release has been recognized as an important cause of growth failure."

The Bristol report showed that stunting of physical growth is commonly associated with a low and/or inappropriate intake of food, especially in the first two years of life, and that this is in turn associated with a low birth weight and stunting of growth. This situation is common in developing countries, but also in some industrialized countries, and it is a major public health problem. To do so is likely to block attempts by parents or doctors to understand and help affected children to attain, both physically and mentally, their true potential stature. We are, etc.,

J. APLEY
D. RUSSELL DAVIS
The Bristol Royal Hospital for Sick Children, Bristol


Pseudomonas Septicaemia Following Superficial Colonization

Sit—Recently my colleagues and I reported (23 November, p. 440) four patients with leukaemia or lymphoma, neutropenia, and apparently trivial infections of the skin or conjunctiva from which Pseudomonas aeruginosa was isolated. All four later developed septicaemia and the same organism was isolated from cultures of peripheral blood: three patients died. It was suggested that the isolation of pseudomonads from apparently localized lesions in patients with compromised antibacterial defences is an indication for systemic antibiotic therapy. Since that communication was submitted for publication two further instances have been observed of colonization by pseudomonads in neutropenic patients. In each case, septicaemia due to the same organism rapidly ensued.

Case 5.—An 8-year-old boy with acute promyelocytic leukaemia became neutropenic (neutrophils 10/μl) during initial chemotherapy with doxorubicin and cytarabine. Though he was febrile on admission, repeated swabs and blood cultures disclosed no pathogens. When he developed pneumonia not responsive to antibiotic therapy a throat swab grew Ps. aeruginosa. Three days later his pyrexia increased and the same organism was isolated from blood cultures. Because the bacteriological findings on the throat swab were already known treatment with amoxycillin and cephaloridine was begun when the blood cultures were taken and he improved rapidly with these antibiotics supplemented by granulocyte transfusions.

Case 6.—A 17-year-old youth with acute undifferentiated leukaemia in relapse was treated intensively with doxorubicin and cytarabine and his peripheral blood neutrophil count fell to 10/μl. Ps. aeruginosa was isolated from routine swabs taken on two occasions and during the fall in the axillary and inguinal skin. No cutaneous lesion was present at either site. Two days later he became febrile and unwell and a repeat blood culture was positive from a blood culture. Gentamicin and carbenicillin were begun at the time of blood culture and granulocyte transfusions were given, and the patient recovered.

Knowledge that pseudomonads have previously been isolated from an inappropriate site, such as the pharynx in case 5, should affect the initial choice of antibiotics for a subsequent febrile episode in a neutropenic patient. In case 6 the occurrence of septicaemia after a positive skin swab but in the absence of any cutaneous lesion suggests that the mere presence of Ps. aeruginosa on the skin of a neutropenic patient may be an indication for systemic antibiotic therapy as well as the local use of antiseptics. Isolation of pseudomonads from a cutaneous lesion, however minor, is an even stronger indication for systemic treatment. I am, etc.,

A. S. D. SPIERS
M.R.C. Leukaemia Unit.
Royal Postgraduate Medical School, London W.12

Prevention of Exercise-induced Asthma by Indoramin

Sit,—I write on behalf of Professor S. Bianco and others in reply to the letters from Dr. K. N. V. Palmer and his colleagues (16 November, p. 409) and from Dr. S. Godfrey (23 November, p. 469).

We are aware of Dr. Palmer’s interest in the possible applications of alpha-adrenoceptor blockade in asthmatic patients and look forward to seeing his data on the effects of these drugs on response to beta-adrenoceptor agonists. We feel that some casual observations on this subject in relation to the action of thymoxamine. It would seem that Dr. Palmer’s more extensive data may be clinically important. I may say that at this point that six of our patients remarked upon the improvement in symptoms after dosage with indoramin.

Dr. Godfrey’s letter deals specifically with our paper (5 October, p. 18). He gives an alternative interpretation of the data which we published, taking exception to the use of the word “prevent” in describing the effects of indoramin on the response to exercise in patients with exercise-induced asthma (E.I.A.). Clearly his criticism has substance, since the general shape of the response in time, in terms of specific airways conductance (SGaw) and FEV1, is broadly similar whether adapted to placebo or to the drug, to that in our study. This is a direct result of the initial bronchodilatation which occurs after indoramin and can be interpreted to indicate that the drug is not preventing the usual response at all. I suspect that larger doses of the drug could have completely swamped the response to exercise but this possibility does not affect the general line of this argument. We were most careful not to claim that our observations proved an abnormal activity of alpha-receptors as the cause of E.I.A. but merely that they were, consistent with this hypothesis.

This is not the place to deal with the details of the third paragraph of Dr. Godfrey’s letter except to state that the random errors of estimations of SGaw and the other measurements were minimized so far as possible by replication and suitable statistical assessment of the raw data. We consider that the effect of the small increase in dose of indoramin in the one subject who was tested at two dose levels might well have been due either to a variation in absorption of the drug, which was given by mouth, or to the known variability of E.I.A. itself. We are grateful for the reference to Sly et al.2 which had escaped our notice.—I am, etc.,

F. J. PRIME
Cardiothoracic Institute, London S.W.3


Mean Changes (± S.E.M.) of Specific Airway Conductance induced by Exercise expressed as % 90-minute Control or 90-minute Post-indoramin Values in 11 Patients studied by Bianco et al.

<table>
<thead>
<tr>
<th>Time after Exercise</th>
<th>Immediate</th>
<th>5 min</th>
<th>10 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (untreated group)</td>
<td>-46 ± 6.9</td>
<td>-51 ± 5.6</td>
<td>-55 ± 5.9</td>
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<tr>
<td>Indoramin group</td>
<td>-25 ± 15.7</td>
<td>52 ± 15.8</td>
<td>56 ± 17.4</td>
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<tr>
<td>Difference (control group)</td>
<td>-21 ± 6.3</td>
<td>52 ± 5.8</td>
<td>56 ± 15.6</td>
</tr>
<tr>
<td>P (paired t test)</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
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<tr>
<td>P (Wilcoxon)</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
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