Mean Adipose Tissue Fatty Acid Composition for Newborn Infants (g/100 g $\pm$ 1 S.D.)

Fatty Acid	London (healthy, all low birth weight, n = 16)	Widdowson <i>et al.</i> ¹		Hashim and Asfour ⁴ (normal, n = 15)	King <i>et al.</i> ³ (normal, term, n = 5)	Baker ⁵ (term, post- mortem, n = 5)
		Dutch (healthy, some low birth weight, n = 12)	British (preterm, post- mortem, n = 14)			
C14:0 (Myristic) ..	4.4 $\pm$ 0.9	3.3 $\pm$ 0.4	3.8 $\pm$ 0.6	3.2 $\pm$ 0.8	4.4 $\pm$ 0.8	3.3
C16:0 (Palmitic) ..	44.5 $\pm$ 2.7	45.8 $\pm$ 1.6	48.9 $\pm$ 3.6	52.0 $\pm$ 3.5	48.4 $\pm$ 5.4	38.6
C18:0 (Stearic) ..	4.4 $\pm$ 1.0	3.8 $\pm$ 0.4	4.1 $\pm$ 0.6	4.1 $\pm$ 0.8	5.7 $\pm$ 2.3	4.5
C16:1 (Palmitoleic) ..	14.7 $\pm$ 2.0	15.2* $\pm$ 1.2	12.6* $\pm$ 1.6	9.5 $\pm$ 1.1	13.3 $\pm$ 3.0	15.9
C18:1 (Oleic) ..	29.7 $\pm$ 1.6	29.0 $\pm$ 1.8	29.6 $\pm$ 3.0	30.8 $\pm$ 1.9	24.8 $\pm$ 2.5	33.5
C18:2 (Linoleic) ..	2.3 $\pm$ 0.9	2.9* $\pm$ 0.7	1.0* $\pm$ 0.8	0.8 $\pm$ 0.7	1.1 $\pm$ 1.1	2.5 (no S.D.s given)

*P < 0.001

leagues. We have no data on the fatty acids in the mothers of our infants, but in general these women belonged to the local working-class community around Hackney. We believe they were unlikely to consume much polyunsaturated fat, which was one suggested reason for the higher linoleic acid (C18:2) levels found in the babies of Dutch mothers. There are, however, differences both in adipose tissue sampling and in chemical methodology. The Dutch babies and our London babies had adipose tissue samples taken by needle biopsy soon after birth, whereas all except one sample of Dr. Widdowson's "British group" were from infants who had died perinatally. When one compares the values obtained in various American studies there are striking differences between the results which might, in part at least, reflect differing methodologies, whereas within the same study comparison of differing clinical groups of infants has usually not shown up chemical differences (for example, the study of King *et al.*³).

There is, then, some variation in the reported fatty acid composition of adipose tissue at birth. This may reflect true biological variation but we feel that at least part of the variation is due to differing methodology.—We are, etc.,

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¹ Fosbrooke, A. S., and Wharton, B. A., *Archives of Disease in Childhood*, 1975, in press.

² Fosbrooke, A. S., and Tamir, I., *Clinica Chimica Acta*, 1968, 20, 517.

³ King, K. C., *et al.*, *Pediatrics*, 1971, 47, 192.

⁴ Hashim, S. A., and Asfour, R. H., *American Journal of Clinical Nutrition*, 1968, 21, 7.

⁵ Baker, G. L., *American Journal of Clinical Nutrition*, 1969, 22, 829.

Scabies in a Spinal Injuries Ward

SIR,—I was recently asked to investigate an outbreak of irritating papules on the forearms and thighs of nursing staff in a spinal injuries ward. The eruption found in the case of eight nurses resembled that seen among ward staff in epidemics caused by cases of Norwegian scabies. No burrows could be found nor were mites isolated. Examination of the 20 patients in the ward revealed that five were heavily infested with *Sarcoptes scabiei*. All these patients had suffered fractures of cervical vertebrae 4, 5, 6, or 7 with consequent loss of cutaneous sensation.

