often taking place in six to twelve months according to the density of anaesthesia produced, with a small proportion lasting from two to three years. One of my cases is still free after ten years, and I have seen four and five years' remission, but these longer periods are unusual.

I have therefore been more inclined for some time past to recommend and to practise injection of the inner portion of the ganglion, in order to obtain a lasting cure. I observe that Mr. Jefferson has divided the middle portion of the sensory root in two or three cases, in order to limit the anaesthesia to the area of the second division, and during the past six months I have in two cases successfully injected the middle portion of the ganglion only—in a man, a private patient, and in a woman at Maida Vale Hospital—for second-division neuralgia. My method is similar to injecting the inner portion of the ganglion, but after pushing the needle through the foramen into the ganglion, the alcohol must be injected even more slowly and carefully than usual, waiting perhaps for ten minutes at a time after each injection of two minims, and testing repeatedly with a pin as a guide to the developing anaesthesia. In both cases total shock anaesthesia of the upper two divisions developed at first, but after ten minutes sensation began to return. More alcohol then deepened the anaesthesia on V.2 but did not affect V.1, and gradually more and more injections of one or two minims at a time produced total lasting anaesthesia

over the second division alone, which was unchanged a week later.

Fig. 3 illustrates anaesthesia resulting from injection of the middle third of the ganglion, in a man, aged 57, who had suffered from flashing pain from the right upper lip to vertex for the past five years: 0.9 c.cm. 90 per cent. alcohol was injected on January 13th, 1932. There was total anaesthesia to pin and pressure on infra-orbital area, upper jaw, and palate as far back as middle of soft palate, and slight numbness and diminished sensibility to touch over the temporo-malar area. Anaesthesia was unchanged eight days later.

FIG. 3

Since I first injected the Gasserian ganglion in 1910 for trigeminal neuralgia, I have treated by this method some hundreds of patients, many of whom have been free of all pain for a number of years, and may be considered as permanent cures. When the injections are done by the methods I advise, slowly and with great care and patience, using light morphine-scopolamine anaesthesia, or local novocain only, according to the temperament and wishes of the patient, the results are most gratifying both to the operator and to the patient, while there is no risk whatever to life, and a minimum of expense and time.

DISLOCATION OF THE PATELLA *

BY

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Dislocation of the patella is a comparatively rare condition, but it is nevertheless one of great interest on account of the factors involved in its cause and treatment.

AETIOLOGY OF OUTWARD DISPLACEMENT

Cases have been recorded of displacement occurring upwards or downwards, or inwards or outwards, but as outward displacement is by far the most common and the one which is found as a congenital condition, I am proposing to confine my paper to this variety.

Apart from the single traumatic dislocation, outward dislocation of the patella naturally divides itself into two main groups:

1. *Congenital*, in which the patella is permanently displaced and fixed on the outer side of the femur.
2. *Recurrent*, in which the displacement occurs either at infrequent intervals or as frequently as the knee is put through its range of movement.

It is generally accepted that the patella is a sesamoid bone in the tendon of the quadriceps. As Mackenzie¹ has said, its function is to improve leverage and thus increase the power of the muscle. It acts as a pulley and, by enabling the quadriceps to work at an angle, increases the work done by each contraction. The patella is an integral part of the extensor mechanism of the knee-joint—in dealing with its displacement, therefore, it is necessary to consider this mechanism in some detail.

THE EXTENSOR MECHANISM

The extensor muscle of the knee is the quadriceps. It consists of four muscles—the rectus femoris, the vastus externus, the vastus internus, and the crureus. All are supplied by branches from the anterior crural nerve, but these branches are so widely separated from one another in their distribution that they can be considered and injured as individual nerves. To obtain insertion, the

