TECHNICAL NOTE

AN OPERATION TO RESTORE EYELID FUNCTION LOST IN LEPROSY[1,2]

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If, as a result of leprosy, fifth cranial nerve damage causes an insensate cornea, and a seventh nerve motor loss prevents function of eyelids, a double threat to the cornea exists. Foreign bodies and the discomfort of an exposed cornea go unnoticed, and the unblinking lid fails in its protective function. Blindness usually results if nothing is done to restore the motor function of the lids, and patients with either arrested or active leprosy may lose their vision because of this dual danger. Little can be done to restore the sensation of the cornea; thus, the prevention of blindness revolves around restoration of motor function to the paralyzed lids. Blepharorrhaphy (closing the lateral half of the palpebral fissure by surgical synechia of the lids) has been used but with only partial success. With the recent great efforts at rehabilitation, plastic surgeons have made increasing use of the method originated by Sir Harold Gillies ([1]) for restoration of lid motion which uses the unparalyzed temporal muscle effectively as a lid motor. Each time the patient bites, the lid is brought in contact with the cornea, wiping away dust, etc., and spreading tears over the otherwise dry cornea. This action is much like the closing of a sagging buttonhole by pulling it taut or the stretching of a lax rubber band.

The usual Gillies procedure has been popularized by N. H. Antia ([2]) of Bombay, who probably has carried it out in the greatest number of cases. He was the first to apply the operation to aid in preventing the blindness in leprosy resulting from lagophthalmos. A modification of the Gillies method has been proposed recently ([4]). The objection that led to the modification was that in the Gillies procedure the polarity of the muscle transplant was reversed. Actually, by using the lower portion of the temporalis tendon and leaving the muscle fibers untouched in their bed, the excursion that the new lid motor will make is assured. It appears best to carry out the operation under local anesthesia and heavy sedation because the excitement stage of recovery from general anesthesia could jeopardize the repair. In any event, the field in which the repair is made is often insensate in leprosy patients.

The method is simple. After infiltrating the surgical field with 0.5 per cent Xylocaine, with 1:200,000 adrenalin to control the copious capillary oozing of the scalp, an 8 to 10 cm. incision is made in the

[1]Received for publication October 7, 1964.

[2]Cases reported in this paper were observed during a term as Fulbright Lecturer in Plastic Surgery, Christian Medical College, Vellore, S. India.

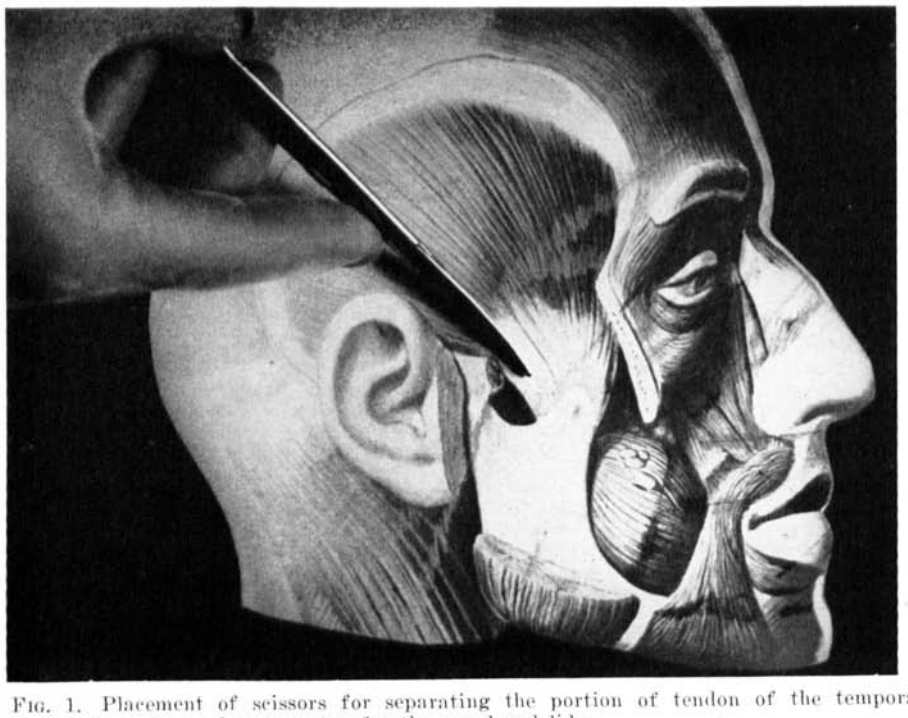

FIG. 1. Placement of scissors for separating the portion of tendon of the temporalis muscle that is to be used as a motor for the paralyzed lids.

