, function and nerve connections of the facial nerve. In addition, he must be familiar with pathologic processes which may affect the nerve as it passes through the temporal bone.

Partial paralysis of the facial nerve invariably has been looked on as being caused by an intracranial lesion which involves the nuclei. When such paralysis occurs in the absence of chronic suppurative otitis media, there is little doubt about the situation of the lesion. But some references have been found in the literature to suggest that when paralysis of the facial nerve occurs in the presence of chronic suppurative otitis media, the lesion involves a certain portion of the nerve trunk within the temporal bone.

That facial paralysis has interested physicians and surgeons for a long time is evidenced by the fact that many reports concerning the facial nerve has accumulated. Histologists and neuroanatomists have contributed the results of their studies and research work so that it may be said that the knowledge of fundamentals is established. The surgeon's interest has been mainly to devise some surgical method to overcome facial paralysis so that disfigurement of the face may be relieve. Needless to say, disfigurement of facial expression caused by paralysis of the facial nerve has far reaching personal, social and economic implications. Therefore, any method of repair that will cause a return of function to the facial muscles is much worth while.

It would seem to be desirble that procedures be instituted in a given case before facial paralysis has become permanent, or before complete reaction of degeneration in the nerve fibers has taken place. Two types of surgical methods of repair have been devised. Much impetus recently has been given to repair of the nerve within the confines of the temporal bone, the usual site of injury in paralysis occurring before and after operations for disease of the middle ear. This method, for obvious reasons, seems entirely rational. Another method by which good results are achieved is utilization of proximal end of one of the adjacent cranial nerves, for example, joining of the spinal accessory or hypoglossal nerve to the distal nerve trunk of the facial nerve as it emerges from the stylomastoid foramen.

It does not fall within the scope of this report to mention all the observations that have led up to the attitude toward surgical operations devised to overcome facial paralysis.

In 1937, *Bunnell*[1] published a excellent article in whcich he brought the literature on the subject up to date and in which he reported the first successful operation for intratemporal repair of the facial nerve.

When *S. Honjo*, discussed with Prof. *Rasmussen* of Montreal Neurosurgical Institute certain clinical observations that *S. Honjo* had made during the past year which indicated that fibers in the nerve trunk held to a definite pattern of location, he said he did not know that such observations had been previously reported. In view of this statement on the part of such an authority as Prof. Rasmussen, *S. Honjo* felt that we should report our observations for what they are worth to others.

The facial nerve is a mixed nerve which has a motor root, the facial nerve proper, and also a sensory root, the nervus intermedius of Wrisberg. These two roots meet in the geniculte ganglion. The motor root arises from a nucleus situated mainly in the lower part of the pons, but some cells (namely, those for innervation of the orbicularis oculi muscle) originate as high as the nucleus of the third or oculomotor nerve, and other cells (namely, those for innervation of the orbicularis oris muscle) are situated as low as the hypoglossal nucleus in the medulla. In the pons the motor root of the facial nerve pursues a very tortuous course. It forms a loop which hooks around the nucleus of the abducent nerve. As it leaves the ventral surface of the brain stem it enters the internal auditory meatus and passes through the temporal bone in the fallopian aqueduct there is a swelling in the nerve which is called the geniculate ganglion. It is in the ganglion that the sensory root or the nerve of Wrisberg originates. The ganglion is connected with the sympathetic system by fibers from the otic ganglion. The geniculate ganglion is similar in structure to a posterior root ganglion and is entirely sensory in function. The cells of origin of the intermedius nerve from a very small, slender nerve trunk which can be identified in the internal anditory canal as it lies between the facial and auditory nerves. It runs centrally into the medulla along the fibers of the auditory nerve and joint the nucleus closely connected with the glossopharyngeal nucleus. Peripherally from the ganglion, fibers run along the great and small superficial petrosal nerves; others proceed along the trunk of the facial nerve in the chorda tympani. The cutaneous regions supplied by the geniculate ganglion, according to *Ramsay Hunt*'s observations include the tympanic membrane, the external auditory meatus and adjacent skin of the tragus, antitragus, concha and part of the lobule. The intermedius nerve conveys impulses of tasts from the anterior two-third of the tongue upwards to the brain through the chorda tympani; this nerve terminates near the glossopharyngeal nucleus. It also contains afferent secretory fibers which join the submaxillary ganglion and innervate the sublingual and submaxillary salivary glands.

