

PREVENTION OF HAIR LOSS BY HEAD-BAND DURING CYTOTOXIC THERAPY

SIR,—Some patients with various forms of malignancy, particularly metastatic breast cancer, receive treatment with cytotoxic drugs, almost always given in a combination regimen. An important side-effect of the use of these drugs, if no precautions are taken, may be total alopecia. Various opinions have been expressed about the value of a head-band. I have found that, when a head-band is used in a certain way, the risk of alopecia has been virtually abolished.

The head-band should be anteriorly placed with its lower margin just above the eyebrows; it should fit snugly down into the sulcus between the pinna and the scalp on each side, and its upper margin posteriorly should be positioned about one inch below the occipital protuberance. It should be slowly inflated to a minimum pressure of 240 mm. Hg, or, in the rare cases in which the patient's blood-pressure is as high as this or above, to the upper part of the sphygmomanometer scale, which is usually 300 mm. Hg. Naturally it should always be inflated before any of the drugs are administered, and it should be left in position with a most careful check on the pressure level for at least seven minutes after all the drugs have been injected intravenously.

With a combination cytotoxic regimen using a 5-day course of cyclophosphamide, 5-fluorouracil, vincristine, and methotrexate, no patient has developed alopecia among the last 20 patients treated. Moreover, a sizeable number of patients have been given several courses of drugs, and 2 have spontaneously expressed the opinion that the condition of their hair has actually improved.

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INTRAVENOUS FEEDING

SIR,—I agree with Dr Allison (Jan. 26, p. 135) that intravenous feeding is of great value in certain groups of seriously ill patients.

The patients in whom we encountered cellular overhydration in association with glucose and insulin intravenous feeding were more cachectic than those described by Dr Allison and had not undergone surgery at the time of study; they weighed 35 kg. or less as a result of long-standing gastrointestinal disease, and were given intravenous feeding to prepare them nutritionally for operation. The regimen used was almost identical to that described by Dr Allison, being based on 50% dextrose and soluble insulin, aimed at maintaining the plasma-glucose at between 100 and 200 mg. per 100 ml. and a urine sugar of 0.25%. 'Vamin' was given as a source of nitrogen. With this regimen, positive nitrogen balances of 6 to 10 g. each day were produced throughout the period of treatment. However, the patients developed large positive water balances, retaining over 1 litre each day. After 5 days signs of cerebral oedema developed, the patients complaining of headache and becoming increasingly drowsy. Despite the excessive water load there was no evidence of increased extracellular-fluid volume, as shown by the absence of peripheral oedema or of hyponatraemia. Increased intracellular-fluid volume was indicated by a 10% increase in mean red-cell volume during this period. These factors led us to diagnose cellular overhydration.

The effects of glucose injection (0.5 g. per kg. of 50% glucose) during hæmorrhagic shock were studied in pigs. Significantly higher intracellular glucose levels occurred in

red blood-cells during shock than in control studies ($P = < 0.01$). Along with these changes mean corpuscular volume fell in control studies, whereas it increased significantly during shock ($P = < 0.0025$). I believe this indicates that a potent factor in glucose intolerance during injury is inability of cells to phosphorylate glucose. I suspect that this process in more chronic form takes place in patients with severe malnutrition.

I have not suggested that insulin causes glucose to pass against a concentration gradient. I would not claim that increased cellular glucose levels are the only cause of cellular overhydration following injury; but I would reiterate that intravenous feeding with glucose and insulin must be undertaken with great care.

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INVESTIGATING FEMALE INCONTINENCE

SIR,—Your editorial (Dec. 8, p. 1303) makes the valid point that problem cases require full urodynamic facilities. We agree that urethral pressure profile measurement is invaluable. However, it is not sufficient to say that the proximal urethral resistance is the critical measurement in stress incontinence unless this is related to the volume of fluid in the bladder at that time. It has been shown that the profile alters with bladder volume.¹ Profiles are therefore measured first with 25 ml. in the bladder and then at functioning capacity as determined by continuous-flow cystometry. Women with good pelvic-floor musculature tend to show an improved profile at larger volumes. Incontinent women, with a poor closure pressure at small volume, may show further deterioration when the bladder is filled to capacity. We agree that, in stress incontinence, this deterioration tends to occur in the bladder neck and proximal urethra. However, stress is not the only symptom related to poor profiles.

The profile consists of two components, the functioning length of the urethra and the pressure exerted along that length. The basic technique described by Brown and Wickham² has a serious weakness in that no check is made of detrusor activity during measurement. Enhorning et al.³ showed that detrusor contraction tended to open the bladder neck, and we have confirmed this.⁴ It is therefore necessary to perform cystometry throughout profile measurement, especially in patients with an irritable bladder or when recording at bladder capacity.

We also agree that the effect of electrical stimulation during profile measurement bears no relationship to the subsequent clinical result from the use of an electronic stimulator. We have shown, however, that incontinent patients whose profiles deteriorate with increased bladder volume tend to benefit from stimulators. This has been our standard diagnostic and prognostic investigation for the past four years.⁴

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