THE SURGICAL MANAGEMENT OF PARETIC SQUINT

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SUMMARY

It is useful to distinguish between paretic squint of congenital and acquired origin. Furthermore, if the binocular reflexes are still in a state of flux, restoration of parallelism is a matter of urgency.

The results of surgery are dependent upon the cause of the paresis, the state of the extra-ocular muscles, the technique of the surgeon, and the patience of the patient.

When the nerve supply of a muscle has been sufficiently interfered with so as to preclude any action of the muscle, it is said to be paralysed. When the interference has been incomplete and some action is still possible, it is said to be paresed.

If paralysis or paresis affects one or more of the extra-ocular muscles, a squint results. This squint is incomitant in that the angle of deviation increases as the gaze is directed towards the main action of the affected muscle. With the passage of time, however, and particularly where there has been some recovery of the muscle, a certain amount of concomitance occurs, so that it may become difficult to establish whether the squint was originally of paretic origin or not.

Paretic squint is by no means always associated with interference with the nerve supply of the extra-ocular muscles. In fact it is commonly due to intra-orbital or intra-muscular pathology which has been present since birth or which has been acquired in adult life. In reviewing the causes of paretic squint the pattern emerges clearly that the more central the lesion, the more likely is it to be of acquired origin. Conversely, congenital paresis of the extra-ocular muscles is more likely to be associated with orbital rather than central pathology. But whatever the cause, the effect is identical in that, initially at any rate, diplopia results from the loss of ocular parallelism. The effect produced by this diplopia is dependent upon several factors, most important of which are the fixity of the binocular reflexes, the acuteness of onset of the lesion and upon its essential cause.

The earlier the onset of the paresis the more disastrous will its effect be upon the developing binocularity, even to the extent of strangling it at birth. Even a minor and transient obstacle in a child whose binocular reflexes are still in a state of flux, will have a very marked effect. The initial diplopia is originally overcome by central suppression of the second image, adoption of an abnormal head posture, or development of a compensatory ptosis.

The adult, on the other hand, will have great difficulty in compensating for the diplopia, particularly if it is of sudden onset. More often than not his mechanism of overcoming the second image is to close the affected eye. In less acute cases a compensatory head posture may be adopted but, unlike the abnormal head posture of the congenital variety, the patient is always aware of this new head position. Central suppression, except where the angle is very large, is unusual in the acquired variety of squint.

In discussing management, it is useful to take into account not only which muscle or group of muscles is involved, and the type and site of the lesion, but also whether the paresis is of congenital or acquired origin. The effect upon the patient of the presence of congenital fibrous bands restricting the action of his muscles, e.g. Duane's retraction syndrome, compared with the acute onset of a lateral rectus palsy in an adult, is as different as the disability produced by Hirschsprung's disease compared with an acute intestinal obstruction in the adult.

It is as well to recall that, where there is paresis of one muscle, there is inco-ordination of its fellows, this being particularly evident when the interference with its action is of sudden onset. The effect is to produce over-action of the antagonist of the affected eye, secondary weakness of the antagonist of the unaffected eye plus overaction of the contralateral synergist. For example, with paresis of the left lateral rectus muscle, there is over-action of the left medial rectus, weakness of the right lateral rectus and overaction of the right medial rectus. Similarly if there is paresis of the right superior oblique muscle, overaction of the right inferior oblique, weakness of the left superior rectus and overaction of the inferior rectus will occur.

If, at the time of onset of the paresis, the binocular reflexes are firmly grounded, the objective and subjective findings will be mutually reciprocal. If not, facultative

suppression in the direction of gaze of the paresed muscle or amblyopia with a constant manifest squint, is likely to be present.

CONGENITAL PARETIC SQUINT

Manifestation

This may manifest itself at birth, in childhood or in later life, the age of manifestation depending upon the severity of the lesion, the degree of compensation present and the precipitating factor. At one end of the scale we find the child with strabismus fixus who, because of his disability, is quite unable to develop any sort of binocularity. At the other, we have the middle-aged person with a congenital palsy of one of the vertically acting muscles for which compensation has been complete all his or her life. A sudden illness, e.g. acute cholecystitis, precipitates the decompensation with resultant diplopia. This may be noticed at first in one direction only, for example when reversing the car, but later vertical diplopia in the primary position becomes increasingly troublesome and demands treatment.