tendons of the four muscles unite and become attached through the ligamentum patellae to the tuberosity of the tibia. In addition, fibrous expansions on each side are attached to the capsule of the joint and to the anterior surface of the upper end of the tibia and the fibula. These are called the lateral patellar ligaments. On looking at the quadriceps and its attachment as a whole, one notices, as Goldthwait and others have pointed out, that in full extension the line of pull of the muscle makes an angle with the line of the patellar tendon. This angle is obtuse outwards, and is caused by the fact that when the knee is in the extended position the tibia and the femur are not in line with one another. For in this position the width of the pelvis and the length of the femoral necks separate the upper ends of the shafts of the femora while the lower ends are in close proximity. This would naturally result in an obliquity of the knee-joint, which, however, is prevented by a relative increase in the length of the internal condyle of the femur. This makes the joint horizontal, but results in throwing the femoral shaft out of alignment with that of the tibia. When the knee is flexed, though, the same conditions do not obtain, and the tibia and femur take up positions which become approximately parallel to one another. As the quadriceps clothes the shaft of the femur and the patellar tendon takes the line of the tibia, there must be an angle, more acute in the female than in the male, between the line of the muscle and that of its tendon, when the limb is extended (Fig. 1).

FIG. 1.—A, Showing angle between line of quadriceps and that of patellar tendon; and B, the alignment of the femoral and tibial shafts.

* From a paper read at a meeting of the British Orthopaedic Association at Nottingham, April 22nd, 1932.

When one observes the quadriceps contracting, one can see that as the knee extends the patella first travels in a straight line over the patellar surface of the condyles, but that as full extension is reached it is pulled laterally. This lateral pull at the completion of extension produces a very definite movement and would cause a dislocation of the patella were there not some natural means of preventing it.

What, then, prevents dislocation? It is primarily prevented by two factors:

1. The size of the patellar surface of the outer condyle of the femur.
2. The broad attachment of the patellar tendon and its inner lateral ligament to the upper end of the tibia and to the capsule of the joint.

These factors, therefore, both act as checks, preventing dislocation when the quadriceps contracts.

FACTORS FAVOURING DISLOCATION

It follows, on theoretical grounds, that the possibility of dislocation would be increased by anything which decreased these checks, or by anything which increased the angle between the muscle and its tendon. In other words, the possibility of dislocation would be increased by:

1. Diminution in the size of the external condyle.
2. A rupture or weakness of the internal part of the capsule and of the internal patellar ligament.
3. Weakness of the vastus internus (making the line of the quadriceps more angular).
4. A lateral attachment of the patellar tendon.
5. Genu valgum, laxity of the patella, or hyperextension of the knee.

It has been found, in fact, that one or more of these causes are usually present when dislocation occurs.

The patellar surface of the external condyle of the femur is a very important factor in preventing dislocation, and when I was considering the possibility of the presence of some developmental abnormality, as a cause of congenital dislocation, it was suggested to me that some assistance might be

FIG. 2.—Lower articular surfaces of right femur of: A, sheep; B, cow; C, gorilla; D, man—showing alteration in relative sizes of internal and external condyles.

obtained from comparative anatomy. I have therefore compared the femora of certain quadrupeds with those of the baboon, gorilla, and man, and have found as the result of these observations that in man alone is the anterior surface of the external condyle definitely larger than the internal. In the cow, the dog, and the sheep the internal condyle is the larger, while in the baboon and in the gorilla there is very little difference. And this is of great interest when one remembers that, apart from man, all walk with flexed knees. (Fig. 2.)

Their femora are approximately in alignment with their tibiae; there is consequently no angle between the extensor muscle and its insertion, and therefore no lateral pull. If these observations are correct, the relative increase in the size of the external condyle has occurred with the advent of the erect posture, and the lateral pull to which the human patella is subjected at the termination of extension is not present in the quadrupeds. If this is so, it seems not unreasonable to suggest that it indicates that the human quadriceps has acquired a new function. In the process of walking, the knee, having been extended, has to be held in the extended position for a momentary period when a forward step is taken. May it not be that the quadriceps is able to maintain this with a minimum of effort as a result of the lateral pull on the patella with a pulley action round the external condyle of the femur? Though the crucial ligaments and the rotation of the tibia lock the knee in extension and prevent hyperextension, may it not be that this mechanism locks the knee in extension and with the minimum of effort momentarily prevents flexion?

