MUSCLE ACTION AND THE SHAPE OF THE FEMUR

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This study was promoted by the investigation over a period of fifteen years of the growth of a child stricken by extensive poliomyelitis of both legs. The changes in shape of the femora, together with the dislocation of one hip in this child, and observations on other patients developing such deformities, stimulated interest in the factors controlling the shape of long bones. The clinical observations are largely concerned with deformities of the upper end of the femur and subluxation of the hip seen to develop in patients under treatment. The experiments which these observations suggested are mostly concerned with the part which muscular imbalance may play in producing such deformities.

CLINICAL OBSERVATIONS

Some of the circumstances in which valgus deformity of the femur may develop and the hip joint subluxate are illustrated by the following three patients.

Case 1. Flaccid paralysis—A girl, four years old when she was first seen in 1947 with acute poliomyelitis, had residual flaccid paralysis which was complete in the right leg. In the left leg the iliopsoas was the only muscle which retained active power. Figure 1 shows the hip joints in 1949 when valgus deformity was starting on the left side and bone atrophy was evident on the right. No great discrepancy in leg length occurred and the child became mobile in walking calipers. The valgus deformity increased on the left side and also appeared on the right, but seven years later, in 1954, the left hip subluxated (Fig. 2), whereas on the right side, in which paralysis was complete, the head of the femur remained in the acetabulum. The subluxation on the left side was dealt with by a varus osteotomy.

Case 2. Spastic paralysis—This patient was recognised as suffering from spastic paraplegia within a year of her birth in 1949, since when she has been under continuous observation. She can now walk, dress and feed herself; her mental age is about five. Valgus deformity appeared and the right hip subluxated (Fig. 3) when the patient was ten years old. Reduction was obtained by adductor tenotomy and manipulation under anaesthesia and the hip was fixed in a plaster spica. The left hip (Fig. 4) dislocated with the same sequence of events during the period that the right one was in its plaster and was treated similarly. Later in 1960 this dislocation recurred and was dealt with by manipulative reduction and iliopsoas transfer.

Case 3. Inequality of leg length—A girl, nine years old, contracted tuberculosis of the left hip in 1940 and was treated conservatively for two years. Figure 5 shows a radiograph of her hip joints at that time. The left hip was then arthrodesed, but premature fusion occurred at the left lower femoral epiphysis and all growth in length ceased in this limb by the age of twelve years. It proved impossible to equalise the legs although three inches were gained by a lengthening operation on the left side. Thereafter a valgus deformity appeared in the right femoral neck and when the patient was eighteen years old the right hip subluxated (Fig. 6). This was treated by a displacement osteotomy.

In these three patients it seems that the common factor associated with the development of valgus deformity, culminating in subluxation of the hip, was muscular imbalance resulting in an overriding force predominating at the lesser trochanter.
Fig. 1
Case 1—Radiograph of the hips of a six-year-old girl two years after the onset of poliomyelitis. A valgus deformity is starting on the left, and bone atrophy on the right.

Fig. 2
Case 1—Five years later the left hip is subluxated. No subluxation occurred on the right side where paralysis was complete. Both femoral necks show valgus deformity.
Case 2—Radiograph of the hips of a girl aged ten years with life-long spastic paralysis, showing valgus deformities and subluxation of the right hip.

Case 2—After reduction of the right hip, and while in a plaster spica, the left hip subluxated.
Case 3—Radiograph of the hips of a girl aged eleven years, two years after the onset of tuberculosis of the left hip.

Case 3—Seven years after arthrodesis of left hip, a valgus deformity has occurred in the right femoral neck and the hip has subluxated.
Case 4—Radiographs of an intertrochanteric fracture, treated by external fixation, in which union occurred with a varus deformity. The lesser trochanter was avulsed.

Case 5—Radiographs showing an intertrochanteric fracture with avulsion of the lesser trochanter which was treated by internal fixation. Union has occurred in varus.
**Coxa vara**—The opposite femoral deformity of coxa vara can also be seen to develop in patients under observation suffering from other disabilities. Many intertrochanteric fractures of the femur unite in normal position without trouble after reduction and fixation. But if the lesser trochanter has been coincidentally avulsed at the original injury and remains displaced, deformity occurs whatever the method of treatment, as illustrated by the following three patients.

**Case 4**—A man of fifty-seven had an intertrochanteric fracture of the left femur; his general condition precluded any form of operation. Although reduction was obtained union was in coxa vara (Figs. 7 and 8).

**Case 5**—This patient had the same type of fracture, in which coxa vara developed in spite of internal fixation (Figs. 9 and 10).

**Case 6**—A third patient had an intertrochanteric fracture of the femur with avulsion of the lesser trochanter. The fracture was reduced and internal fixation secured reasonable position, but this did not stop the development of a coxa vara deformity with complete disruption of the nail and plate (Figs. 11 and 12).

