THE INTERNAL SPERMATIC FASCIA WITH REFERENCE TO REPAIR OF INDIRECT INGUINAL HERNIA

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The object of this communication is to stress the importance of a thorough exposure of the defect at the internal inguinal ring during the course of operation for repair of indirect inguinal hernia in adults. This step, which is usually dismissed all too briefly in descriptions of the operation, is an essential prerequisite to the performance of an accurate repair. A defect is always present in these cases but requires careful dissection to be clearly displayed, and it is obvious that unless this is done the repair can be only approximate and the risks of recurrence are thereby increased.

Hughes and Fathi (1948), in a short but clear and precise paper, have drawn attention to the same subject, but the manoeuvre they advocate (see below) is not, in my experience, quite so easy as it sounds.

The operation referred to above is the one described, with slight modifications, by Ogilvie (1937), Edwards (1943), Lytle (1945), and others. On anatomical and pathological grounds it seems clearly to be the operation of choice. Its essential feature is the direct repair of the stretched internal ring after excision of the sac, without altering in any way the normal anatomy and mechanism of the inguinal canal.

But the difficulty is that the stretched ring is not immediately apparent; its margins are very indefinite and the transversalis fascia itself is not at all easy to identify. It is hoped to show that this difficulty can be overcome by the simple procedure to be described. At least I have found it extremely helpful in the course of some 200 operations.

Anatomy

It is necessary first to refer briefly to the anatomy of the internal inguinal ring. The ring is simply an opening in the transversalis fascia for the passage of the spermatic cord from the abdominal cavity into the inguinal canal. Lytle (1945) has shown that it is strengthened by an obliquely placed U-shaped fibrous sling over which the cord bends; but this is not really relevant to the present argument except to show that the normal ring has three well-defined margins—namely, superior, medial, and inferior—while the fourth one—that is, lateral or more precisely supero-lateral to the cord—is indefinite.

From the margins of the ring a tube of fascia—the internal spermatic fascia—is prolonged on to the cord, to which it closely adheres. In fact the cord does not pierce the transversalis fascia at all, but merely evaginates it at the internal ring (Fig. 1). The continuity of the internal spermatic fascia with the transversalis fascia makes it extremely difficult to decide what are the limits of the ring when viewed from the external aspect

—that is, as seen at operation. *Gray's Anatomy* (1946) actually states: "This opening [the internal inguinal ring] is not visible externally since the transversalis fascia is prolonged on these structures [spermatic cord or round ligament] as the internal spermatic fascia." This observation can be easily verified on any normal subject in the post-mortem room and at every operation for inguinal hernia.

The Operation

When an indirect sac is present it is found to lie lateral and superficial to the cord, and always within the internal spermatic fascia. After the cord has been lifted out of its bed the fascial tube must be deliberately incised, and the sac may then be picked up with forceps and freed up to its neck by blunt dissection. It is excised as usual and the stump allowed to retract.

If the cord is now lifted up with slight traction and the conjoint tendon retracted outwards, the appearance will be more or less as shown diagrammatically in Fig. 2. The internal spermatic fascia will be seen to spread out funnel-wise as it approaches the internal ring, which at this stage is not visible. A rent is apparent in the fascia through which the sac was dissected, and is often reduced to a mere slit as the cord is lifted up. The stump of the sac will have disappeared within this rent. It is remarkable how this fascial tube, in spite of the rough treatment which it often receives during the dissection of the sac, becomes more or less accurately re-formed after the excision of the sac and maintains its attachments to the margins of the ring. Furthermore, the extent of the defect at the ring can be neither seen nor felt unless the finger is actually slipped through the rent just mentioned.

FIG. 2.—Showing appearance after the cord is lifted up with slight traction and the conjoint tendon is retracted outwards. The cremaster is omitted for the sake of clarity.

To expose the defect clearly and to display the margins of the ring, it is now necessary to divide the internal spermatic fascia all round the cord. It should be deliberately picked up with forceps and cut with scissors while gentle traction is exerted on the cord.

Slight difficulty may sometimes be experienced on the medial side of the cord, which is here tethered to the margin of the ring. The small cremasteric vessels are blamed for this by Hughes and Fathi. They stress the importance of these vessels, and state: "After the cremasteric vessels covered by their fascia have been exposed they are divided between silk ligatures. With the division of this 'ligament' the cord can be displaced laterally." However, I have never been able to expose them to my satisfaction, nor am I convinced that it is necessary to do so, because once the fascia has been divided the cord can be retracted laterally, and probably carries the vessels with it.

The stretched internal ring is now clearly displayed (Fig. 3), and usually admits the index finger easily. The cord is retracted laterally and the margins of the defect are approximated by interrupted sutures or a continuous suture medial to the cord. The newly reconstructed ring can in

FIG. 1.—Diagrammatic horizontal section through the internal inguinal ring.