When the quadriceps is very weak or completely paralysed transplantation of the insertion of the biceps to the patella places this muscle in an ideal position for producing this lateral pull. It is possible that this accounts for the variation in opinion as to the value of this operation. A transplanted biceps, though not strong enough to extend the leg against gravity, may, on this theory, be strong enough to hold the knee extended for a momentary period and so enable walking to be done without the need of an instrument.

The lateral pull of the quadriceps may also account for the occurrence of congenital dislocation of the patella. For if the external condyle has retained its primitive size, the first contraction of the quadriceps, either *in utero* or after birth, may by its lateral pull dislocate the patella outwards and this may subsequently become fixed in the displaced position by secondary contractures. One of my x-ray photographs appears to support this suggestion. In this it can be seen that the external condyle is smaller than normal. It is, however, impossible to say whether this condition was present at birth or whether the under-development is due to lack of functional use. That the congenitally dislocated patella is sometimes associated with bony abnormalities is certain and it is itself sometimes abnormal, being occasionally rudimentary in size and occasionally double.

Dislocation of the patella may be also associated with a hereditary abnormality as illustrated by a group of cases reported by Moore.² In this group cousins married, and cases of recurrent dislocation of the patella occurred in several succeeding generations at the age of 9 or 10. Moore investigated one of these cases and found that the vastus internus was under-developed.

SYMPTOMS

These may vary considerably. In the traumatic cases there may be pain with sudden inability to walk, and, if unreduced, inability to straighten the knee. In the recurrent type not associated with trauma there may be no pain but great weakness of the leg. In the congenital type it is usually noticed that the shape of the knee is not normal and the child is ungainly in walking and frequently falls. I am able personally to report seven cases—of these, three had bilateral dislocations, five were congenital, and two recurrent. Of the congenital cases, one had double patellae, and in one the patellae were rudimentary. In the recurrent cases there was definite wasting of the vastus internus, probably due to infantile paralysis. Five of the patients were girls and two boys.

METHODS OF TREATMENT

For the single traumatic dislocation, reduction, followed by fixation for a time with the knee in the extended position, may be all that is necessary. For the recurrent and the congenital case, however, operative treatment is indicated. In discussing the causes of dislocation, emphasis has been laid on the importance of the external condyle both as preventing dislocation and as making walking possible with a minimum of effort. It might appear, therefore, that the first step in treatment should

be to increase the size of the external condyle of the femur, and, indeed, if the displacement did not involve any other factors, an operative increase in the size of the condyle might be all that was necessary. That, however, is usually not the case, although in the recurrent type of dislocation, where secondary factors are not involved to the same extent, Brackett and Albee have increased the size of the condyle by a bone graft, with good results. For the recurrent type, other methods have also been used with the object of altering the position of the patellar tendon, by transplanting the inner or the outer half or the whole tendon inwards. Supracondylar osteotomy of the femur with rotation of the lower end has also been done. Thinking that the weakness was in the capsule, some have plicated this or have transplanted a surplus from the inner to the outer side of the joint. Others again have, by fascial slings, attached the patella to the inner side of the femur, or have transplanted the gracilis on the semitendinosus to the inner side of the patella.

These methods are all said to have given good results in the recurrent type of dislocation, where the problem is not to replace the patella, but merely to hold it in position.

The Congenital Type

In the congenital type, however, something more is needed, and for these Mouchet³ has devised an operation in which the patella and its tendon are detached from their displaced positions and threaded through the capsule of the joint. The tendon is then reattached to the tibia at a point on its inner side. In this operation no attempt is made to realign the muscle.

It is obvious that in treating a congenital dislocation three objects should be kept in view:

1. To replace the patella.
2. To correct the line of pull of the quadriceps and of its tendon.
3. To maintain it in position.

It would seem, therefore, that the first step should be to realign the muscle and its tendon, and the second, to increase the size of the external condyle. In my experience, however, realignment of the muscle carried out by the method which I shall describe has in itself been quite sufficient to prevent recurrence of dislocation, and has enabled the quadriceps to function normally. The method, which has probably been used by many surgeons, consists of a combination of some of those already mentioned. I first saw it performed by Mr. Emslie, and him I have to thank for permission to report one of the cases in my list.

