

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> <sup>1</sup>		Hashim and Asfour <sup>4</sup> (normal, term, n = 15)	King <i>et al.</i> <sup>3</sup> (normal, term, n = 5)	Baker <sup>5</sup> (term, post-mortem, n = 5)
		Dutch (healthy, some low birth weight, n = 12)	British (most 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.*<sup>3</sup>).

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*, 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 fore-arms 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.*<sup>1</sup> 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 observation<sup>2</sup> 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,<sup>1</sup> 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 Peat<sup>1</sup> reported that feeding full-term normal newborn infants with a humanized cow's milk (Similac) that had been alkalized 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-alkalized Similac. Realizing that if these findings were confirmed the feeding of such an alkalized

TABLE I—Comparative Data of Five Newborn Infants on Alkalinized Milk and in Five Controls

	Group on Alkalinized Milk (n=5)	Control Group (n=5)
Mean gestation (weeks (range))	32.5 (30-36)	31.5 (30-33)
Mean birth weight (grams (range))	1658 (1530-1840)	1627 (1360-1810)
Mean milk volume/day (ml)	417	387

TABLE II—Results of Stool Examination and Growth Measurements in Five Newborn Infants on Alkalinized Milk and in Five Controls

	Group on Alkalinized Milk (n=5)	Control Group (n=5)
No. of stools examined	50	42
No. <i>L. bifidus</i> isolates	8	5
Mean counts/gram stool	$1.2 \times 10^6$	$1.3 \times 10^6$
Other organisms: Mean counts/gram of stool	$4.8 \times 10^8$	$1.7 \times 10^9$
Mean stool pH (range)	6.46 (5.42-8.10)	6.43 (4.89-7.68)
Mean weight gain (g/day (range))	27.1 (21.5-32.8)	24.8 (22.6-28.6)
Mean length increase (cm/week (range))	0.95 (0.65-1.09)	0.98 (0.76-1.19)
Mean increase skin fold thickness (mm/week (range))	0.79 (0.53-0.92)	0.82 (0.47-1.12)
Mean increase head circumference (cm/week (range))	0.87 (0.69-0.97)	0.83 (0.72-0.92)

milk might afford some protection against gastroenteritis, we repeated Harrison and Peat's study, but in newborn infants of low birth weight to enable us to assess them over a longer period.

Ten infants were fed S.M.A.-S26, but five of them (chosen alternately on entering the unit) had 1 ml of 8.4% sodium bicarbonate added to every 100 ml of milk, bringing the pH of the milk from a mean of 6.98 to a mean of 7.37. The two groups were found to be comparable (table I). All infants received intravenous fluids during the first few days, all (except one in the control group) required oxygen for respiratory distress, and all (except one in the control group) received parenteral antibiotics during the first two weeks of life. Growth rate was measured by weight, length, skin fold thickness in three areas, and head circumference during the period from the first day after intravenous fluids were discontinued till the day of discharge from the unit (which varied from the 38th to the 55th day). Stools were examined twice a week for pH and by surface viable count on various culture media for the bacterial content. The modified, reinforced clostridial medium of Willis *et al.*<sup>2</sup> was used for the selective isolation of *L. bifidus*.

Lactobacilli were isolated from a small number of stools in each group (table II). Other organisms cultured were mostly *Escherichia coli*, *Klebsiella* spp., and *Streptococcus faecalis* but *Bacteroides* spp., *Clostridium welchii*, and non-haemolytic streptococci were isolated occasionally in both groups. There was no significant difference between the groups in any of the parameters measured.

Admittedly these infants were in no way comparable with the group studied by Harrison and Peat, but infants under intensive care, as ours were, are particularly prone to infection in all parts of the body and therefore are likely to benefit from any measures that might discourage colonization of the bowel with pathogenic organisms. Our inability to confirm the findings of Harrison and Peat in our group of infants is therefore unfortunate.—We are, etc.,

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<sup>1</sup> Harrison, V. C., and Peat, G., *British Medical Journal*, 1972, 4, 515.

<sup>2</sup> Willis, A. T., *et al.*, *British Medical Journal*, 1973, 4, 67.