It is emphasised that when the avulsed lesser trochanter is reattached as part of the internal fixation, coxa vara does not then develop.

**EXPERIMENTAL OBSERVATIONS**

In order to visualise the characteristic shape which some muscle forces acting on the femur tend to imprint on it in life, the following experiments were made.
MATERIALS AND METHODS

A small box frame was constructed and covered over to form a laminated wood table which was firmly clamped down to a bench in order to support the skeletal parts studied. A dried right hip bone was securely fixed to the table with screws, supporting blocks and wood plastic in such a way that femora fitted to the acetabulum and maintained roughly parallel to the horizontal could be photographed anteriorly and laterally. The plane of the pelvic inlet was, therefore, horizontal although normally in man it is at an angle of 60 degrees. This enabled the head and neck of the femur to be photographed from in front without being obscured by an overhanging ilium. At the same time it would be in a position of approximately 30 degrees of flexion, a reasonably average position in which to record its response to applied forces.

Right sided cadaveric femora were stripped of the main bulk of soft tissue, only certain muscle attachments being left in situ, and decalcified in nitric acid and stored in formalin. The attachments of the tendon of iliopsoas to the lesser trochanter, the glutei to the greater trochanter, and adductors to the linea aspera and adductor tubercle were preserved to provide a junctional tissue between applied forces and the bony areas on which they acted. This arrangement made certain that the applied forces were acting as nearly as possible in the same lines as those of the natural forces. In this connection it is pointed out that the bulk of the fibres of the iliopsoas tendon are attached to the posterior surface of the lesser trochanter, many of the fibres running in the periosteum before finally sinking into the superficial lamellae.
Experiment I—Anterior view of a femur with 25 kilograms psoas force acting. The head has been pushed upwards and backwards in the acetabulum and a valgus deformity has been produced. The lesser trochanter has moved anteriorly, an indication of the lateral torsion of the upper shaft with this deformity.

Experiment I—Lateral view of a femur undergoing psoas stress. When compared with Figure 13, posterior deflection of the head and anterior bowing of the upper shaft are seen to have occurred.

The true dispersion of mechanical force at the lesser trochanter would not be simulated by a tension wire simply affixed to this point, and it was felt desirable to let natural attachments be the final transmitters of applied forces.

Tendon and muscle sutures were made on the adherent iliopsoas, abductor and adductor attachments using No. 191 wire (Zimmer Ltd.) and the head of the femur was placed in the acetabulum. The wires were carried away from the experimental table and passed over pulleys supported on retort stands and weights then added. A counterpoise of 0·5 kilogram was applied longitudinally to the lower end of the femur in order to give the bone stability, but otherwise it was freely movable. Anterior and lateral views of the femora were taken both before and during the application of forces, each bone providing its own visual control records for assessing the type of bony deformation resulting. Observations were made on ten decalcified femora undergoing varying types of experimental strain.

RESULTS

Experiment 1. The effect of iliopsoas action alone on femoral shape—The head of the femur was placed in the acetabulum and a force of 0·5 kilogram applied to the lesser trochanter. This, with the longitudinally acting counterpoise was sufficient to hold the decalcified bone parallel to the table, but no deformity resulted (Figs. 13 and 14).

Depending on the degree of hardening caused by formalin storage, weights varying from 15 to 25 kilograms applied to the iliopsoas attachment produced considerable deformity in
the decalcified femur. Seen from above (Figs. 14 and 15) the head of the bone was pulled anatomically upwards and backwards and the line of the neck approximated with that of the shaft, and a valgus position was taken up. In this process fat was squeezed out of the vascular foramina in the neck of the femur as it changed its shape. That this untwisting, or lateral torsion, of the bone extended at least into the upper shaft was shown by the lesser trochanter—which is normally postero-medial—adopting an antero-medial position, although no corresponding lateral rotation of the condyles occurred.

In lateral views, which were taken with the two condyles superimposed, posterior deflection of the head and neck was very apparent (Fig. 16) when compared with the control views of

the same bone (Fig. 13). Lateral views also revealed an increase in the natural anterior curvature of the femoral shaft—most marked in the upper third—when forces were applied to the lesser trochanter. On removal of the iliopsoas force the bone returned to its normal shape.

Thus the action of iliopsoas alone on the shape of the decalcified femur can be summarised as causing a valgus deformity, a posterior deflection of the neck, an untwisting of the upper diaphysis and an increased anterior curvature of the shaft.