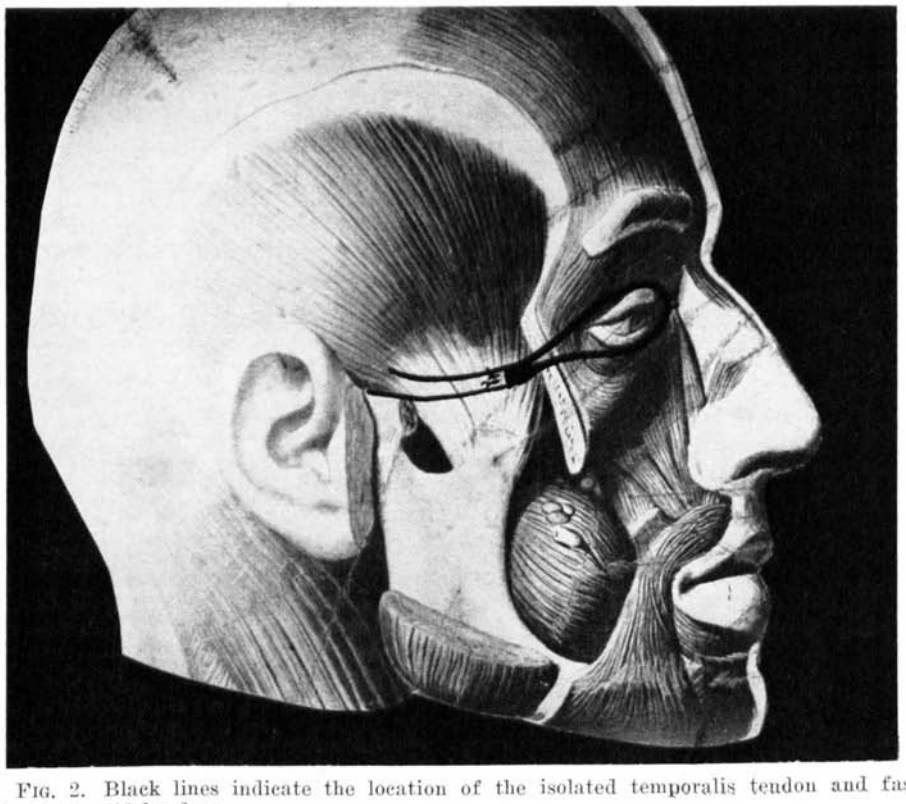

FIG. 2. Black lines indicate the location of the isolated temporalis tendon and fascial strips in the lid borders.

direction of hair growth in order to damage as few hair follicles as possible and prevent an obviously bald scar. Two strips of fascia about 2 mm. in width and long enough to reach from the temporalis tendon to the inner canthal ligament, are cut from the temporal fascia in the process of exposing the muscle belly. About a fourth of the temporal muscle tendon is used, viz., that portion attached to the lower or caudal muscle fibers (Figs. 1 and 2). This portion of the muscle and tendon is oriented horizontally in the transverse, the direction in which the eyelid must be pulled in order to close it. The strips of fascia taken need not be oriented in any particular direction. I have examined carefully bits of temporal fascia and cannot recognize any particular orientation of the fibers. The strips of fascia are tunneled through the lid borders and then fixed together under the inner canthal ligament (Fig. 2). It is very important to fix them under the ligament, for failure to do this can produce a bowstring web over the medial canthus. The fascial strips are then fixed to the temporal muscle tendon with care to adjust the tension to give the exact amount necessary to close the lids precisely. Under local anesthesia, this is relatively simple. Often with the inebriation of sedation, the patient is so intrigued with the new function that it is difficult to get him to stop during the suturing and wound closure. The eye is covered and taped shut during the postoperative healing period.