Another precipitating cause is parturition, and, particularly where there have been obstetrical complications, unless one is aware of the ultimate aetiology of the diplopia, grave prognostic import of the squint may be unnecessarily assumed.

One should always bear in mind that decompensation may occur at any age. The younger the age, the less severe does the obstacle have to be to produce a squint. We are all familiar with the infant or child who develops a horizontal squint for no apparent reason until one discovers that there is a vertical deviation as well, usually due to inferior oblique overaction which may be secondary to superior oblique paresis or superior rectus palsy. This vertical element probably remains compensated for until some obstacle, for example intercurrent illness or an error of refraction, intervenes and breaks down the binocular reflexes which are not yet unconditionally fixed.

Detection of the vertical deviation in a small child is not at all easy, but its presence should be suspected in the absence of any other reasonable cause for the squint. It should be diligently searched for, for in early surgical correction lies the only hope of the child obtaining binocularity later. Repeated operations are, more often than not, necessary, and, if cure is promised after one procedure, one must conclude that the surgeon is either inexperienced or over-optimistic. Alternating occlusion, the use of +2 D spectacles and other conservative methods of treatment constitute a waste of valuable time. Likewise postponement of surgery until the child is older will deny it any chance whatever of gaining binocularity.

One should stress, however, that, before embarking upon surgery, very careful evaluation of the case should be undertaken, particularly so as to eliminate intra-ocular pathology, the most important of which is of course retinoblastoma. Examination with general anaesthesia is not only wise, it is essential.

Indications for Surgery

The indications for surgery in the congenital paretic group are therefore (*i*) to prevent decompensation where it is threatened, (*ii*) to correct it early where it has occurred, and (*iii*) to remedy any abnormal head posture if this is remediable. With regard to the latter, and where compensation is good, particularly if this harmonious state of affairs has existed for some time, overenthusiastic surgery can lead to disastrous results. An excellent example of this type of case is one in which a Hummelsheim procedure is undertaken in the presence of a Duane's retraction syndrome of the A type where weakness of adduction is present.

The aim of surgery in congenital paretic squint is to restore full function where it is restorable and to provide the patient with the largest possible binocular field. If neither aim can be achieved, surgical intervention is, at the best, cosmetic.

ACQUIRED PARETIC SQUINT

The onset of acquired squint is often dramatic and, like the actor striding onto the stage, demands attention. As in the congenital variety, the discovery of the cause is of primary importance. It is beyond the scope of this article to describe or discuss the many causes of acquired paretic squint, particularly those which result from intracranial mischief. In these cases, particularly where the nerve supply to a muscle or a group of muscles has been damaged, the amount of spontaneous recovery which will occur may be difficult to assess initially. If the binocular reflexes have been firmly grounded, surgical correction of the squint is usually not a matter of urgency, and one is wise to continue with conservative measures for several months until one is certain that no further spontaneous recovery will occur. If, however, the binocular reflexes are in a state of flux, earlier surgical intervention may become necessary. The use of daily alternating occlusion in these children may, if continued too long, extinguish all desire for binocularity and should therefore be used with great caution.

Acquired paretic squint of intra-orbital origin may prove to be the exception to the rule of waiting for recovery to occur. Among the important conditions in this regard is that of a blow-out fracture of the orbit with incarceration of the orbital contents which herniate through the break in the orbital floor. This results in paresis of upward gaze as well as in some restriction of depression. Early extra-periosteal orbitotomy with the insertion of a silastic patch over the fracture restores function fully, but, once the orbital contents have been allowed to become bound down by fibrous tissue, it is seldom possible to correct the resultant diplopia fully even with repeated procedures.

Another important exception, and one where one cannot afford to wait with treatment, is the ophthalmoplegia associated with rhabdomyosarcoma. In this condition the tumour arises from one of the extra-ocular muscles and may produce a paretic squint before its space-occupying properties become evident by producing proptosis. Early orbitotomy, exenteration and the use of cytotoxic drugs with radiotherapy may materially affect the ultimate outcome.

Ophthalmoplegia associated with endocrine exophthalmos presents special problems of a different kind as to whether to interfere surgically or not. Initially in this condition the accent is on the orbital oedema, particularly where proptosis threatens the cornea. It gradually shifts to the problem of diplopia which occurs as the round-

cell infiltration of the muscles gives way to the formation of scar tissue and contracture. Once this stage has been reached, conservative treatment by retrobulbar and general steroid therapy amounts to closing the stable door once the horse has fled. The reason for this is evident when one operates on these cases, as there is a curious lack of elasticity of the muscles. The inferior rectus is the most commonly affected muscle.

