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SATURDAY 14 MARCH 1981

LEADING ARTICLES

Gastric ulcer: benign or malign?.....	843	Health services research.....	845
Hypernephroma.....	844	The senile amyloidoses.....	846

CLINICAL RESEARCH • PAPERS AND SHORT REPORTS • PRACTICE OBSERVED

Evidence for a circulating sodium transport inhibitor in essential hypertension L POSTON, R B SEWELL, S P WILKINSON, P J RICHARDSON, R WILLIAMS, E M CLARKSON, G A MACGREGOR, H E DE WARDENER.....	847
Glucose turnover and metabolic and hormonal changes in ethanol-induced hypoglycaemia N M WILSON, P M BROWN, S M JUUL, S A PRESTWICH, P H SÖNKSEN.....	849
Biochemical tests for diagnosis of pheochromocytoma: urinary versus plasma determinations P F PLOUIN, J M DUCLOS, J MENARD, E COMOY, C BOHUON, J M ALEXANDRE.....	853
Pelvic inflammatory disease and the intrauterine device: findings in a large cohort study M P VESSEY, D YEATES, ROSEMARY FLAVEL, KLIM MCPHERSON.....	855
Maintenance of weight loss in obese patients after jaw wiring J S GARROW, G T GARDINER.....	858
Treatment of severe aplastic anaemia with antilymphocyte globulin or bone-marrow transplantation BRUNO SPECK, ALOIS GRATWOHL, CATHERINE NISSEN, URS LEIBUNDGUT, DONATELLA RUGGERO, BRUNO OSTERWALDER, HANS PETER BURRI, PIERRE CORNU, MICHEL JEANNET.....	860
Tea consumption: a cause of constipation? LISELOTTE HØJGAARD, SUSANNE ARFFMANN, MAJA JØRGENSEN, EINAR KRAG.....	864
Pseudomembranous colitis after treatment with metronidazole GEORGE THOMSON, ALISTAIR H CLARK, KAY HARE, WALTER G S SPILG.....	864
Mefenamic acid nephropathy: further evidence P L DRURY, L G ASIRDAS, G V BULGER.....	865
Women and duodenal ulcer NORMAN R PEDEN, ERIC J S BOYD, K G WORMSLEY.....	866
Pitfalls in Practice: Employment law—II NORMAN ELLIS.....	867
Medical Records—I: A personal view KEITH WALKER.....	869
Sex Problems in Practice: What can a general practitioner do? MICHAEL COURTENAY.....	873

MEDICAL PRACTICE

Percutaneous needle biopsy of the lung D J ALLISON, ANNE P HEMINGWAY.....	875
Dealing with the Disadvantaged: Communicating with patients with a language problem JANET THRUSH.....	878
Pollution and People: Contamination of food: mycotoxins and metals DAPHNE GLOAG.....	879
Domiciliary night nursing service: luxury or necessity? M H MARTIN, M ISHINO.....	883
ABC of ENT: Nasal obstruction HAROLD LUDMAN.....	886
Four years' experience of an interim secure unit JAMES HIGGINS.....	889
Secondary prevention in survivors of myocardial infarction JOINT RECOMMENDATIONS BY THE INTERNATIONAL SOCIETY AND FEDERATION OF CARDIOLOGY SCIENTIFIC COUNCILS ON ARTERIOSCLEROSIS, EPIDEMIOLOGY AND PREVENTION, AND REHABILITATION....	894
Any Questions?	882, 885, 896
Materia Non Medica—Contributions from ROBERT C TAYLOR, C R TRIBE AND P A BACON, PENELOPE A BROUGHAM.....	893
Medicine and Books	897
Personal View BRIAN LIVESLEY.....	900

CORRESPONDENCE—List of Contents	901
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OBITUARY	913
-----------------------	-----

NEWS AND NOTES

Views	911
Medical News	912
BMA Notices	912

SUPPLEMENT

The Week	915
Mrs Short tackles the numbers game WILLIAM RUSSELL.....	916
From the Council: Community health doctors' report endorsed	917
The robustness of the London Health Planning Consortium model JOHN CHARLTON.....	920
What price the London Hospital Plan? G H WARD, P A WEST.....	922
Correction: Milage payments for hospital doctors ...	923
Employment of locums	924