Further reconstructive surgery for correction of the paralysis of the oral muscle may be carried out at the same time, but that is another problem in the complete rehabilitation of faces paralyzed by leprosy. Lid function is restored, and with every contraction of the muscles of mastication the patient blinks, wiping the cornea. It is not the natural action of the sphincter orbicularis oculi or the levator palpebrarum, but it simulates it closely enough to restore lid function. The photograph of a patient (Case 1) shows the preoperative failure of the lids to close (Fig. 3) and the degree of lid function restored (Fig. 4) by this procedure.

Most authorities agree that neglected cases of lagophthalmos and corneal anesthesia cause the bulk of the blindness in leprosy patients ([5]); yet the other causes should be kept in mind if one deals with Hansen's disease. Direct involvement of the eye and adnexa oculi are more rare but lead to loss of eyes. Treatment and resulting erythema nodosum leprosum reactions can be dangerous if neglected ([3]). One must recall continuously that the probable incidence of all diseases that cause blindness (glaucoma, senile cataracts, trachoma, etc.) is assumed to be approximately the same in leprosy patients as in the normal population. Too often such problems are neglected because blindness is thought empirically to be associated with leprosy. Tuberculosis, syphilis and malnutrition are common in populations with a high incidence of leprosy, and these diseases also add their contribution to the attrition of eyes. Leprosy, as is often emphasized, is a disease of multiple disciplines crossing the borders of all specialties.

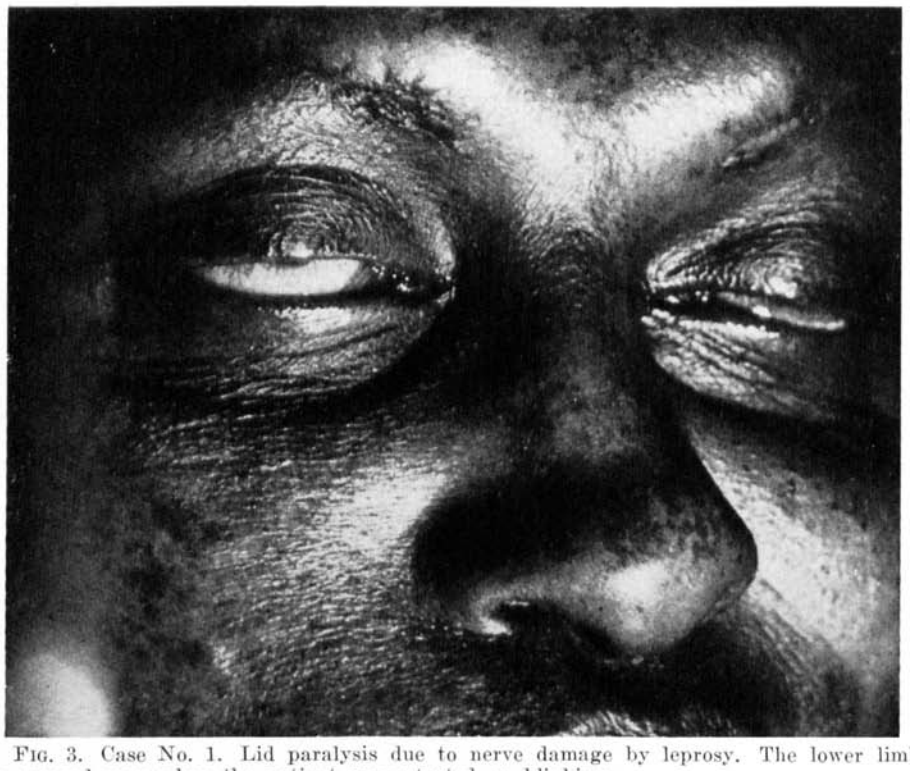

FIG. 3. Case No. 1. Lid paralysis due to nerve damage by leprosy. The lower limbus was exposed even when the patient concentrated on blinking.

