Correspondence

Comparison of oral prednisolone and intramuscular depot triamcinolone in patients with severe chronic asthma

Sir,—I am very interested in the report of Dr RF Willey and his colleagues (May 1984;39:340-4).

Having had over 15 years experience of long term therapy with triamcinolone acetonide in asthmatic patients, I would like to contribute a few comments of my own. According to the references cited it might be assumed that so far only one paper has been published on this type of therapy. In fact, there have been a great many publications dealing with this problem.

Our group (apart from papers published in Polish medical journals) has published seven reports on this type of therapy in English and German medical journals.1-7

During the third and fourth Charles Blackley symposia in Nottingham, in 1978 and 1981, we presented our experiences of this type of therapy. Since triamcinolone acetonide was rarely used in the United Kingdom, however, our communications did not arouse much interest.

The principles included in Dr Willey’s report seem open to question, as does his interpretation. This would seem to derive from his assumption that the weight of triamcinolone acetonide in dosage is equivalent to that of triamcinolone. This is, however, not the case. According to information made available to investigators by ER Squibb Ltd triamcinolone acetonide (in rats) is 10 times more potent in anti-inflammatory and nine times more potent in neoglucogenetic properties than triamcinolone alcohol and prednisolone. To achieve an equivalent dosage of triamcinolone and prednisolone the weight of triamcinolone acetonide should be multiplied by 12. Therefore Dr Willey’s statement to the effect that 80 mg of triamcinolone acetonide every 28 days is equivalent to 3·43 mg of prednisolone daily is incorrect. In fact, it is a very high dose, equivalent to over 35 mg of prednisolone daily.

In long term therapy we never exceed 1·7 mg of triamcinolone acetonide (80 mg every six weeks) as a calculated daily dosage, and when the need to exceed this dosage arises this means that the treatment must be changed and another type of steroidal drug applied, because 1·7 mg of triamcinolone acetonide daily is, in our opinion, equivalent to 16 mg of triamcinolone in tablet form.

In long term treatment of the majority of steroid dependent patients, we usually achieve good results with 1·0-1·2 mg of triamcinolone acetonide daily (equivalent of 2-3 tablets of triamcinolone). We never give injections of triamcinolone acetonide at regular intervals, but give them only when a patient exceeds the safe dosage of bronchodilators. This type of treatment excludes an unnecessary excess of glucocorticoid administration. When Dr Willey’s therapy is analysed it is not surprising that 30% of the patients revealed signs of myopathy. We observed only two patients who developed myopathy among over 300 patients treated with triamcinolone acetonide over a period of two years. These two patients had been taking 80 mg of triamcinolone acetonide every three weeks without medical supervision. Nevertheless, our study on a group of 20 patients, treated with triamcinolone acetonide for an average of 6·4 years and without clinical signs of myopathy, confirmed that in 15 of them an abnormal electromyographic tracing could be observed.

Four out of 12 of Dr Willey’s patients showed menstrual disturbances. We do not know how many of the female patients were still menstruating (the ages of the whole group were given as 15-76 years). This side effect ought to have been discussed in terms of the percentage of females of reproductive age.

In our study menstrual disturbances were quite frequent, so we decided that women of reproductive age were not to be treated with triamcinolone acetonide.

Easy bruising was the most frequent side effect of triamcinolone acetonide treatment, particularly in the elderly (we call this “kenalog hands”). This is very often the reason for taking patients off the triamcinolone acetonide regime. It is extraordinary that this side effect was not observed in Dr Willey’s study.

The authors conclude that better results can be achieved with triamcinolone acetonide than with prednisolone. This is true, but with a much higher equivalent dosage.

I fully agree that even with a high triamcinolone acetonide dose adrenal cortex suppression was less pronounced. Our “twin studies” (4) have revealed that long term triamcinolone acetonide therapy (more than six years) causes less damage to the adrenal cortex (21% abnormal tetraacosactrin tests) than an equivalent dose of oral triamcinolone or prednisolone (42% abnormal tetraacosactrin tests).

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7 Droszcz W. Vergleichende unter Sudungen bei Langfristiger Anwendung von Depot-Triaminol und Depot-Betamethason in Behandlung Obstructiver Ventilationsstorungen unter besonderer Berucksichtigung der Neben-
Correspondence

Peek flow rate records in surveys: reproducibility of observers’ reports

In the paper by Dr KM Venables and colleagues (November 1984; 39: 828–32) we regret that there are errors in the first paragraph of the methods section, in which it is stated that recordings from 61 men were studied. Of the 23 persons employed in the electronics factory, 18 were in fact women. The beginning of the last paragraph of page 828 should read: “Recordings from 61 subjects formed the basis of the study. Thirty eight subjects (all male) were currently employed in a steel coating plant... and 23 (18 female) were employed in an electronics factory...” Elsewhere in the paragraph the word men should be taken to indicate subjects.

Bronchial reactivity to inhaled histamine and annual rate of decline in FEV1 in male smokers and ex-smokers

Smoking, allergy, and the differential white blood cell count

In the two papers by Dr RG Taylor and others (January 1985) we regret that page numbers are missing from two of the references. In ref 10 on p 16 the pages are 17–22 and in ref 24 on p 21 they are 9–16.
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