Paterson *et al.*¹ writing on the development of Norwegian scabies during immunosuppressive therapy tabulated the aetiological factors so far reported in cases of

Norwegian scabies, among which was lack of cutaneous sensation occurring in leprosy, syringomelia, and tabes dorsalis. The scabies cases in this ward epidemic, though all presenting a massive infestation, in one at least amounting to many hundreds of mites, did not show the picture of Norwegian scabies. The trunk and limbs had profuse scattering of erythematous papules each surmounted by a typical burrow. There was, however, no hyperkeratosis or crusting. One young adult had multiple palmar burrows with minimal erythema similar to that frequently seen in very young infants. I suggest that the loss of cutaneous sensation following fracture of cervical vertebrae allowed unrestricted multiplication of mites owing to lack of itching and therefore scratching and that this tends to confirm Mellanby's observation² that "in man it is the active finger nails of the host which keep down the parasite population." The duration of the infestation was unknown, probably only a few months, and this was presumably too short to allow the development of the hyperkeratosis and crusting seen in true Norwegian scabies. The mites, however, were sufficiently numerous to result in a ward epidemic involving nursing staff. It was only this involvement of staff with intact sensation that led to detection of the infestation.

Treatment with 1% gamma benzene hexachloride (Quellada lotion) was rapidly effective with no case of failure to respond.—I am, etc.,

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¹ Paterson, W. D., Allen, B. R., and Beveridge, G. W., *British Medical Journal*, 1973, 4, 211.

² Mellanby, K., *Scabies*. Hampton, Middlesex, Classy, 1972.

Operator/Anaesthetists

SIR,—With the declaration of the President of the General Dental Council on the subject of operator/anaesthetists (24 May, p. 453) dentists now know that they will be judged to be guilty of infamous professional conduct if they act in that capacity.

Will the B.M.A. approach the General Medical Council for a ruling relating to doctors acting as operator/anaesthetists? While this practice is not widespread, it certainly does happen—for example, psychiatrists performing electric convulsion therapy and thoracic surgeons performing bronchoscopy under intravenous barbiturate plus a muscle relaxant, physicians performing gastroscopy under intravenous anaes-

thetic doses of diazepam, general practitioners opening abscesses etc. under "a whiff of gas." Even the anaesthetist himself assumes the dual role if he first anaesthetizes the patient then proceeds to the rear to perform caudal or lumbar epidural puncture and catheterization.—I am, etc.,

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Practolol, Indoramin, and Asthma

SIR,—I.C.I. have recently circulated new prescribing information about practolol recommending that in view of its side effects its use should be limited to patients with organic cardiac disease and those with asthma or bronchitis.

Practolol is cardioselective and is less likely to cause bronchospasm than other beta-blockers, but patients with asthma often do get a troublesome increase in wheeziness if given the drug. We have recently been looking at the effect of combining practolol with the alpha-adrenergic blocker indoramin hydrochloride in patients with asthma and angina to see if pretreatment with the alpha-blocker would prevent the effect of beta-blockade on the bronchial tree.

Six patients suffering from reversible airways obstruction and angina were studied. They were observed for one control week, then given one week on practolol 100 mg twice daily, one week on indoramin 20 mg three times daily, and one week on a combination of the two drugs. They kept diary cards and measured their own peak expiratory flow rate (P.E.F.R.) twice daily throughout. Four of the six complained of increased wheezing with practolol and two had to stop the drug. The average drop in P.E.F.R. for all six was 11% (range +3% to -37%). During the week on indoramin alone there was subjective improvement in both asthma and angina. When practolol was reintroduced the four patients who had complained of wheezing before were again affected and all four stopped the drug. The average drop in P.E.F.R. compared with control was 19% (range -6% to -50%).

The combination of these drugs appeared to make the bronchospasm worse. A possible explanation is that with blockage of alpha and beta adrenoceptors the cholinergic innervation was left unopposed.

Little has been reported about increased airways obstruction in asthmatics on practolol since the early studies,¹ but clinical experience and the observations above show that it may be a greater problem than at first suggested.—We are, etc.,

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¹ Macdonald, A. G., and McNeill, R. S., *British Journal of Anaesthesia*, 1968, 40, 508.

Milk pH and Gastroenteritis in Newborn Infants

SIR,—Harrison and Peat¹ reported that feeding full-term normal newborn infants with a humanized cow's milk (Similac) that had been alkalinized to pH 7.2 produced a stool of low pH (mean 5.5), of reduced buffering capacity, and a bacterial flora of predominantly *Lactobacillus bifidus*. The infants tended to regain their birth weight earlier than a control group on non-alkalinized Similac. Realizing that if these findings were confirmed the feeding of such an alkalinized