ly with this acceleration, so that her height for her bone age remains where it was at the time of diagnosis, on the third centile. This case demonstrates a chance association between Turner's syndrome and growth hormone deficiency. It emphasizes the importance of growth velocity in making pediatric endocrine diagnoses. Treatment will enable induction of puberty at the appropriate age without fear of making this patient's short stature worse. From the psychologic point of view this effect is most important and for this reason, as well as to attain a socially acceptable final height, this association is worth seeking.

London W1N 8AA, C. G. D. BROOK, M. D., M. R. C. P. England


Table 1. Immunologic Findings in the IgA-Deficient Angiimmune Deficiency Associated with Angioimmunoblastic Lymphadenopathy

<table>
<thead>
<tr>
<th>FINDING</th>
<th>CASE 1</th>
<th>CASE 2</th>
<th>CASE 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum immunoglobulins (mg/dl):</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IgA (90-330)*</td>
<td>30</td>
<td>80</td>
<td>15</td>
</tr>
<tr>
<td>IgM (41-280)</td>
<td>120</td>
<td>284</td>
<td>105</td>
</tr>
<tr>
<td>IgG (570-1,900)</td>
<td>540</td>
<td>600</td>
<td>1,100</td>
</tr>
<tr>
<td>Cell-membrane analysis of involved lymph nodes (%) of at least 200 cells examined:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phagocytosis of latex particles</td>
<td>2</td>
<td>4</td>
<td>17</td>
</tr>
<tr>
<td>Sheep erythrocyte rosette</td>
<td>10</td>
<td>40</td>
<td></td>
</tr>
<tr>
<td>IgG erythrocyte-antibody rosette</td>
<td>1</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Erythrocyte-antibody-complement rosette</td>
<td>12</td>
<td>5</td>
<td>24</td>
</tr>
<tr>
<td>Surface immunoglobulins (% of at least 200 cells examined):</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IgA</td>
<td>4</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td>IgD</td>
<td>2</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>IgE</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>IgG</td>
<td>6</td>
<td>6</td>
<td>12</td>
</tr>
<tr>
<td>IgM</td>
<td>16</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td>Kappa</td>
<td>14</td>
<td>7</td>
<td>13</td>
</tr>
<tr>
<td>Lambda</td>
<td>9</td>
<td>5</td>
<td>6</td>
</tr>
</tbody>
</table>

*Figures in parentheses denote normal values.
†Detected by immunofluorescence.

To the Editor: We read with interest the recent CPC (March 16, 1978) discussing angioimmunoblastic lymphadenopathy as an unusual disease having a lymphoma-like clinical picture, with serologic abnormalities of a collagen-vascular disease. However, unlike the finding in many of the rheumatoid diseases, the presence of IgA deficiency has not been noted. We have reviewed our recent cases of this syndrome (a total of nine patients) and noted that three of them were IgA deficient.

All three patients were men in the sixth or seventh decade of life, with one (Case 1) presenting with a proctodeum of nonderforming polyarthritis involving small and large joints with proximal muscle weakness. Delayed hypersensitivity as measured by common skin test antigens (streptokinase-streptodornase, PPD, mumps and dermatophytom) was absent. All patients were initially treated with corticosteroids, and two (Cases 1 and 3) died of Pneumocystis carinii pneumonia after chemotherapy.

Immunologic analysis of the IgA deficiency (Table 1) revealed the absence of free alpha chains in the serum, but two of the patients had light chains in the urine (kappa). Cell-marker analysis of mononuclear-cell suspensions from involved nodes was done in these patients according to previous methods. These studies demonstrated a paucity of lymphoid cells showing T-cell or B-cell surface properties.

The mechanism by which T-cell and IgA deficiencies arise in these patients and how they are related to each other remains unknown. However, our findings may have pathogenic relevance because patients with acquired IgA deficiency are known to have autoantibodies and IgA is known to be a T-cell-dependent antibody, being absent in nude mice that lack T cells. Hence, angioimmunoblastic lymphadenopathy may be a disease state in which loss of normal T-cell function, perhaps as a result of exposure to an exogenous agent, leads to altered cellular immunity (inhibition of delayed hypersensitivity skin reactions and susceptibility to pneumocystis), selective hypogammaglobulinemia and the appearance of autoantibodies.

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To the Editor: Alopecia, occurring after the use of cytotoxic chemotherapy, often presents an esthetic and psychologically disturbing problem and may on occasion result in refusal to accept a treatment that is of proved efficacy in a variety of solid tumors and hematologic neoplasms.

The use of a scalp tourniquet, which reduces the blood flow to the scalp and hair follicles during the intravenous administration and subsequent period of highest plasma cytotoxic concentration, has already been shown in noncomparative studies to reduce the frequency of alopecia both in single-agent chemotherapy with vincristine (N Engl J Med 283:1469, 1970) and multiple chemotherapy using vincristine, cyclophosphamide, 5-fluorouracil and methotrexate (Lancet 1:354, 1974).

We have performed a comparative study on 73 patients who have received monthly courses of Adriamycin, 30 to 50 mg on the first day, intravenously, cyclophosphamide, 300 to 600 mg on the third to fifth days, intravenously, and vincristine, 1 to 2 mg on the second day, intravenously, or VM 26, 50 mg on the second day, intravenously.

In hematologic cancer, prednisone, 45 mg per day given by mouth, was also administered from the first to seventh days.

Thirty-six patients received this chemotherapy regimen in conjunction with use of the scalp tourniquet. Five patients, however, could not tolerate the tourniquet because of discomfort and were therefore included in a group of 31 controls.

