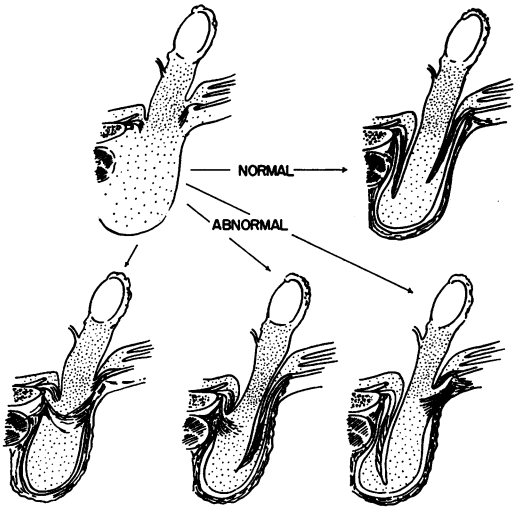

Fig 4 *A diagrammatic scheme to show the mechanism of production of an ectopic testis by fibrous invasion of the gubernaculum preventing distal growth of the processus vaginalis and cremaster muscle*

congenital adrenocortical hyperplasia in which full scrotal-like swelling occurs in females (Maxted *et al.* 1965) indicating marked gubernacular change of a male character. The actual stimulus to androsteroid production in the testis is still uncertain and, due to wide species variation, animal experiment is only of general value. But in view of the great rapidity in testicular retrogression after birth and little evidence of fœtal gonadotrophic hypophyseal activity in man (compared with the rabbit) it must be assumed to be primarily of maternal or placental origin; probably the latter is the more important.

Thus the changes of testicular descent constitute an initial establishment of the necessary components and a mesenchymatous path at embryonic stages followed by a burst of activity in the weeks prior to birth, under gonadotrophic stimulus of the interstitial cells of the fœtal testis, so producing the necessary androsteroid to achieve descent and final male secondary sex development. However, the descent process is dependent upon a free mesenchymatous pathway into which the processus vaginalis and cremaster muscles can extend their growth at the time of descent. If therefore the gubernacular mesenchyme is encroached upon by fibrous tissue, as when the scrotal wall is forming or for any other reason, then the processus vaginalis will be prevented from growing at that point, though elsewhere growth may be normal: when descent occurs quite normally under hormonal stimulus the system will be tethered and have a short processus vaginalis at that point, i.e. a 'tail of Lockwood' so that the testis will be unable to complete its normal descent and will swing out of place to become ectopic (Fig 4). A complete septum can exist across the external ring as a full manifestation of this process so preventing any descent beyond that point (Backhouse 1964).

The essential difference between a cryptorchid and ectopic testis from the anatomical point of view is thus primarily a hormonal failure on the one hand, which may be androsteroid deficiency, probably due to inability to convert pregnenolone to progesterone in the chain of synthesis or deficiency of target organ response, whereas the other is normally a mechanical derangement. The fact that the ectopic testis is often defective even after operation may be due to its having remained too long in its ectopic high temperature position or due to the very susceptible blood supply of a prepubertal testis, as Fahlström *et al.* (1963) have shown, which fails as a result of the necessary trauma of surgery.

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The Iliac Compression Syndrome *alias* 'Iliofemoral Thrombosis' or 'White Leg'

During recent years we had become interested in a group of patients coming to our vascular clinics with progressive severe disability of the leg, usually the left. These were the worst and most chronic type of post-thrombotic syndrome of the leg with swelling of the whole limb (thigh, calf and ankle), pain (usually worse on walking) and often progressive severe ulceration. All these patients had in common a previous history of

'white leg' or iliofemoral thrombosis, which usually occurred at a very young age (usually 20 years).

Instead of adequate recanalization of these big veins, patients appeared to be left with chronic severe obstruction of the main venous outlet of the limb (i.e. the iliac veins). It was not until we had investigated a series of these cases by the technique of pertrochanteric serial venography, which gave really adequate visualization of the iliac veins, that our attention became focused on the vena caval bifurcation as the probable site of the actual obstruction.

Analysis of the clinical data allowed us to define the clinical picture of this syndrome more accurately and it became apparent that there was usually an acute phase (the actual sudden episode of thrombosis with swelling) followed by a long chronic phase of deterioration of the leg. The characteristics of these two phases were as follows:

Acute phase: Age: young, usually between 18 and 30. Onset: sudden swelling of left leg, usually after an operation, pregnancy, major or minor illness. In a few cases both legs were involved and in still fewer the right only. Occasionally a single pulmonary embolus accompanies this phase.

Chronic phase: Lasts for the rest of the patient's life. (1) Chronic swelling and aching of the whole of the left leg. (2) Venous claudication: the whole leg aches and becomes intolerably congested when the patient walks and there is often pain in the calf. (3) A tendency to recurrent thromboses in the leg over the years. (4) Pigmentation and chronic ulceration of the ankle.

Investigation of a series of these cases by pertrochanteric venography (Cockett & Thomas 1965) showed that in most of them the thrombosis and partial recanalization of various degrees reached the vena caval bifurcation and there ended abruptly. It appeared that the essential obstruction in these cases was just where the right common iliac artery crossed over and presumably compressed the left iliac vein.

However, it was not until we started to explore the bifurcation at operation that the true state of affairs was revealed. When the caval bifurcation had been mobilized and the iliac artery lifted off the left iliac vein and out of the way, a local fibrous stricture was seen in the vein. Recanalization occurred to some extent up as far as the stricture and then stopped. This local fibrous stricture, which appeared to be caused by a local adherence of the anterior and posterior walls of the vein just where the iliac artery crossed it, was a complete or nearly complete bar to passage of blood from the left iliac vein to the cava. Up to date we have explored 18 of these cases and have found this lesion in 15.

Various types of operation are under trial to relieve this obstruction: so far the most successful has been a direct attack on the local compression band. This has been done either by vein patch angioplasty or, more recently, by direct excision of the local fibrous stricture and end-to-side anastomosis of the left iliac vein direct to the cava.

Our present concept of this syndrome is that the original lesion is a pressure fibrosis of the caval bifurcation, caused by varying degrees of compression between the aortic bifurcation and the lumbar lordosis.

The degree of obstruction produced by this lesion is very variable according to the relative arrangements of the two bifurcations; it is particularly liable to occur when the two bifurcations are unusually high, sited on the maximum convexity of the lumbar lordosis. When the obstruction is bad enough it predisposes to iliac thrombosis which then makes the obstruction much worse with its accompanying fibrosis.

In order to substantiate this hypothesis it would be necessary to find at post-mortem cases of this compression lesion, before thrombosis had taken place.

This lesion has already been described by Ehrich & Krumbhaar (1943) and by McMurrich (1906).

In a series of 12 post-mortem bifurcations examined by Mr David Negus at St Thomas' Hospital this pre-thrombotic lesion has been found in no fewer than 4.

Since describing this iliac compression syndrome in May of this year I have seen many more cases. I have now operated on 18 and we have investigated about 50 by venography. We now know that, far from being a rarity, this lesion is the common cause of what used to be termed 'iliofemoral thrombosis' or 'white leg'. It is an outstanding example of a slight aberration of anatomy being the basic cause of an important pathological lesion.

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