Dipyron in Bronchial Asthma

SIR,—We have found that premedication with dipyron before bronchoscopy makes the passage of the bronchoscope easier. We have also found dipyron to be an effective postoperative analgesic (mainly after abdominal surgery) when given on the second or third day after operation, and that in the patients in whom it was effective there was an increase in gas exchange in the lungs. This was perhaps due to deeper respiration resulting from the relief of pain, because there was no change in the respiratory minute volume after dipyron in 20 healthy persons.

These findings prompted us to try dipyron in 82 patients suffering from an attack of asthma. In every case 2.5 g of the drug given intravenously interrupted the attack, and seven of the 82 patients were in status asthmaticus. In 31 of the 75 patients with acute asthma other drugs (aminophylline, orciprenaline, or hydrocortisone semisuccinate) had already been given without effect. In all the patients the intravenous injection of dipyron resulted in the immediate disappearance of cyanosis and relief from the feeling of suffocation. In the seven patients with status asthmaticus the condition had been present for two to three days and had not been relieved by the usual pharmacological agents. One of the patients needed a second injection of dipyron.

I think the effectiveness of dipyron in bronchial asthma needs further investigation, but I thought I should report the above findings now because of Vane's discovery¹ that aspirin-like drugs act by inhibiting the biosynthesis of prostaglandins. Dipyron belongs to this class of drugs. It is also known that prostaglandins E_2 and $F_{2\alpha}$ exert opposing effects on the airways resistance in man.^{2,3} The bronchoconstrictor action of prostaglandin $F_{2\alpha}$ is 10,000 times stronger in asthmatic patients than in healthy subjects.⁴ Recently Flower and Vane⁵ have described the selective inhibition of prostaglandin synthetase in brain by the antipyretic drug paracetamol. Dipyron is a weak analgesic, antipyretic, and anti-inflammatory drug. Could it be that dipyron is a selective inhibitor of the biosynthesis of prostaglandin $F_{2\alpha}$ in the lungs?—I am, etc.,

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5 Flower, R. J., and Vane, J. R., *Nature*, 1972, 240, 410.

Dip-slide in Urology

SIR,—My colleagues and I recently evaluated the Uricult dip-slide in gynaecological practice and came to the same conclusion as Mr. F. R. Jackaman and others (27 January, p. 207).¹ However, we were able to enhance the usefulness of this technique by testing for bacterial sensitivity to antibiotics. This was achieved by placing antibiotic sensitivity discs on the lower end of the dip-slide, the film of urine being sufficient to

hold them in place. Sensitivity to the antibiotic is easily seen as a clear zone of inhibited bacterial growth around the disc (see fig.).

By using this method the practitioner can, within 24 hours of taking a clean-catch specimen of urine, detect the presence of

clinically significant bacteriuria (with at least as high a degree of accuracy as the standard laboratory method) and be guided with regard to chemotherapy. This will allow a more selective use of skilled laboratory staff, reduce the interval before commencing treatment, and be considerably cheaper for the patient.—I am, etc.,

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Durban, South Africa

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Sleep and Growth Hormone

SIR,—I was surprised and perplexed to note in your leading article (27 January, p. 188) reviewing stimuli of growth hormone secretion the general conclusion that "insulin-induced hypoglycaemia remains the procedure of choice when investigating the anterior pituitary's capacity to secrete growth hormone." Alternative praise was assigned to glucagon as a stimulus and, in this capacity, glucagon was rated above arginine on the basis of study in a small number of subjects.

While I might discuss the known and hypothetical differences in the mode of growth hormone stimulatory action of each agent or argue that the total available data do not necessarily leave the hazardous insulin-induced hypoglycaemia the stimulatory agent of choice (or glucagon the best alternative), I would like to merely point out that natural sleep is undoubtedly the phenomenon (not an artificial agent) with which to study growth hormone release. I believe this statement is substantiated in the literature.¹⁻¹¹

In six years' experience in the study of more than 350 subjects sleep has provided an exceptionally and incomparably reliable tool with which to study the natural release of growth hormone in normal subjects as well as in subjects with aberrations in growth and metabolism. Moreover, a single blood sample drawn during the first 60-90

minutes after sleep onset affords a reliable measure of growth hormone release and does not require artificial provocation.¹⁰⁻¹¹—I am, etc.,

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Denver, Colorado, U.S.A.

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Cremation Certificates

SIR,—Dr. I. E. Doney (10 March, p. 616) may be a little optimistic in his belief that cremation fees will soon be things of the past. Acceptance of the Brodrick Report will first have to be translated into legislation that repeals the Cremation Act of 1902 and its subsequent regulations. In the present crowded Parliamentary timetable who can say when this might be? It would appear just as likely that this report will share the fate of so many reports commissioned by Government and be shelved indefinitely.

Though I myself have never come across form H (a certificate of anatomical examination) in over 16,000 consecutive cremations, I am able to enlighten him about form G. This is a record, bearing a serial number of each cremation, kept by the superintendent of the crematorium and not normally, therefore, seen by a doctor.—I am, etc.,

J. A. G. GRAHAM

Health Department,
Worthing

SIR,—Dr. I. E. Doney (10 March, p. 616) asks about form G. This is the Register of Cremations kept by the registrar of the cremation authority in accordance with Regulation 17 of the Cremation Regulations, 1930.—I am, etc.,

W. P. CARGILL

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Treatment with Calcitonin

SIR,—Your leading article (17 February, p. 371) on treatment with calcitonin prompts me to suggest that patients for whom calcitonin therapy is being considered should be examined to see if they are suffering from deafness. With pure-tone and impedance audiometry it should be possible to establish if fixation of the stapes is the cause of their deafness. It would then be interesting to see if any improvement of hearing occurs with calcitonin therapy. Such an improvement in hearing has been recorded.¹

I feel that unless such an investigation is carried out on a nationwide basis sufficient