Two cases are cited to illustrate the operative procedure.

CASE 1. A male patient (Figs. 3 and 4) about 65 years of age (birth date unknown) had been under intensive treatment for tuberculoid leprosy. He had numerous stigmata of the disease, including progressive corneal scarring due to an exposure keratitis associated with a lagophthalmos and paralysis of the facial nerve on the right side. Previous rehabilitative surgery had been carried out without major incident. It was felt that incipient blindness could be prevented if lid function were restored. In order to provide a motor to the paralyzed lids, a modified Gillies type of temporalis transfer was done. The surgery was performed under sedation with 3 gm. of Seconal (195 mgm. of Secobarbital sodium) and local anesthesia with 0.5 per cent Xylocaine and 1:200,000 adrenalin (Lidocaine hydrochloride).

Before wound closure, the effectiveness of the lid motor was tested by having the patient clench his teeth. When he did so, the lid would close and wipe across the previously exposed cornea. Aureomycin ophthalmic ointment (Chlortetracycline HCl) was instilled into the eye at the close of the operative procedure. A voluminous pressure dressing was left over the eye for 24 hours. For the 10 days following this, the eye was taped shut to prevent movement. Once a day it was irrigated with physiologic saline, and Aureomycin ointment was instilled into the conjunctival sac. The cotton skin sutures were removed on the third postoperative day.

The patient was then able to blink by clenching his jaw consciously. Also infrequent blinks were produced when chewing. Lacrimation, which had been a problem before surgery, was no longer troublesome.

CASE 2. A young adult male, about 30 years of age, had a unilateral facial paralysis of more than a year's duration due to lepromatous involvement of the facial nerve. There was some scarring of the cornea and a chronic conjunctivitis due to long-standing

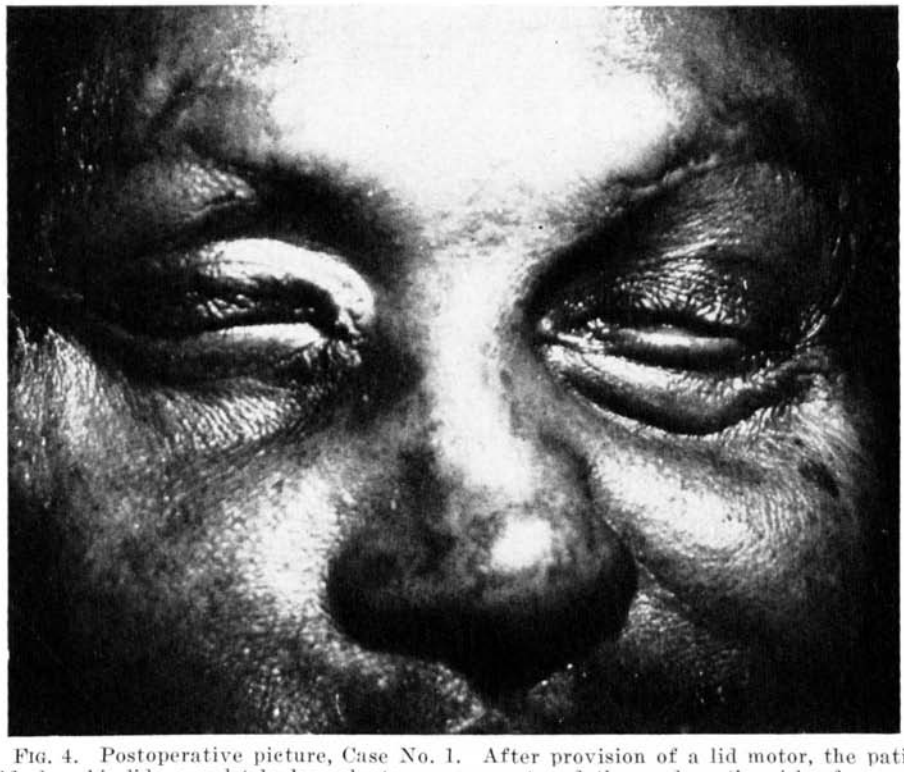

FIG. 4. Postoperative picture, Case No. 1. After provision of a lid motor, the patient could close his lids completely by voluntary movement and thus reduce the risk of exposure keratitis.

exposure. Although his general treatment was barely under way, it was felt wise to proceed with correction of the lagophthalmos in order to prevent further corneal damage and possible resulting blindness. In order to provide a motor to the paralyzed lid, a modified Gillies type of temporalis transfer was performed, with sedation and local anesthesia as in Case 1.