Beyond the geniculate ganglion the facial nerve bends suddenly and proceeds downward in that portion of the facial canal wich is situated in the upper part of the median wall of the tympanic cavity and enters the middle part of the ear immediately above the oval window. From this point it runs in a backward direction under the anterior end of the horizontal semicircular canal and turns sharply downward behind the middle ear and leaves the skull at the stylomastoid foramen. As it passes through the middle ear it sends off a branch for the stapedius muscle, and the chorda tympani leaves the trunk of the nerve just beyond and passes through the glasserian fissure to joint the lingual nerve. The communicating branches to the auricular branch of the vagus are given off a short distance above the stylomastoid foramen.

Diagnosis of the situation of a lesion causing facial paralysis may be determined accurately anywhere in the course of the pathway of the nerve from the lower end of the precentral cortical gyrus to the innervation of the peripheral muscle when disease of the middle ear is not present. Cerebral lesions, because they cause the upper neuron type of paralysis, spare the occipitofrontalis, orbicularis palpebrarum and corrugator supercilii muscles. Lesion in the upper part of the pons would be accompanied by involvement of the pyramidal tract on the same side. A lesion in the lower part of the pons should produce involvement of the opposite side. If the lesion is in the medulla, the eye on the same side should turn inward from involvement of the absent nerve, and there should be accompanying effect on the pyramid and fillet.[2] Lesions proximal to the geniculate ganglion usually cause impairment of hearing and equilibrium. Involvement of the geniculate ganglion (Ramsay Hunt Syndrome) is manifested by herpes of the external auditory canal and the adjacent auricle. If the lesion is situated distal to the geniculate ganglion, the resulting paralysis is of the lower neuron or flaccid type and involves all the facial muscles. All are familair with the typical clinical picture of facial paralysis. If sensations of taste are absent from the anterior two-thirds portion of the tongue, the lesion is situated between the geniculate ganglion and the point at which the chorda tympani leaves the nerve trunk to pass through the middle ear. Involvement of the branch innervating the stapedius muscle or the chorda tympani is not always easy to determine in chronic suppurative otitis media.

It is apparent that for the patient to have the aforementioned syndromes, the entire trunk must be affected by the lesion. That complete involvement of the nerve trunk is not always present in peripheral lesions causing partial paralysis has been clinically observed.

Randall[3] recounted a case in which the face of a four months' old baby was partially paralyzed. The baby was found to have a tympanum filled with polyps. Mention was not made of the part of the face involved. In exploring to locate the origin of the polyps, *Randall* encountered a loose spicule of bone and removed

it. It proved to be the anterior half of the stapes. The middle ear healed but the facial paralysis did not clear until after suppuration of the glands in front of the ear had ceased. *Randall* observed another patient who had slight facial paralysis. Paralysis increased to complete involvement in the course of a few weeks. Removal of a sequestrum at the apex of the cochlea was followed by recovery of facial func tion. In another case of a young woman, aged 21, paresis of the lower distribution of the facial on the first day after operation was observed. Full recovery from the facial paralysis did not take place until after a lapse of four years. *Weille*,[4] in discussing a case reported by Gahill and associated at meeting of the staff of the Massachusetts Eye and Ear Infirmary, said that because only the lower portion of the face was involved (in the case under condition) he thought the lesion must have been centrally rather than peripherally located.

Whether partial facial paralysis is encountered preoperatively or postoperatively in a given case, the portion of the face involved may provide the information necessary to determination of the site of the lesion.

Of most importance to otologists in evalution of pathologic factors are the anatomic relationships of the facial nerve within the middle ear. The otologist seems to be concerned with injury of the nerve below the level of the horizontal semicircular canal more often than he is with the promontory of the middle ear. Pathologic lesions are more likely of affect the facial nerve under the anterior end of the prominence of the horizontal canal and near the eminentia pyramidalis. Necrosis of the bone in this region or resorption of the bone caused by the insidious destrustive effect of cholesteatoma are the two main pathologic causes of involvement of the facial nerve.