**Experiment 2.** The effect of gluteal action alone on femoral shape—No apparent deformity of the femur resulted when the decalcified bone was suspended from the apparatus by 0.5 kilogram weights attached to the greater trochanter and lower end of the bone respectively. Deformation was, however, more readily produced by gluteal action on the greater trochanter than by the pull of iliopsoas. Weights of 9 to 15 kilograms produced a considerable varus deformity of

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*FIG. 17*

Experiment 2—Anterior view of the upper end of a femur acted on by a force of 15 kilograms at the greater trochanter. The varus deformity is marked. The lesser trochanter is situated posteriorly, which indicates that medial torsion of the shaft is caused by unbalanced gluteal action.

*FIG. 18*

Experiment 2—Lateral view of a femur acted on by 15 kilograms gluteal force. The head and neck are deflected anteriorly and bowing of the shaft has been abolished.
the femoral head and neck (Fig. 17), while at the same time the natural twist of the neck and upper part of the shaft was increased, with the lesser trochanter taking up a more posterior position than normal. The normal anteverision of the head and neck was also increased, the natural anterior curvature of the shaft was abolished, and the diaphysis was rendered straight (Fig. 18).

**Experiment 3. The balancing of iliopsoas and gluteal action on the femur**—In the above experiments it was apparent that the deformities produced by the iliopsoas were the converse of those caused by gluteal action. To demonstrate this further, iliopsoas and gluteal forces that, acting alone, were known to produce severe deformities in femoral shape, were applied together. It was found that a force of 25 kilograms acting at the lesser trochanter was balanced—in so far as its effect on femoral shape was concerned—by a force of 15 kilograms acting on the greater trochanter. Figures 19 and 20 show a femur undergoing these stresses. Its shape is the same as in Figures 13 and 14, when no forces were applied. It is therefore concluded that the tendency to femoral deformity produced by an iliopsoas force is abolished by a gluteal force acting at the greater trochanter when these are in a ratio of 5:3.

**Experiment 4. The effect of adductor forces on the shape of the femur**—Besides the 0.5 kilogram counterweight at the lower end of the bone, 0.5 kilogram must be applied to the greater trochanter in order to hold the head in the acetabulum, otherwise heavy weights attached to the adductor group pulled the shaft of the bone transversely across the body of the pubis, thus vitiating the experiment. Figures 21 and 22 show the results of applying 25 kilograms to the adductor muscle group. Besides an obvious medial deflection of the shaft, varus deformity has occurred. This was accompanied by medial torsion of the upper portion of
Experiment 4—Anterior view of femur acted on by 25 kilograms adductor force, showing medial bowing of shaft, and varus deformity of the head and neck. Medial torsion of the shaft is indicated by the disappearance posteriorly of the lesser trochanter.

Experiment 4—Lateral view of femur with 25 kilograms applied to adductor muscle group. The natural anteverision of the head and neck has been almost entirely abolished.

The bone as shown by the lesser trochanter disappearing when viewed anteriorly. The condyles, both before and during the application of forces in this experiment, were kept resting on the table, thus precluding lateral or medial rotation of the bone as a whole as an explanation of the change in position of the lesser trochanter. Medial torsion of the upper part of the femur was accompanied by a diminution of the normal anteverision of the neck (Fig. 22), although true posterior deflection was not seen.

**DISCUSSION**

The clinical observations show that valgus deformity of the femoral neck can and does occur in bones which were previously normal on radiological examination, and that this deformity can develop in the presence of muscular imbalance in which the iliopsoas predominates; it can also be caused by flaccid paralysis, spastic paralysis, or gross skeletal deformity without any paralysis at all. Dislocation of the hip may be a late sequel to the valgus deformity that follows derangement of muscle function.

When there is an abnormal preponderance of muscular action at the greater trochanter the opposite deformity of varus can develop in a previously radiologically normal hip.

The clinical findings were confirmed and amplified by the experiments, which showed that an iliopsoas force acting at the lesser trochanter of a decalcified femur caused an immediate valgus deformity, posterior deflection of the neck and lateral torsion of the shaft with an
increase in its anterior convexity. Conversely a gluteal force at the greater trochanter caused coxa vara and anterior deflection of the neck with medial torsion and straightening of the shaft. Adductor action caused medial deflection and torsion of the shaft with some varus deformity of the neck of the femur.

It is well known that antero-posterior radiographs of the normal hip can be made to show an apparent valgus deformity by rotating the leg. Pseudo-valgus can be made to disappear by suitable change of position during radiography. It is conceded, however, that such a valgus deformity may be present although the patient's knees have been correctly orientated, and in these instances it is presumably the result of an extreme degree of anteversion of the femoral head and neck, which is extremely variable (Frazer 1940), and which is to be distinguished from lateral torsion of the upper part of the femur with which the lesser trochanter is positioned more anteriorly. In the clinical records presented here, pseudo-valgus cannot be upheld because each patient showed no valgus deformity when first seen; the deformity developed over a period of time. The experiments furthermore produced a directly visible valgus deformity by application of an unbalanced force to the lesser trochanter as was suggested clinically. It must therefore be concluded that valgus deformity developing in life in a previously normal bone is a true entity and not a radiological artefact, that it is accompanied by other alterations in bone shape, and that it is the result of iliopsoas preponderance. Similar reasoning shows that coxa vara is also a real deformity, even though it is only usual to see one of its characteristics, the diminution in the neck-shaft angle in radiographs; one way in which it develops is as a result of unbalanced gluteal action at the greater trochanter.