CORRESPONDENCE

Beta-blockers in asthma Anne E Tattersfield, FRCP; C Skinner, MRCP; D A Jackson, MB 901	How dangerous are falls in old people at home? P H Millard, FRCP 905	Giving all registrars a fair chance of becoming consultants J J Shipman, FRCS 907
Effect of antiepileptic drugs on the hypothalamic-pituitary axis J Dana-Haeri, MD, and A Richens, FRCP .. 902	Congenital rubella affecting an infant whose mother had rubella antibodies before conception S Krugman, MD 905	Do sick doctors need more than the GMC? Two founder members; T G Tennent, FRCPsych 908
Double pathology as a cause of occult gastrointestinal blood loss K D Bardhan, MRCP; P K Datta, FRCS; M Davies, MRCP 902	Factors contributing to mortality in paracetamol-induced hepatic failure R J Flanagan, BSc, and others 905	Reorganisation and structure of community medicine S S Bakhshi, MFCM 908
Status epilepticus treated by barbiturate anaesthesia J D Miller, FRCS 903	Chlorosis, anaemia, and anorexia nervosa L J Bruce-Chwatt, FRCP; W M Jordan, BM 906	Organisation and management of scientific services in Scotland R A Bird; A D Farr, PHD 908
How many beds do we really need—for example, in neurosurgery? W B Jennett, FRCS; Carol B Sedzimir, FRCSed, and others; Mona V Rivlin, MFCM; J J Jones, AFMC; R D Weir, FFCM, and G H Mooney, MA; G P A Winyard, MRCP, and others 903	Radiosensitisers H B Hewitt, MD; G E Adams, PHD 906	Medical advisory machinery R C Millward; A R M Freeman, MRCPsych 909
A matter of life and death K L Gupta, MRCP 904	The dark future for child health G H Cooper, MB 907	Index-linked pensions D L Williams, FRCGP; E O Evans, FRCGP .. 910
Epidemic of fractures during period of ice and snow J G Avery, MFCM 905	Design for a school computer module J D Leach 907	The medical profession and drug firm hospitality J M Christy; J S Martin, FRCS; K Norcross, FRCS 910
	Using computerised lists of doctors M J C Brown, MB 907	Corrections: A cautionary tale (Moshy); An aid to reducing unnecessary investigations (Hayes and MacWalter) 910
	Bed requirements for undergraduate teaching J O Forfar, FRCP, and R G Mitchell, FRCPed 907	
	City centres and general practice R Lefever, MRCP 907	

We may return unduly long letters to the author for shortening so that we can offer readers as wide a selection as possible. We receive so many letters each week that we have to omit some of them. Letters must be signed personally by all their authors. We cannot acknowledge their receipt unless a stamped addressed envelope or an international reply coupon is enclosed.

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. Titles of papers are not, however, included in the correspondence section.

Beta-blockers in asthma

SIR,—The reminder from Dr June M Raine and others (14 February, p 548) that beta-blocking drugs can precipitate serious asthma and death in vulnerable patients was useful. Although these complications are more likely to occur with non-selective drugs such as nadolol they can also occur with cardio-selective drugs and no beta-blocking drug should be considered free of risk.

I wonder whether, in retrospect, a more aggressive approach with salbutamol should have been tried initially in the patient they describe. Beta-agonists and beta-blockers are competing for bronchial and other beta-receptors so it should be possible to overcome beta-blockade with large doses of beta-agonists. In normal subjects a 60-fold increase in the dose of salbutamol was necessary to achieve bronchodilatation after 80 mg propranolol.¹ A similar increase would be expected in asthmatic patients; so that doses of the order of 10 mg salbutamol every 10 minutes by inhalation and intravenously are probably needed, and should be given until bronchodilatation occurs—heart rate, electrocardiogram, and blood pressure permitting. Although the dose sounds alarmingly high, side effects should not occur since the other actions of salbutamol would also be blocked by the beta-blocking drug; and this has been our experience with normal subjects. Isoprenaline would reverse the cardiac effects of beta-blockade more effectively, but if given alone may reverse these and cause beta₂-stimulation before reversing bronchial beta-blockade. The relative amounts of salbutamol and isoprena-

line to be given will depend on the clinician's assessment of whether bronchial or cardiac beta-blockade is contributing more to the patient's condition. In addition to salbutamol and steroids, inhaled ipratropium or atropine is worth trying and probably aminophylline, though its bronchodilator effect may be attenuated by propranolol² and its cardiovascular effects in the presence of propranolol may not be beneficial.

An alternative approach would be to try the effect of prostaglandin E₂ (PGE₂), which stimulates adenylate cyclase through a receptor separate from the beta-receptor and so should bypass the effects of beta-blockade, which it does in normal subjects.³ This theoretically attractive approach has not, to my knowledge, been used in ill patients so would need to be tried cautiously, particularly as PGE₂ is rather irritant to inhale.

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¹ Gribbin HR, Baldwin CJ, Tattersfield AE. *Br J Clin Pharm* 1979;7:551-6.

² Mackay AD, Baldwin CJ, Tattersfield AE. *Thorax* 1980;35:239.

³ Lewis RA, Seth RV, Tattersfield AE. *Clin Sci* 1981; 60:17P.

SIR,—Although the dangers of adrenoceptor beta-blocking drugs in asthma were pointed out nearly 20 years ago¹ these risks are still not widely enough appreciated as Dr June M Raine and her colleagues point out in their

report (14 February, p 548) of a near-fatal attack of bronchospasm in an asthmatic given nadolol for hypertension.

Dr Raine suggests that if a beta-blocking drug is considered essential in an asthmatic patient treatment should preferably be started under medical observation with peak flow monitoring. I think that this is wise, but it may not always be enough. In an asthmatic subject the acute bronchoconstrictor response to a beta-blocking drug can vary substantially from time to time.² Hence even a small or absent response to one or more test doses does not guarantee that a severe bronchoconstrictor response to the same dose will not occur at a later date. Such a severe reaction may be especially likely if the patient is simultaneously exposed to other bronchoconstrictor influences, such as cold or exercise.³

In most instances the bronchoconstriction induced by beta-blockers in asthma promptly reverses, wholly or partly, with aerosol administration of a beta-stimulant bronchodilator drug. Hence before giving a beta-blocker to an asthmatic it is important to ensure that the patient has an adequate aerosol technique and that he actually carries an inhaler. The dose of bronchodilator (beta-agonist) can be double or treble the usual dose to overcome the antagonist.

Occasionally, as in the present case, the bronchoconstriction does not respond even to large doses of beta₂-stimulant drugs. A possible explanation for this might be that the bronchoconstriction is not a consequence of beta-blockade at all but results from some