### Cerebral Lupus—Wider Implications

SIR,—Your leading article (8 March, p. 537) very rightly draws attention to the fact that the choroid plexus bears many struc-

tural similarities to the renal glomerulus. Just as the central nervous system involvement of systemic lupus erythematosus is attributed to immune complexes at the choroid plexus, the spinocerebellar degeneration seen in the so-called paramalignant syndromes may be due to immune complexes lodged there, caused by tumour-derived antigens. Certainly the finding of such immune complexes in the kidney in patients with tumour elsewhere has been reported,<sup>1,2</sup> though unfortunately in neither case was any attempt made to study the choroid plexus of these patients at necropsy, as is technically eminently feasible.<sup>3</sup>

It is interesting to correlate the structural similarity at these two sites with a biochemical one. Enzymes involved in the  $\gamma$ -glutamyl cycle,<sup>4</sup> specially the transpeptidases, which are involved in the very first step of glutathione degradation and play such an important role in the uptake of amino-acids into the cell, are found in their maximum concentration in these very organs—in the kidney and at the apical portions of the epithelial cells in the choroid plexus. It is quite simple to understand then why patients with erythrocyte  $\gamma$ -glutamyl synthetase deficiency and haemolytic anaemia<sup>5</sup> have aminoaciduria and signs of C.N.S. involvement, including psychosis and spinocerebellar degeneration.

One is then able to speculate that perhaps the immune complexes in cerebral lupus or in the paramalignant syndromes interfere with aminoacid transfer by obstructing the  $\gamma$ -glutamyl cycle at some point directly or indirectly; and if investigations along these lines, which have so far been neglected, prove fruitful the last sentence in your leading article (8 March, p. 537), "the recognition that the brain is not immunologically privileged . . . may have implications for the pathogenesis of other neurological diseases," would perhaps serve as a starting point to revolutionize our concepts about the enigmatic and obscure degenerative diseases of the central nervous system.—I am, etc.,

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<sup>1</sup> Lewis, M. G., *et al.*, *Lancet*, 1971, 2, 134.

<sup>2</sup> Coenzan, M. E., *et al.*, *New England Journal of Medicine*, 1973, 289, 520.

<sup>3</sup> Atkins, C. J., *et al.*, *Annals of Internal Medicine*, 1972, 76, 65.

<sup>4</sup> Meister, A., *Annals of Internal Medicine*, 1974, 81, 247.

<sup>5</sup> Konrad, P. N., *New England Journal of Medicine*, 1972, 286, 557.

### Race Relations

SIR,—I read with interest your apology (17 May, p. 400) about an advertisement which you had published (3 May, p. xiv).

Is it not a crazy world we live in when a Kuwaiti oil company cannot advertise for an Arab doctor, preferably Kuwaiti, without offending the Race Relations Board?—I am, etc.,

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### Disaster Planning—Fact or Fiction?

SIR,—In spite of recent symposia on problems of disaster planning the suspicion remains, as you suggest in your leading article (24 May, p. 406), that preparations are far from adequate.

At hospital level a disaster may be regarded as a non-specific stress test for the whole institution. The single most important step in any plan is the mobilization of a very small number of officers in the medical, nursing, and administrative fields who have the authority and experience to make the hospital respond appropriately to the stimulus which is being applied. Provided such a control team can rapidly be mobilized, it is not difficult to elicit a maximal and controlled response by the hospital to any kind and size of stimulus.

The problem of adequate preparedness in all the emergency services throughout the whole country seems to me a somewhat similar problem—basically a problem of control and responsibility. Each of the emergency services has national headquarters, but there is no mechanism for united or co-ordinated control of the emergency services to meet disaster. Until the question of responsibility and authority is settled, it is difficult to see how the uncertainty of which you complain can be eliminated.—I am, etc.,

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### B.M.A.: Need for Radical Change

SIR,—I would like to endorse everything that Dr. J. P. Lee-Potter has stated in his letter (24 May, p. 446). A great number of doctors, both members and non-members of the B.M.A., are convinced that there is a need for a radical change in the administration of B.M.A. affairs, which appear to be out of touch with present-day problems and far too concerned with maintaining an existing and rather cumbersome pattern of administration, much of which is not related to present-day medical activities.

I was amazed to read in *Pulse* that the new Secretary of the B.M.A. will be nominated or "recommended" by a committee of the Association and that the post will not be advertised publicly. Indeed, *Pulse* went so far as to suggest that the new Secretary has already been "appointed" subject to confirmation by the full Council.

Many of us have gone through a fairly