Antenatal pressure necrosis of thoracoabdominal wall

Sir,

We read with great interest the report of Dr. L. K. Sharma of a case of 'Congenital skin aplasia affecting trunk' (*Archives*, 1973, **48**, 813), as we were preparing a very similar case report but had reached a different conclusion about the cause.

The mother, a primigravida aged 22 years, had an uneventful pregnancy though she smoked 20 cigarettes per day throughout. At term, an erect lateral pelvic x-ray showed a breech presentation with extended legs. After artificial rupture of the membranes, a normal breech delivery ensued. There was a large placenta (725 g) and a previously undiagnosed fetus papyraceous. The baby, a male weighing 3.15 kg, had bilateral symmetrical skin and subcutaneous loss over the lateral part of the lower costal margin and upper abdomen (Fig. 1). The periphery of these areas showed complete

FIG. 1.—Thoracoabdominal skin defects on the 3rd day of life. (a) Right side, (b) left side.

healing and scarring which obviously had already taken place *in utero*. Epithelization was completed 3 weeks after birth and the only treatment was local applications of Aserbine cream and framycetin (Soframycin) gauze dressings.

In our patient the area of skin loss was centred around and followed the course of the costal margin. We suggest that the skin lesions were due to pressure necrosis and the area was compressed by the flexed arms being held tightly against the costal margin by the extended legs of a breech presentation in a primiparous woman. When the baby was placed in this intrauterine position, the arms filled the skin lesions (Fig. 2). In view of the

FIG. 2.—Patient held in intrauterine position showing arm filling skin defect.

fact that healing had occurred *in utero* the pressure must have been released before birth.

We find the other explanations discussed in Dr. Sharma's report unacceptable in our case and one can visualize the same pressure occurring in vertex delivery or flexed breeches to account for thoracoabdominal skin defects. These lesions could well have a different aetiology from the more common midline scalp defect and to call them all skin aplasia and seek a common aetiology is misleading—hence our title.

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