THE BLOOD SUPPLY OF THE GREATER TROCHANTER

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We perfused 16 human femora with a 50% barium sulphate suspension and studied the intra-osseous vessels by microfocal radiography and histology. There were few anastomoses between the vessels of the greater trochanter and those of the adjacent cancellous bone of the shaft. Ischaemia of the trochanter may contribute to nonunion after trochanteric osteotomy.

Detachment of the greater trochanter is an excellent method of exposure of the hip (Charnley and Ferreira 1964), but it has a considerable morbidity and is now reserved for difficult or revision surgery (Schutzer and Harris 1988). Complications have been reported in 4% to 26% of cases and include bursitis, nonunion, breakage of the osteotomy fixation device and proximal displacement of the trochanter with weakening of the hip abductor mechanism (Charnley 1972; Amstutz and Maki 1978; Pellicci, Wilson and Sledge 1982; Nutton and Checketts 1984). Improvements in the method of reattachment (Harris and Crothers 1978; Schutzer and Harris 1988) attempt to overcome these problems.

In addition to mechanical considerations, however, biological factors may also be important. Amstutz et al (1982) found that osteoporosis and a poor bone bed for reimplantation contributed to displacement. Bone necrosis from impaired blood supply at a fracture site may be associated with delayed healing and slow revascularisation (Catto 1976; McKibbin 1978).

There have been few specific studies of the blood supply of the greater trochanter. Our aim was to demonstrate its vascular connections in man.

MATERIALS AND METHODS
We selected 16 cadavers after post-mortem examination within 48 hours of death on the basis of freedom from disease likely to affect either the long bones of the lower limb (prolonged immobilisation, steroid treatment, previous hip surgery) or the peripheral vascular system (atherosclerosis, diabetes mellitus). The subjects were of either sex with an age range of 19 to 95 years.

We performed vascular perfusion via the common iliac artery using a pressure-regulated pump after thorough warming of the limb to remove post-mortem tissue stiffness. The vessels were flushed with warm water and then with 4 l of hot 50% barium sulphate suspension. The femur was removed intact with a thin muscle covering, fixed in formol saline and decalcified in 5% nitric acid solution.

The proximal third was then sectioned in the coronal plane to give slices 5 mm in thickness. Microfocal radiographs were taken of the sections using a Hilger-Watts X-ray generator and fine-grain, non-screen film.
(CEA Singul-x) to produce clear magnified images of the vessels. Paraffin-embedded sections were cut at 10 μm and stained with haematoxylin and eosin for correlative histology.

RESULTS
Arterial vessels entered the greater trochanter from its medial, lateral and superior surfaces. Very free anastomoses between these penetrating arteries provided a rich vascular network within the cancellous bone of the trochanter (Fig. 1). A striking observation, however, was the separation of this trochanteric network from the vessels of the femoral neck and shaft. A line of demarcation was seen with few anastomoses between the diaphyseal and trochanteric vessels (Figs 1 to 4). This line of separation ran obliquely downwards and laterally from the trochanteric fossa to the quadrate tubercle, close to the site of the former epiphyseal growth plate of the trochanter.

DISCUSSION
The greater trochanter is a traction apophysis. It develops in cartilage with a centre of secondary ossification appearing at about two to four years of age and fusing with the metaphysis at maturity.

The mechanically different pressure epiphyses have a blood supply separated from the vessels of the metaphysis, until closure of the epiphyseal plate. After closure, some anastomoses between epiphyseal and metaphyseal intra-osseous arteries and veins are seen (Brookes 1971).

Previous studies have demonstrated the blood supply to the proximal femur (Trueta and Harrison 1953) and to the trochanteric epiphysis in the child (Crock 1965). Our results suggest that, in the adult, the greater trochanter has a separate blood supply even after bony fusion with the shaft. There are few anastomoses with the adjacent diaphyseal vessels and a relatively avascular plane separates the two circulations.

We used a well-established perfusion technique for human cadavers (Trueta and Harrison 1953; Bridgeman and Brookes 1990), but the proportion of vessels filled with barium may vary between subjects. Histological findings of the degree of vascular filling and the type of vessel, however, allow a more accurate interpretation of the radiographs. Since the pattern of vessels described was seen in all our 16 subjects and intra-osseous anastomoses were clearly demonstrated in the same femora between epiphyseal and metaphyseal arteries in the femoral head, it seems unlikely that the absence of anastomoses between the greater trochanter and shaft is artefactual.

The greater trochanter must therefore rely heavily on extra-osseous vessels for its blood supply. Howe, Lacey and Schwartz (1950) demonstrated this supply in a series of elegant dissections. From branches of the circumflex femoral vessels, which encircle the trochanter, penetrating vessels enter it on its anterior, lateral and posterior surfaces. The medial circumflex femoral artery also supplies branches which enter the trochanter medially in the trochanteric fossa. Branches of the gluteal vessels enter the bone at the insertion of the gluteus medius.

Trochanteric osteotomy usually runs close to the avascular plane and probably severs branches arising from the circumflex femoral arteries. The osteotomised
trochanter will thus rely solely on the blood supply from the gluteal vessels. If the trochanter is rendered ischaemic, as we suggest, subsequent revascularisation will be inhibited if the site of reattachment consists of a small area of osteoporotic cortical bone. Damage to the diaphyseal vessels by reaming or cement implantation will also delay revascularisation. The avascular plane which we report is likely to be another barrier.

We suggest that trochanteric ischaemia may be an important factor in the failure of union, and that methods of promoting revascularisation could be of considerable value in reducing its incidence.

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