The aim of the operation is to replace the patella, freeing the outer side of the quadriceps and at the same time transplanting the tibial tuberosity inwards and displacing the patella to a slightly lower level than normal. (I have, where necessary, done a simple supracondylar osteotomy to correct genu valgum at a preliminary operation. This is sometimes not easy, and might with advantage be done at a subsequent operation.) The skin incision is made on the outer side of the thigh, extending from the middle third and curving slightly inwards, to end internal to the anterior border of the tibia 1 inch below the tibial tuberosity. Through this skin incision the outer border of the quadriceps is freed. The capsule on the outer side of the patella is divided, and this division is carried downwards through the periosteum on the outer side of the tuberosity for about $1\frac{1}{2}$ inches—the periosteum on the inner side of the tuberosity is also divided for a similar distance. With a chisel the patellar tendon, together with a flake of bone, is raised from the tibia, leaving its lower end attached to the periosteum and so maintaining the tension of the muscle. A raw area is then made on the surface of the tibia on the inner side of the tuberosity.

As the outer side of the lower part of the quadriceps muscle and the tendon are now free, the patella can be pulled over to the inner side and placed in its normal position, and the muscle realigned. This produces a gap in the capsule on the outer side, with a surplus of capsule on the inner side. This surplus is detached as a flap with a proximal pedicle and is drawn under the rectus femoris to the outer side of the joint. By sutures on its inner side the patella and its tendon are then fixed in position, the bone attached to the tendon being applied to the raw area on the tibia. The effect of this is to bring the patella to a slightly lower

FIG. 3.—Steps of operation. A, Freeing outer border of vastus externus and both sides of patellar tendon, and making a raw surface on inner side of tibia. B, Muscle and tendon displaced inwards. C, Sutured and so held in position, the surplus capsule being displaced from the inner to the outer side of the joint.

level than normal. Part of the displaced flap of capsule is then sutured in position to fill the gap in the capsule on the outer side, and as an added precaution, though it is not essential to the operation, part is brought back over the rectus and sutured to itself, thus forming a sling. The wound is then closed. (Fig. 3.)

The after-treatment consists in fixing the knee in the extended position until the wound is healed—then gradually commencing movements, and walking in a calliper.

In my experience this method has been used six times, two cases being recurrent and four congenital. Of these,

FIG. 4.—Line drawing from x-ray after operation on right leg, showing displacement of patella downwards and inwards.

the last was done too recently to allow of a report on its success or otherwise, but the remaining five have been completely successful. One was done 10 years ago, two 6 years ago, one 4 years ago and one a year ago. In none has there been any sign of recurrence, and all have full movement. (Fig. 4.)

SUMMARY

My conclusions are that dislocation of the patella is predisposed by the erect posture and may be due to imperfect development of a recently acquired function of the quadriceps. It results from an abnormality which may be present in: (1) the muscle itself; (2) the articular surface of the femur; (3) the attachment of the patellar tendon to the tibia.

A satisfactory and simple method of correcting this is to displace the muscle and its tendon inwards and to transplant the tibial tuberosity in such a way that the patella is brought down below its normal position. This ensures stability and enables the quadriceps to work with the maximum of efficiency.

REFERENCES

- ¹ Mackenzie, Sir W. Colin: *The Action of Muscles*, 1918, p. 157.
- ² Moore, B. H.: *Journ. of Bone and Joint Surg.*, July 12th, 1930, p. 654.
- ³ Mouchet, A.: *Bull. et Mém. Soc. de Chir. de Paris*, 1923, xlix, 511.

TREATMENT OF CHRONIC SUPPURATIVE DISEASE OF THE MIDDLE EAR *

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I have chosen the subject of the chronically discharging ear for a number of reasons. First of all this is a common disease. In many cases it resists all forms of treatment, and runs its course, not for months, but for years, and a chronic discharge inevitably leads to disability in the shape of hearing—a disability which, in certain cases, becomes profound. Moreover, although intracranial complications are comparatively rare, yet they do arise, and make up for their infrequency by the high rate of mortality.

There is still another reason why I have chosen this subject, and it is that treatment is apt to become stereotyped. We rely on one or two forms of drops, or, alternatively, a course of syringing. Both may be completely inefficient. In this disease treatment must be adapted to the particular case. Each ear has to be separately studied, and an attempt has to be made in every instance to determine the various causes of the chronicity.