The tourniquet was placed in position immediately below the hairline and was inflated to a pressure of 50 mm Hg above systolic pressure. It was found that the pressure subsequently stabilized approximately 30 mm Hg above the systolic pressure. Five minutes later, the cytotoxic agents were injected. The tourniquet was maintained in position for a further 20 minutes and then removed.

Degree of hair loss was evaluated after a minimum of four courses of chemotherapy, with the following results: with the tourniquet, no
or minimal hair loss in 25 patients (67.5 per cent) and alopecia in 12 (32.5 per cent); and without the tourniquet, no or minimal hair loss in nine (29 per cent) and alopecia in 22 (71 per cent) (P < 0.005).

One may conclude from these results that the scalp tourniquet affords significant protection for patients exposed to a chemotherapy regimen with a high rate of hair loss as side effect. Although protection cannot be guaranteed for all patients, reasonable assurance may be given that hair loss is likely to be either avoided or only minimal.

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EVALUATING COMPUTED TOMOGRAPHY

To the Editor: I read with interest the Medical Progress article by Abrams and McNeil in the February 2 issue of the Journal, which reviewed the literature on computed tomographic scanning of the brain to early 1977. At the same time I was concerned about the numbers of neurodiagnostic studies performed at the Peter Bent Brigham and Beth Israel hospitals before and after the initiation of computed tomographic scanning. Although a breakdown for the type of problems studied by cerebral angiography and radionuclide scanning is not given, I would have to conclude that many patients who had a diagnosis made by computerized tomography have also had cerebral angiography and radionuclide scans. It is possible that the extensiveness of the angiographic procedure was decreased after installation of computed tomography, but this reduction is not so indicated. No explanation for this lack of decrease in cerebral angiographies or radionuclide scanning has been offered.

The number of radionuclide scans performed in this hospital has decreased by 70 per cent during the first year of computed tomographic scanning. When cerebral angiographies performed for cerebral vascular disease are excluded from the statistics (no change in the frequency of angiograms obtained for aneurysms or patients with transient ischemic attacks would be expected simply because of the introduction of computed-tomography capability), the number of cerebral angiographic procedures performed dropped by 80 per cent. We use cerebral angiography only in difficult situations such as making a differential choice between an unusual-appearing infarction and tumor, or to localize more accurately a small single metastatic nodule. Our neurosurgeons have been gaining confidence in operating on patients who have problems that appear quite straightforward by computed tomographic diagnosis alone. They have reported no adverse effects from this approach.

A large literature proclaims the tremendous diagnostic efficacy of computed tomographic scanning and how it will obviate more dangerous tests. The statistics of the Peter Bent Brigham group do not support this claim. In view of our own experience I must therefore conclude that some hospitals are giving great attention to the diagnostic capabilities of computerized tomography but are abdicating their role in offering education and direction for its efficient and appropriate use.

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We agree with the authors that one important evaluative approach would compare the information provided by this technic with other imaging technics under controlled conditions of observation. However, it is also critical to assess directly the effect of any added diagnostic information from computed body tomography on decision making in the clinical setting. The evaluation method that we have devised classifies patients according to clinical problems and relies on information obtained from the referring physician and patient records at different points in time. Our first six months' experience demonstrates that computed body tomography improves diagnostic understanding in 41 per cent of patients, increases physician confidence in previously chosen therapy in 50 per cent and contributes to a decision to change therapy in 17 per cent. The clinical contribution of computed body tomography varies with the particular area of examination and appears to be greatest in the mediastinum, retroperitoneum and pancreas.

We also believe more emphasis needs to be placed on the clinical importance of information provided by computed body tomography on the location and extent of pathologic process. We have extended the series of patients cited by the authors assessing its usefulness in planning radiation therapy, and continue to find that it frequently modifies planning decisions. Furthermore, the precise localization of retroperitoneal, pancreatic and lymph-node masses provided by computed body tomography often makes it the procedure of choice during the performance of needle aspiration-biopsy procedures, and, when positive, such biopsies can obviate the need for an exploratory laparotomy.

Despite our confidence in the contribution of computed body tomography in patients with certain clinical problems, we urge that clinical-efficacy evaluations be vigorously pursued. It is vital to extend our knowledge of the relationship between a patient's clinical presentation and the ultimate contribution of any examination, including computed body tomography. These kinds of information will provide physicians with the guidelines for the performance of computed body tomography and improve the efficacy with which this expensive technology is applied.

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To the Editor: The recent review by Abrams and McNeil omits mention of the hazards of the computed tomography. Although the technic is apparently considered to be benign, it is not sufficiently emphasized that the contrast-enhancement phase involves the intravenous administration of up to several hundred milliliters of iodinated contrast medium to patients of advanced age, as well as those suffering from diabetes or cardiac, liver or renal disease. The literature in English already contains three case reports of renal failure after contrast injection performed during this form of scanning. It is also likely that reports of the other possible reactions to scan contrast media will follow, including allergic-like reactions, neurogenic or vagally mediated aberrations of heart rate and blood pressure and direct cardiac toxicity.

In addition to the above complication we recently observed the precipitation of acute pulmonary edema in an elderly woman with congestive heart failure when she assumed the supine position in the scanner. It should also be emphasized that the scanner itself is so bulky as to insulate patients effectively from direct observation. Signs of decompensation of vital functions may thus be difficult to detect early. Clearly, the medical implications of computed tomog-