

# BRITISH MEDICAL JOURNAL

SATURDAY 25 JUNE 1983

## LEADING ARTICLES

<b>Reye's syndrome: 20 years on</b> A P MOWAT . . . . .	1999	<b>Reflux nephropathy</b> PRISCILLA KINCAID-SMITH . . . . .	2002
<b>Treating the menopause</b> I D COOKE . . . . .	2001	<b>Regular Review: Fraud in science</b> LARRY ALTMAN, LAURIE MELCHER . . . . .	2003

## CLINICAL RESEARCH • PAPERS AND SHORT REPORTS • PRACTICE OBSERVED

<b>α Gliadin antibody levels: a serological test for coeliac disease</b> CLIONA O'FARRELLY, J KELLY, W HEKKENS, B BRADLEY, A THOMPSON, C FEIGHERY, D G WEIR . . . . .	2007
<b>Seminal fluid excretion of cytomegalovirus related to immunosuppression in homosexual men</b> ROBERT J BIGGAR, H KERZEL ANDERSEN, PETER EBBESEN, MADIS MELBYE, JAMES J GOEDERT, DEAN L MANN, DOUGLAS M STRONG . . . . .	2010
<b>Schistosomiasis mekongi diagnosed by rectal biopsy</b> G LORETTE, M R JAAFAR, M F GROJEAN, TH DUONG . . . . .	2012
<b>Mycoplasma hominis septicaemia</b> H FRIIS, A PLESNER, J SCHEIBEL, T JUSTESEN, K LIND . . . . .	2013
<b>Comparison of thiazides and amiloride in treatment of moderate hypertension</b> J P THOMAS, W H THOMSON . . . . .	2015
<b>Successful treatment of middle aged and elderly patients with end stage renal disease</b> DAVID H TAUBE, ELIZABETH A WINDER, CHISHOLM S OGG, MICHAEL BEWICK, J STEWART CAMERON, CHRISTOPHER J RUDGE, D GWYN WILLIAMS . . . . .	2018
<b>Simple drug delivery system for use by young asthmatics</b> R L HENRY, A D MILNER, J G DAVIES . . . . .	2021
<b>Cellulose granulomas in the lungs of a cocaine sniffer</b> C B COOPER, TONY R BAI, E HEYDERMAN, B CORRIN . . . . .	2021
<b>Bursal fluid lactate determination and the diagnosis of bursitis</b> RAYMOND J NEWMAN, GORDON D W CURTIS, MARY P E SLACK . . . . .	2022
<b>Ineffectiveness of haemodialysis in atropine poisoning</b> D P WORTH, A M DAVISON, T G ROBERTS, A M LEWINS . . . . .	2023
<b>Chronic graft versus host disease presenting with polymyositis</b> N PIER, V DUBOWITZ . . . . .	2024
<b>General Practice in the Year 2000: Point of view of an overseas doctor</b> ABBAS VIRJI . . . . .	2025
<b>Practice Research: Improving the care of asthmatic patients in general practice</b> MICHAEL MODELL, JENNY M HARDING, ELIZABETH J HORDER, PETER R WILLIAMS . . . . .	2027
<b>Interesting GPs of the Past: Samuel Taylor Chadwick: 1809-73</b> IVOR FELSTEIN, NASIM NAQVI . . . . .	2031

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## MEDICAL PRACTICE

<b>Guidelines for community menopausal clinics</b> GILLIAN M CRAIG . . . . .	2033
<b>Outside Europe: Value of stool examination in patients with diarrhoea</b> BARBARA J STOLL, ROGER I GLASS, HASINA BANU, M IMDADUL HUQ, M U KHAN, MAFIZUDDIN AHMED . . . . .	2037
<b>Aviation Medicine: Medical aspects of airline operations I: Health and hygiene</b> RICHARD M HARDING, F JOHN MILLS . . . . .	2041
<b>New Drugs: Anticonvulsant drugs</b> D L W DAVIDSON . . . . .	2043
<b>ABC of Computing: To computerise or not</b> A J ASBURY . . . . .	2046
<b>USSR Letter: Demand for a home nursing service</b> MICHAEL RYAN . . . . .	2050
<b>Lesson of the Week: Allergy to aminophylline</b> C HARDY, O SCHOFIELD, C F GEORGE . . . . .	2051
<b>Any Questions?</b> . . . . .	2036, 2040, 2057
<b>Materia Non Medica—Contributions from</b> PRADIP K DATTA, JAMES H LEAVESLEY, A J ROBERTS, G A C BINNIE . . . . .	2036; 2052
<b>Medicine and Books</b> . . . . .	2053
<b>Medicine and the Media—Contributions from</b> JULIAN TUDOR HART, MARTIN MITCHESON, WENDY V HARMAN . . . . .	2057
<b>Personal View</b> J C GRIFFITHS . . . . .	2058