During a radical operation for otitis media the facial nerve easily may be injured in the region in question in an effort assiduously to remove every vestige of disease. Great caution is necessary to avoid traumatization of the nerve with instruments. To help avoid such trauma the anesthetist or nurse must report any facial convusive movement and be warned instantly to keep close watch of the face when the surgeon is working in this region.

REPORT OF CASES

Case 1.– A woman, aged 36 years, had been performed on a left radical mastoidectomy for chronic mastoiditis in September 1956. About ten hours later it had been noticed that she was unable to draw in water through a drinking tube. Five days later paralysis of the face was complete. Galvanic stimulation of the muscles had been used for several months without improvement. The patient was admitted to the Royal Victoria Hospital of Montreal in May, 1957. At this time there was complete paralysis of the left facial muscles. The neurologic examination revealed a hysterical type of anesthesia over the distribution of the fifth cranial

nerve which extended down to the clavicle. The patient's personality was much affected by the facial deformity. The cavity created by the aforementioned radical operation was well healed. Five days after admission it was decided to perform an intratemporal bone exposure of the facial nerve. Exposure of the facial nerve from the region of the horizontal semicircular canal to the stylomastoid foramen showed a defect in the outer aspect of the nerve at a distance of less than 1 cm. below the horizontal canal. It appeared to be caused by impaction of a splinter of bone. The spicule was attached to a mass of inflamed tissue. The nerve had not been completely severed. The defect had filled in with a neuroma. This was removed and the neurilemma of the nerve split at the stage in the operation. Rapid healing of the wound took place. The patient was sent home and underwent treatment in the form of massage and electric stimulation. Improvement had taken place in six months.

The remarkable feature in this case was that delayed postoperative partial facial paralysis followed ten hours after operation. Within five days paralysis was complete. In radical operations for mastoiditis the chorda tympani usually is traumatized; consequently, testing for taste does not give particularly important information. It may be assumed from the operative findings that injury to the outer aspect of the nerve caused paralysis of the lower half of the face. Later, swelling of the nerve within the facial canal caused complete paralysis.

Case 2.– A man, aged 34 years, complained of pain and foul discharge on the left ear. The ear had discharge intermittently since his childhood. The lower part of the face had been paralysed for a week. It was the appearance of the facial deformity that had caused the patient to seek advice. He also stated that he had had attacks of vertigo for years.

On examination the left ear canal was filled with foul, purulent discharge. When removing the pus the middle ear was seen to be filled with polyps which bled on the slightest manipulation. Functional examination revealed that the patient heard a loud spoken voice in the left ear when the exclusion apparatus was used in the right ear. A diagnosis was made of chronic suppurative otitis media with cholesteatoma and partial facial paralysis associated with probable circumscribed labyrinthitis. Operation was performed on. Exposure of the antrum revealed that a cholesteatoma had produced a large cavity in the tympanic attic and antrum. On removal of the cholesteatoma it was seen that the horizontal canal had been completely eroded. The labyrinth was not disturbed further. The facial nerve exposed by the disease on the promontory above the anterior end of the horizontal. When the under surface of the nerve was manipulated, the muscles around the left eye twitched. All remnants of the matrix of the cholesteatome were removed. On the third postoperative day the lower part of the face began to function. The patient was discharged from the hospital on the twenty-first postoperative day. Facial paralysis had completely disappeared, and hearing had

improved to such an extent that the patient could detect a loud whispered voice in the left ear when the exclusion apparatus was used in the right ear.

SUMMARY

Clinical evidence is presented which seems to show that partial facial paralysis occurring before or after operations such as are mentioned herein has diagnostic significance in location of the part of the nerve trunk involved. The institution of early operative relief for the compressed or injured nerve seems to be a rational method of treatment. Partial facial paralysis occurring in the presence of chronic suppurative otitis does not necessarily mean that the lesion causing the paralysis is situated in the intracranial nuclei.

REFERENCES

1) BUNNELL, S.: Surgical repair of the facial nerve. *Arch. Otolaryng.*, **25**: 235, 1937.
2) PAPEZ, T.W.: Subdivisions of the facial nucleus. *J. comp. Neurol.*, **43**: 159, 1927.
3) RANDALL, B.A.: Various Otiti facial paralyses. *Tr. am. Otol. Soc*, **13**: 341, 1914.
4) WEILLE, F.: Discussion. *Arch. Otolaryng.*, **23**: 498, 1936.