The sparse literature on dislocation of the hip in flaccid palsy is noteworthy for insistence on the fact that the deformity does not occur in a completely paralysed limb (Bradford 1883, Sever 1911, Jones 1920). Case 1 recorded here confirms this. Elmslie (1920) went so far as to suggest that it probably occurred when poliomyelitis affected an already congenitally dislocated hip. Valgus deformity is of common occurrence in this condition. Moreover, Blundell Jones's (1954) important contribution to the treatment of flaccid paralytic dislocation of the hip is based on the correction of valgus deformity by osteotomy. Dislocation of the hip occurs in spastic paralysis as often as in flaccid states (Watson-Jones 1952, Blundell Jones 1954, Somerville 1959) and, from the evidence given here, is preceded by valgus deformity caused by iliopsoas preponderance, and can be treated by iliopsoas transfer.

It is likely that dislocation can arise in the absence of paralysis when muscular imbalance is caused by mechanical factors. For instance, in inequality of leg length the pelvic slant presents the glutæi with a considerable adduction of the hip joint in the longer limb which they must overcome before starting abduction; this places them at a mechanical disadvantage with respect to the iliopsoas. It is characteristic that a valgus deformity preceded the dislocation in the patient with this condition recorded here.

In these three types of hip dislocation the femoral head is found lying posterior to the acetabulum. To reach this position it has to push its way into the massive, fleshy origins of glutæus minimus and medius on the lateral iliac surface. The clinical observation that, in disorganised muscle function, iliopsoas preponderance is the probable cause of posterior dislocation of the hip is confirmed by experiment, because unbalanced action at the lesser trochanter not only produces a valgus deformity but also causes the femoral head to point posteriorly and thus tend to subluxate into the gluteal fossa.

The action of muscles on bone shape raises some interesting genetic considerations. It seems that the genetic systems that determine the appearance of a bone also endow it with the inherent plasticity which is such a notable characteristic of osseous tissue (Harrison 1960). Viewed from the evolutionary standpoint, muscle action and mechanical factors generally become of great importance in imprinting shape on bone because their effects can be fixed in the genotype by the process of genetic assimilation (Waddington 1957). In this way natural
selection has been able to adapt bone form to the mechanical influences which will be present in life, and the shape of a femur, for example, may still develop to a considerable extent in the complete absence of muscle pull (Willis 1936).

Nevertheless, the shape that a bone possesses represents a compromise in expression of extrinsic as well as intrinsic factors. Biochemical factors may modify the shape and disposition of foetal bones (Duraiswami 1950, Fraser and Fainstat 1951, Kalter and Warkany 1957, Dagg 1960) or may even suppress a bone's appearance altogether (Russell 1948). The absence of muscle pull can lead to failure of development of the canine lesser trochanter (Tower 1937), which has its own osseous centre and presumably intrinsic representation. The results presented here suggest that unbalanced muscle action can also alter bone shape to a considerable extent in human development, because bone, like any other substance, will deform in response to an applied force, in spite of inherent growth tendencies. The results permit the possibility of defining the shape which any particular muscle group tends to imprint on bones, and suggest that the cause of certain congenital bony malformations may reside not in disturbances of purely intrinsic morphological mechanisms, but in derangement of muscle balance from whatever cause arising in foetal life. It is recognised that the way in which a bone deforms in these circumstances is not merely a case of bending alone—as in the experiments—but probably involves remodelling mechanisms as well. Another factor promoting change in bone shape may be a disturbance of the haemodynamics of bone in conditions of muscular imbalance, because it seems likely that the venous drainage of bones is affected by the activity of neighbouring muscles (Brookes 1958; Brookes, Elkin, Harrison and Heald 1961), thus disturbing the nutrition of bone substance. If bone is indeed rendered partially ischaemic by impeding the venous return, bone removal and osteoporosis are to be expected (Brookes 1960a and b), with the softened bone thus rendered more susceptible to distortion by abnormal forces.

SUMMARY
1. Femoral neck deformities that developed in patients under clinical observation are described.
2. Experiments made on ten decalcified femora produced similar deformities.
3. The two sets of observations are correlated and discussed with reference to the role of muscular imbalance in the causation of deformities of the femoral neck.

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