THE SURGERY OF PARETIC SQUINT

There are certain surgical principles which are useful to remember when planning to operate on any case of paretic squint:

1. One should always bear in mind the fact that a great deal of one's activities are associated with looking downwards and straight ahead and that, where it is impossible to attain full function in all directions of gaze, it is in these positions that one aims at re-establishing parallelism.
2. It is better to weaken an elevator rather than a depressor for the above reason. For example, with superior oblique paresis it is better to recess the ipsilateral inferior oblique muscle than the inferior rectus of the opposite eye.
3. If there is a vertical squint and marked torsional activity is present, operation on an oblique rather than on a rectus muscle is advisable.

Congenital Paretic Squint

When compensated, it is better to undercorrect the defect rather than to aim at full orthophoria, as even slight overcorrection will increase rather than minimize the patient's discomfort.

In uncompensated cases, particularly in young children, where the squint is constantly present, recession of the direct antagonist as well as weakening of the contralateral synergist is indicated before a strengthening procedure on a paretic muscle, particularly if a Hummelsheim procedure is contemplated.

When there is vertical deviation in the uncompensated cases, it is better to correct the horizontal component first. When compensated, however, vertical deviations assume a much more important role and should be corrected simultaneously with or even before the horizontal component.

Acquired Paretic Squints

Provided that progressive intra-orbital or intracranial disease has been eliminated and that further spontaneous improvement is not anticipated, it is very seldom that diplopia can be relieved without surgery to the extraocular muscles.

Here one should always aim at full orthophoria as no compensatory mechanism has been learned and small degrees of deviation can be much more troublesome than large ones.

Where the muscle itself has been involved, the aim should be to strengthen the individual muscle and to weaken its antagonist. Therefore resection of the affected muscle plus recession of its antagonist is indicated with, perhaps, a Hummelsheim procedure in addition.

Where the nerve supply is involved, strengthening of the antagonist of the other eye plus weakening of the antagonist of the same eye is aimed at restoring symmetrical ocular movements in a given direction rather than restoring function of a single paresed muscle.

Where both elevation and depression are involved in one eye, for example incomplete third nerve palsy, it is probably better not to touch the affected eye at all but to recess the superior and inferior recti of the opposite eye.

It cannot be overemphasized that it is seldom that the paretic squint can be corrected at one operation and that staged procedures are more often than not required to relieve the patient of his disability. The surgery of acquired squint is far more exacting than that of the congenital variety and requires careful planning, meticulous surgical technique and infinite patience on the part of both the surgeon and the patient. There is no doubt that it is better to do less on two eyes than more on one, so as to avoid permanent restriction of movement.

Amount of Surgery

In my hands, I find that recession of more than 4 mm on the medial recti, more than 5 mm on the lateral recti and inferior obliques and more than 3 mm on the superior and inferior recti tends to produce permanent restriction of movement in the line of primary action of those muscles. Resections of more than 8 mm are unwise and likely to restrict the movement of the antagonist of the same eye. Free tonotomy and myectomies in my experience have been uncontrollable and therefore unjustifiable procedures where one is attempting to restore function.

It should be remembered that these measurements are not absolute but merely act as a guide when planning surgery. So much depends, for example, on whether the muscle to be resected is stretched or relaxed at the time of measurement and whether one's sutures are inserted at the very edge of the muscle to be recessed or 2 - 3 mm back from the cut edge.

It should also be remembered that the amount of recession and/or resection which is necessary to correct any given amount of deviation not only depends upon the individual surgeon and his technique, but on other factors which should also be taken into account.

The size of the globe and the amount of proptosis present influence the result, in that the larger the size and the more the proptosis, the greater will be the effect of the surgery.

The strength of the muscle, that is if it is paresed rather than paralysed, will likewise affect the result.

The amount of contracture of the antagonist, usually related to the length of time that the paresis has been present, will influence the amount of muscle adjustment necessary. This is best assessed by forced duction under general anaesthesia.

The age of the patient is important in that the younger the patient, the less surgery is usually necessary to produce the required correction.

The stability of the angle is also important as, if the angle of deviation is fixed, more surgery is required to correct the squint than if it is variable.

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