diet; but many more subjects would be required to test these aspects adequately.

We should be very happy to discuss our growth data in greater depth with individual investigators.

Growth patterns after surgery for virilising adrenocortical adenoma

Sir,—We agree with the notion of Salt et al in their article on growth patterns after surgery for virilising adrenocortical adenomas that more information is needed on the natural history of growth and pubertal development in these children.1 We therefore have recently published observations on growth in 10 children aged 0-8 to 11-8 years with hormonally active adrenocortical tumours (nine carcinomas, one adenoma) before and after surgery (excess of androgens 10/10, of glucocorticoids 5/10, of oestrogens 1/10).2 In addition, five of 10 patients had a palpable abdominal tumour at the time of initial presentation. In only one patient increased longitudinal growth alone had initiated the diagnostic workup.

Only two of our patients had a preoperative height SD score >+2. In one patient, an increase in height SD score above baseline was noted simultaneously with the first signs of precocious sexual maturation. However, in five of seven patients followed up postoperatively, in whom removal of the tumour controlled symptoms of hormone excess, height SD score initially declined after surgery. We took this as evidence for catch-down growth, comparable with that in patients with congenital adrenal hyperplasia and delayed initiation of treatment.3 Although Salt et al state that no catch-down growth had occurred in their patients,4 at least in five of them a period of rapid growth was followed by normalisation of growth velocity and a trend for the bone age to come back in line with the chronological age, similar to our patients. The fact that in most of their patients final height SD score was higher than height SD score at the time of diagnosis is no evidence against postoperative catch-down growth, as at that time the majority of patients were of an age at which most children may not have shifted yet to the definitive centile range that they tend to follow for the remainder of their growth.

Finally, the course of the two patients described by Salt et al who had androgen and glucocorticoid excess agrees with our observation that in instances where high concentrations of both hormones affect the growing bone simultaneously, the androgen effect on linear growth and bone maturation appears to dominate. This is different from normal puberty, where hypercortisolism can inhibit longitudinal growth in the presence of physiologic concentrations of gonadal steroids.4

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Terbutaline sulphate Turbuhaler in severe acute asthma

Sir,—Pedersen et al have shown the efficacy of 0-25 mg of terbutaline administered by Turbuhaler (Astra) in acute asthma.1 We similarly treated 20 patients with a mean age of 9-9 years (range 5-15) who had acute asthma and severe airway flow limitation.2 They were taking prophylactic drugs (inhaled nedocromil sodium and/or budesonide) and inhaled β2 agonists before coming to our unit.

Spirometry (Vitalograph) was performed (best out of three) at 0, 30, and 60 minutes before each dose of terbutaline and again at 90 minutes. The patients inhaled 2-5 mg of terbutaline sulphate (five doses of 0-5 mg/2 minutes), followed by a further 2-5 mg and 1 mg (two doses of 0-5 mg/2 minutes) at 0, 30, and 60 minutes. The results, as a percentage of predicted values, are shown in the table. One way analysis of variance of repeated measures and adjacent difference contrasts for pairs showed significant differences (p<0-05) between pairs for all but the 60-90 minute pair for FEF25-75%.

In two cases, despite clinical improvement, there were no important changes in spirometry even after the administration of 0-5 mg nebulised salbutamol.

Clinically, all patients achieved a normal breathing pattern between the first and third dose without side effects.

In conclusion, the Turbuhaler seems to be effective in severe acute asthma.

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Results of spirometry

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>0</th>
<th>30</th>
<th>60</th>
<th>90</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC</td>
<td>66-4</td>
<td>89-2</td>
<td>96-8</td>
<td>102-7</td>
</tr>
<tr>
<td>FEV1</td>
<td>50-2</td>
<td>72-4</td>
<td>82-3</td>
<td>87-8</td>
</tr>
<tr>
<td>PEFR</td>
<td>50-0</td>
<td>70-3</td>
<td>77-7</td>
<td>86-0</td>
</tr>
<tr>
<td>FEF25-75%</td>
<td>32-5</td>
<td>56-3</td>
<td>67-6</td>
<td>71-7</td>
</tr>
</tbody>
</table>

FVC = forced vital capacity, FEV1 = forced expiratory volume in one second, PEFR = peak expiratory flow rate, FEF25-75% = forced expiratory flow between 25% and 75% of the forced vital capacity.
Growth patterns after for virilising adrenocortical adenoma.

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