FIG. 3.—Showing the stretched internal ring.

fact be made as small as desired and should fit snugly round the cord.

In some cases the ring has been so stretched that its lower fascial margin is practically non-existent. In such cases the sutures may be anchored to Poupart's ligament inferiorly as described by Edwards, but even then it is usually possible

with care to pick up with the needle a strand of tissue that will hold the stitches quite well just above Poupart's ligament and deep to the lower leaf of the cremaster.

Summary

The anatomy of the internal inguinal ring is reviewed.

The importance of a complete exposure of the stretched ring in operations for indirect inguinal hernia is stressed, and a simple method for ensuring this is described.

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Medical Memoranda

Parathion Poisoning

The increasing use of organic phosphorus insecticides has created a new occupational hazard to agricultural workers. Hunter (1950) has drawn attention to this group of compounds, which includes "parathion" (*para*-nitrophenyl-diethyl-thiophosphate), and has described casualties which have occurred elsewhere, although he noted that no cases of poisoning had been reported in Great Britain. Detailed information concerning organic phosphorus insecticides has been recorded by Goldblatt (1950), together with a description of the signs and symptoms of their toxic effects and the specific antidotal action of atropine. The case of parathion poisoning here recorded should serve to emphasize the nature of the risk, the importance of its prompt recognition, and the value of treatment with atropine.

CASE REPORT

A man aged 46 had been engaged for three weeks in preparing an insecticide spray for hops, using a commercial preparation containing 20% of parathion. The preparation was clearly labelled as to its poisonous nature, and workers were instructed to wear protective clothing.

On June 20, 1950, while preparing a lorry tankful of spray solution (24 oz. (680 g.) of the 20% preparation to 500 gallons (2,270 litres) of water), a drop of the 20% preparation splashed on to his forearm, covering an area about the size of a sixpenny piece. He washed it off with water within 30 seconds; this was at 4 p.m. Within a few minutes a raised weal appeared, but no further local injury. During the evening he remained well, took food, and smoked some cigarettes.

At 9.45 p.m. he experienced a sudden severe pain at the back of his head, spreading forward to the forehead. A few minutes later he vomited violently and developed persistent abdominal pain. This was soon followed by a feeling of weakness in the legs, after which he remembered nothing until the next morning in hospital.

He was admitted to Worcester Royal Infirmary at 11.25 p.m. Atropine, 1/60 gr. (1.1 mg.), had already been administered as a first aid, so that the pupils were dilated but reacting. The patient was very drowsy, sweating profusely, and appeared to have violent headache and abdominal pain; his pulse rate was 60 and of good volume; his blood pressure was 110/90; speech was slightly slurred; there was occasional muscular twitching in the upper and lower limbs; respirations were shallow but not distressed.

Dr. V. Keane (then house-physician) continued treatment with atropine sulphate, 1/75 gr. (0.87 mg.) intravenously; then 1/100 gr. (0.65 mg.) hourly for three doses; then two-hourly for two doses; then hypodermically hourly for three doses. In the morning the patient was rational and in the afternoon clinical examination showed him to be normal. He was discharged well on the fifth day after admission.

COMMENT

The patient's history, condition, and recovery on treatment with atropine are indicative of parathion poisoning. His statement indicates that he was aware of only trivial contact with the toxic material, but as parathion is a cumulative poison it seems likely that he had already absorbed and retained a harmful amount before the final intoxication, which was then further aggravated by smoking and by taking food.

The toxic action of parathion and related organic phosphorus insecticides is due to their inhibiting effect on the activity of choline esterase. Atropine is the specific antidote, but the order of dosage required may be much more than the usual therapeutic quantities. In this case a total of 11/100 gr. (7.1 mg.) of atropine sulphate was administered.

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Auricular Fibrillation After Electric Convulsion Therapy

Transient cardiac irregularities are a recognized complication of the various forms of shock therapy employed in the treatment of the psychoses. The following case is of interest because of the prolonged duration of the arrhythmia.

CASE REPORT

A man aged 54 was admitted to hospital in October, 1949, suffering from an agitated depressive state of approximately 12 months' duration. His main complaints were insomnia, inability to concentrate, depression, delusions of guilt and unworthiness, and a feeling which recurred often, and for no reason that he knew, of being urged to strike out at those around him. His blood pressure varied between 230/130 and 150/100, for which fluctuation emotional factors seemed to be largely responsible. No cardiac irregularity or enlargement, tachycardia, or other abnormal physical signs were present clinically.

Electric convulsion therapy was given on alternate days, and was terminated after eight shocks, there being considerable improvement in his mental condition. However, after the last convulsion he complained of palpitation, and on examination