A 45 year old woman presented with a 4 year history of fever attacks, generalised fatigue, arthralgias, and recurring painful nodules of the subcutaneous tissue in different localisations. Extensive diagnostic procedures had been carried out in several rheumatological and dermatological departments of university hospitals (13 admissions). Under the diagnosis of nodular panniculitis and because of the deep infiltration, incisions and surgical debridement were often necessary. At the physical examination she had a temperature over 39°C, an approximately plum sized painful nodule in the subcutaneous tissue of the left shoulder, and ulcerations of the left hand and elbow (after surgical debridement) (fig 1).

Laboratory tests showed a raised white cell count of 11.6 x 10⁹/l; an erythrocyte sedimentation rate of 55 mm/1st h, and C reactive protein 215 mg/l. Transaminases were increased, with a γ-glutamyltransferase of 154 U/l, alanine aminotransferase 53 U/l, and aspartate aminotransferase 38 U/l. There were no signs of autoimmunity, including detectable autoantibodies. Histological examination showed a severe purulent inflammation with multiple colonies of bacteria localised in the connective and fatty tissue.

Incidentally, by cleaning the patient’s bedside table we found a needle, a syringe with yellow-brown fluid, a wet shaver, and a destroyed mercury thermometer (fig 2). Analysis of the content of the syringe showed traces of lidocaine, non-organic acid salt, blood components, and bacteria. We confronted the patient with the question of artificial lesions, which she did not deny, but psychological support was not wanted. We learnt that this patient was admitted to hospital some weeks later again in another surgical ward for intervention and reinvestigation of the nodular panniculitis.

COMMENT
Factitial panniculitis (panniculitis artefacta) is caused by self injection of different substances designed to produce an acute inflammation, often with systemic disease.1 2 It can mimic other autoimmune diseases.3 Panniculitis is one possible expression of self induced skin diseases.4 The major underlying theme is an attempt to alter or escape from an unsatisfactory situation by calling attention to oneself as a patient, victim, or bearer of a mysterious problem. The patients have psychosomatic or severe psychiatric problems.5 In every patient this can be viewed as a cry for help. In our case there was evidence of an underlying sexual conflict.

A final scenario is Munchausen’s syndrome first described by Asher 1951, named after a German folklore character “Baron Münchhausen” who told fantastic stories.6 Patients with this syndrome create a complex disease picture, usually with a lengthy history, and visit many doctors and hospitals, seeking help but often leaving suddenly and with anger. They may undergo multiple dangerous operations for diagnostic reasons; in our case it took 14 admissions to discover that the inflamed nodules were artificially induced.

Therapeutic options are limited and the longer term approach is not satisfactory. The acute situation should be treated with antibiotics to cover a mixed range of organisms. Lesions that fail to respond to treatment or recur despite appropriate measures should arouse suspicion. Often an affected area can be occluded for a week with a bandage to see whether there is improvement. None the less, the social and psychiatric care should be offered.7 Regrettably, as demonstrated in our case, these offers will usually be rejected by patients with Munchausen’s syndrome. In most instances, to improve the prognosis, it is felt appropriate to report these
patients to other colleagues, hospitals, and social services in order to prevent the patient from undergoing unnecessary testing or procedures.5

REFERENCES

Ligamentous laxity and rugby injuries

Joint laxity may be an advantage in sports such as gymnastics but a disadvantage in physical contact sports. Greater laxity has been shown to increase the risk of knee injury in professional footballers but not in high school players. A study in New Zealand has related ligamentous laxity to injury in amateur, first division club, rugby players.

Fifty one players were examined using a modified Beighton-Horan nine point scale (0 or 1 point for each of: passive opposition of thumb to flexor aspect of forearm (two sides), passive hyperextension of little finger at metacarpal phalangeal joint beyond 90˚ (two sides), elbow hyperextension to 15˚ or greater (2 sides), knee hyperextension (two sides), and palms flat on floor with knees extended). A stiff limb joint due to previous injury was recorded as lax if the uninjured joint was lax. Three categories of overall laxity were used: tight (score 0–3), hypermobile (4–6), and excessively hypermobile (7–9). Thirty nine players were classified as tight, eight as hypermobile, and four as excessively hypermobile. Strength testing in hamstrings and quadriceps was performed in nine players in the tight group and nine others.

The mean laxity score was 2.0. Over the rugby season there were 31 shoulder, hip, knee, ankle, wrist, or hand joint injuries in 23 players. Injury rates were similar in the three defined laxity groups but when the two hypermobile groups were combined (scores 4–9) they had a significantly higher injury rate than the tight group (116.7 injuries/1000 playing hours v 43.6/1000 playing hours). There was a non-significant trend towards greater strength in quadriceps and hamstrings in the tight group. Among the hypermobile group greater strength was not associated with fewer injuries.

Ligamentous laxity may increase the risk of injury in first division club, rugby players. (The effect of playing position was not assessed in this study.) The injury rate among these hypermobile amateur players was similar to that previously reported in professional rugby union players (120/1000 playing hours). The authors of this paper suggest that young athletes who are hypermobile might be advised to take up sports in which hypermobility could be an advantage.

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