<b>CORRESPONDENCE—List of Contents</b> . . . . .	2059
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<b>OBITUARY</b> . . . . .	2066
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## NEWS AND NOTES

<b>Views</b> . . . . .	2068
<b>Medicolegal—Negligence: further guidance from the courts</b> . . . . .	2069
<b>Medical News</b> . . . . .	2069
<b>BMA Notices</b> . . . . .	2070
<b>One Man's Burden</b> MICHAEL O'DONNELL . . . . .	2071

## SUPPLEMENT

<b>The Week</b> . . . . .	2072
<b>Senior Hospital Staffs Conference</b> . . . . .	2073
<b>From the HJSC: Reducing juniors' hours of work</b> . . . . .	2077
<b>Hospital Junior Staff Conference</b> . . . . .	2079
<b>Unemployment among hospital junior staff: BMA inquiry</b> . . . . .	2080
<b>LMC Conference: GMSC chairman's speech; selected decisions</b> . . . . .	2083
<b>The "necessary" strike—French medicine in turmoil</b> . . . . .	2085
<b>Community Medicine Conference: selected decisions</b> . . . . .	2086

## CORRESPONDENCE

<b>Changing insulin treatment: whose responsibility?</b> K Whyte, MB, and others..... 2059	<b>Postmarketing surveillance of the safety of cimetidine</b> I K Crombie, PHD..... 2062	<b>Farr sighted</b> B Benjamin, FIA..... 2063
<b>Changes in blood lead concentrations in women in Wales 1972-82</b> E King, BSC..... 2059	<b>Doctors should learn 100 words of Punjabi or Hindustani</b> N Higson, MB..... 2062	<b>Hypocholesterolaemia and cancer?</b> G Rose, FRCP; B K Kishore Shetty, MB.... 2064
<b>The sticky eyed infant</b> F J Bone, MRCPATH; E M C Dunlop, FRCP. 2060	<b>Asian and non-Asian morbidity in hospitals</b> J J Jones, PHD..... 2062	<b>Influence of ranitidine on plasma metoprolol concentrations</b> D Jack, PHD, and others..... 2064
<b>Luxuskonsumption, brown fat, and human obesity</b> G R Hervey, MB, and G Tobin, PHD..... 2060	<b>Pneumocystis pneumonia and disseminated toxoplasmosis</b> J S Milledge, FRCP, and Elizabeth Hudson, MRCPATH..... 2062	<b>Erythrocyte ferritin content in idiopathic haemochromatosis and alcoholic liver disease with iron overload</b> L Muiyile, MD, and others; M B Van Der Weyden, FRCP, and others..... 2064
<b>Non-hormonal treatment of osteoporosis</b> A St J Dixon, FRCP..... 2061	<b>Abnormal growth hormone release in response to human pancreatic growth hormone releasing factor in acromegaly</b> G M Besser, FRCP..... 2063	<b>Aviation medicine</b> G Gilray, MFCM..... 2065
<b>Effect of doxapram on heavy sedation produced by intravenous diazepam</b> A P J Lake, FFARCS, and T Houston, FFARCS 2061	<b>Treatment of peripheral vascular disorders</b> R R Freedman, PHD, and P Ianni, MA..... 2063	<b>Epidemiology: a losing cause?</b> P C Walker, MB..... 2065
<b>Profile of recovery after general anaesthesia</b> J M Cundy, FFARCS, and K Arunasalam, FFARCS..... 2062		<b>GP anaesthetists</b> P James, FFARCS..... 2065

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*Correspondents should present their references in the Vancouver style (see examples in these columns). In particular, the names and initials of all authors must be given unless there are more than six, when only the first three should be given, followed by et al; and the first and last page numbers of articles and chapters should be included.*

**Changing insulin treatment: whose responsibility?**

SIR,—Changeover to the new 100 unit insulin started about two months ago. In connection with this we would like to draw attention to a recent case which might have had serious consequences.