THE MIDDLE-EAR CLEFT

The "middle ear" must be understood to include the whole of the middle-ear cleft, which starts in the nasopharynx, runs up the Eustachian tube, enters the middle ear or tympanum proper, invades the upper regions of that cavity which is known as the attic, and from there leads upward through a small tunnel or aditus into the mastoid antrum. From the antrum, in the majority of cases, open numerous air cells which extend throughout the temporal bone and carry the infection to the neighbouring portions of the cranium, to the meninges of the middle and posterior fossae, to the cerebrum and cerebellum, and to the venous sinuses. The nasopharynx, Eustachian tube, attic, aditus, antrum, and air cells are lined throughout by one continuous membrane. The mucous membrane, it is true, differs in different parts—for example, in the Eustachian tubes and in the interior of the tympanum itself it is rich in mucus-secreting glands, whereas in the posterior part of the

tympanum, in the mastoid antrum, and in the air cells, it is thin and deficient in glands.

In a case of chronic discharge from the middle ear, it is necessary to determine in which part of the middle-ear cleft lies the origin of the chronicity. For example, if the cause is in the nasopharynx—perhaps, an infected pad of adenoids—the treatment will have to be aimed at this. If, on the other hand, the condition is due to an osteomyelitis involving the mastoid process, and mastoid antrum and air cells, the removal of adenoids will be of no benefit, and treatment will have to consist of some form of mastoid operation.

SITES OF INFECTION IN CHRONIC OTITIS

I want to describe five typical situations, with the differential diagnosis of each. I shall deal, first, with a chronic discharging ear following a recurrent infection of the nasopharynx; secondly, with a discharging ear due to chronic infection of the mastoid antrum and air cells; thirdly, with a case in which the bulk of the disease lies chiefly in the region of the attic; fourthly, where the tympanum itself bears the brunt of the trouble; and, fifthly, where there is gross involvement of the whole of the middle-ear cleft. Each situation gives a perfectly clear and distinct clinical picture, and the majority of discharging ears can be placed definitely in one or other of these five classes.

FOCUS IN THE NASOPHARYNX

In the first group a focus of infection in the nose or throat leads to a recurrent infection of the Eustachian tubes and of the middle ear. A typical history is that of a recurrent discharge in a child, usually starting with a cold or sore throat. This is quickly followed by an attack of pain in the ear. If this is the first attack, the pain is frequently severe and lasts a few hours or even one or two days. In the untreated cases relief is finally obtained when the drum gives way before the accumulated pus in the middle ear, and a free discharge passes into the meatus. If the attack is one of a series, the pain in the ear is often slight—even absent—and of short duration. The drum, which has been ruptured before and has healed over with a thin scar, easily gives way, and the discharge starts with corresponding ease. After the ear has discharged the patient is relieved, the pain subsides, the temperature—which has been high at the onset—soon returns to normal, and the child recovers from the acute phase.

The character of the discharge, as well as the history, helps in forming the diagnosis. I have already mentioned that the mucous membrane of the Eustachian tubes is rich in mucous glands. A discharge which is due to infection of the tubes is consequently mucous and, later, muco-purulent. It is thick and sticky, adhesive and tenacious, and can be pulled out in long strands from the meatus. The site of the perforation, determined by a careful examination, is diagnostic. The external auditory meatus is mopped out. (If there is difficulty in getting a clear view by this method, a little gentle syringing will do no harm.) After the drum has been cleansed, the perforation is seen anteriorly, and generally in the lower part. It varies in size from a pin's head to a perforation involving half the drum. When the perforation has been clearly defined the patient is made to inflate the Eustachian tube by holding the nose, closing the mouth, and blowing down. This will blow up bubbles of mucus from the tube into the middle ear, and these will pass through the perforation, giving clear evidence of the source of the discharge. The duration of the attack varies considerably. If the child is otherwise healthy and in good condition the discharge may dry up entirely in a few days. The perforation heals over quickly, and

* Delivered to the Croydon Division of the British Medical Association on December 15th, 1931.