The effectiveness of the lid motor was tested at the time of surgery by having the patient clench his teeth. The resultant contraction of the temporalis muscle closed the previously paralyzed lid. Aureomycin ophthalmic ointment was instilled into the eye as in Case 1, and the eye was covered with a pressure dressing. After 24 hours the eye was dressed, and then daily for the next 10 days it was irrigated with physiologic saline and treated with Aureomycin ointment as in the previous case. The lids were taped closed during this time to minimize movement and allow firm healing. The cotton skin sutures were removed on the third postoperative day.[3]

SUMMARY

In leprosy, blindness may result from paresis of the fifth and seventh cranial nerves and resultant exposure-keratitis secondary to loss of lid function (lagophthalmos). Restoration of lid function protects the cornea, corrects the keratitis due to exposure and thus may prevent blindness. Lid function is restored by creating a motor mechanism for the paralyzed eyelid. A portion of the temporal muscle and strips of temporal fascia are used to create a lid motor. The insensate

[3]Postoperative follow-up on these patients was not possible because of local conditions.

cornea is thus wiped and moistened with every movement of the temporal muscle, unconsciously, as when the jaws are moved in chewing, or by conscious effort.

RESUMEN

En la lepra, la ceguera puede sobrevenir por la paresia del quinto y séptimo nervios craneales y la resultante queratitis por exposición secundaria a la perdida de la función palpebral (lagoftalmos). La restauración de la función palpebral proteje la córnea, corrije la queratitis debida a la exposición y por lo tanto, puede prevenir la ceguera. La función palpebral es restaurada por la creación de un mecanismo motor para el párpado paralizado. Una porción del músculo temporal y bandas de la fascia temporal, son usadas para crear un motor palpebral. La córnea insensata es entonces limpiada y humedecida con cada movimiento del musculo temporal, inconscientemente cuando las mandibulas se mueven al masticar, o por esfuerzos conscientes.

RÉSUMÉ

Dans la lèpre, la cécité peut provenir de la parésie des cinquième et septième nerfs craniens et de la kératite secondaire qui résulte de la perte de la fonction des paupières (lagophtalmos). La restauration de la fonction des paupières protège la cornée, corrige la kératite due au manque de protection et prévient dès lors la cécité. La fonction des paupières est restaurée par la création d'un mécanisme moteur pour la paupière paralysée. Un fragment du muscle temporal et des lambeaux du fascia du temporal sont utilisés pour restaurer la motricité de la paupière. La cornée insensibilisée est alors essuyée et humectée à chaque mouvement du muscle temporal, inconsciemment comme lorsque les mâchoires sont mobilisées pour la mastication, ou suite à un effort conscient.

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REFERENCES

1. Andersen, J. G. Surgical treatment of lagophthalmos in leprosy by the Gillies temporalis transfer. Brit. J. Plast. Surg. **14** (1961) 339-345.
2. Antia, N. A. Reconstruction of the face in leprosy. Internat. J. Leprosy **31** (1963) 597-598 (Abstract).
3. Choyce, D. P. The eyes in leprosy. *In* Leprosy in Theory and Practice. Ed. by R. G. Cochrane. Bristol, John Wright & Sons, Ltd., 1959, p. 161.
4. Johnson, H. A. A modification of the Gillies temporalis transfer for the surgical treatment of the lagophthalmos of leprosy. Plast. & Reconst. Surg. **30** (1962) 378-382.
5. Mann, I. Ophthalmic impressions of a leprosarium. Leprosy Rev. **26** (1955) 10-14.