A man of 47 was admitted to this hospital in hyperglycaemic ketoacidotic coma with a blood glucose concentration of 50 mmol/l (900 mg/100 ml). He had been a diabetic for six years and had recently had a minor operation in a specialised unit in another hospital. After the operation his usual insulin treatment, 11 marks of 80 strength Monotard MC insulin (Insulin Zinc Suspension, Porcine, 44 units), was changed to 11 marks of U100 administered with the new syringe. This meant that his daily insulin dosage was inadvertently reduced from 44 to 11 units.

It may be that such a changeover was left to a junior doctor—for example, a pre-registration house officer—who may not have been fully informed about the new strength insulin. Moreover, in specialised hospitals there may not be expert help with diabetic problems immediately at hand.

Diabetic patients attending Stobhill General Hospital receive careful counselling from the pharmacists with the help of visual aids. We have not encountered any problems. Even in this large district general hospital, however, a survey of 15 medical and surgical pre-registration house officers has shown gaps in their knowledge about the new strength insulin. If the changeover is to proceed automatically and safely when diabetic patients are admitted to hospital, for whatever reason, then an experienced pharmacist or doctor must

be nominated to supervise this fundamental change in insulin treatment.

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**Changes in blood lead concentrations in women in Wales 1972-82**

SIR,—Dr P C Elwood (14 May, p 1553) refers to the possibility of an analytical drift contributing to the apparent fall in blood lead concentrations in Welsh women over the years 1972-82, but he then appears to dismiss it as a possible contributor. As a longstanding practitioner in the art of blood lead analysis, I want to comment.

Before about 1967 most blood lead analyses were carried out using the wet chemical dithizone method—the so called classical method. Even with this as we refined it (in my own laboratories from about 1956 onwards) the precision and probably accuracy increased to a peak in about 1968. This arose from a combination of “better” chemical reagents, rigorously controlled analytical procedures, and, above all, the use of heparinised lead-free syringes, which overcame the bugbear of syringe contamination and blood contamination during transfer from a syringe to a container of unknown lead contamination. As might have been forecast from the relative ease of contamination of everything by lead, all movements in blood lead

concentrations were thereafter essentially downwards.

In the mid-1960s, variations of atomic absorption spectroscopy were introduced. There were two variants—namely, the presentation of whole blood in ashed or part ashed form, and the presentation of a solvent extract of whole blood. Unfortunately, at that time the atomic absorption instruments were relatively crude and, moreover, were sold to the unwary as being “element specific” when, of course, they were not. From my own and other laboratories’ experience, the blank for unashed whole blood was anything from 0.5  $\mu\text{mol/l}$  (10  $\mu\text{g}/100\text{ ml}$ ) to 1.4  $\mu\text{mol/l}$  (30  $\mu\text{g}/100\text{ ml}$ ), and the failure by many laboratories to understand this gave rise to a number of falsely high blood lead values, culminating in the Avonmouth episode. No doubt the same applied to reports of non-industrial blood lead concentrations. The second variant—solvent extraction—did not suffer as much from this artefact, but there must remain uncertainty as to the validity of, say, many pre-1970 blood lead data obtained by atomic absorption spectroscopy, with a probability of their giving false highs.

After that period, analytical procedures were tightened, with increasing care being taken to avoid the organic matrix problem allied to improved instrumentation, and blood lead concentrations by atomic absorption duly came down. The idea that the instruments were element specific remained, however, and not until the mid-1970s was an inorganic matrix accepted as giving a blank between 0.2  $\mu\text{mol lead/l}$  (4  $\mu\text{g}/100\text{ ml}$ ) and 0.3  $\mu\text{mol lead/l}$  (6  $\mu\text{g}/100\text{ ml}$ ), depending on the technique and instrument. In turn, this was either taken into account, or countered by more sophisticated instruments, to give another downward trend.<sup>1</sup>

Parallel with these developments microblood sampling became popular to avoid venous sampling. This required a new level of “lead freeness” in the microcontainers, which took some time to establish. To my knowledge one batch of these in about 1974 gave results raised by about 0.5  $\mu\text{mol lead/l}$  (10  $\mu\text{g}/100\text{ ml}$ ). I do not know the size of the batch,