Table 1. Diagnoses in 30 Patients with Transient Proteinuria and Incidence Rates of Proteinuria.

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No. of Patients with Transient Proteinuria</th>
<th>Total No. of Patients</th>
<th>Incidence of Proteinuria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congestive heart failure</td>
<td>10</td>
<td>42</td>
<td>24%</td>
</tr>
<tr>
<td>Seizures</td>
<td>5</td>
<td>13</td>
<td>38%</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>4</td>
<td>11</td>
<td>36%</td>
</tr>
<tr>
<td>Infection, other sites</td>
<td>3</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Chronic obstructive pulmonary disease</td>
<td>2</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>6</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Fever *</td>
<td>8</td>
<td>49</td>
<td>16%</td>
</tr>
</tbody>
</table>

*Fever is considered independently, and these patients may have other diagnoses.

proteinuria. Other possible mechanisms include adrenergic discharge or fever accompanying the seizure. Once again, the proteinuria accompanying seizures is selective for albumin, suggesting a glomerular origin.

Our experience with febrile proteinuria (which occurred in 16 per cent of our febrile patients) suggests that this disorder may occur more frequently than Marks et al. have reported (5.6 per cent of febrile patients). In both series, respiratory infections were the most frequent causes of the fever (30 per cent in our current study and 30 per cent in that of Marks et al.). The mechanism may be the fever itself or an immunologic mechanism in which antigen-antibody complexes, stimulated by infection, are temporarily deposited on the epithelial side of the glomerular membrane.

Although albumin was the predominant urinary protein, three of our patients with pneumonia also had increased alpha globulin, which may represent the orosomucoid seen universally in Jensen and Henrikson’s patients. In their study, increased excretion was thought to have a prerenal basis, since the serum concentration of the protein was also elevated, probably as an acute-phase reactant.

The remaining cases of transient proteinuria have little in common other than the physiologic stress of the acute illness that prompted admission and the accompanying psychological stress. Presumably, these stresses affect all patients with acute medical illnesses, so we are at a loss to explain why only some patients had transient albuminuria.

On the basis of these findings, the clinician who notes proteinuria with normal renal function in patients with congestive heart failure, seizures, or febrile illnesses need not begin diagnostic work-up for proteinuria. A repeat urinalysis 10 days after admission should confirm the diagnosis of transient proteinuria.

References

Evidence for a Secular Trend in Age of Menarche

Grace Wyshak, Ph.D., and Rose E. Frisch, Ph.D.

We present data documenting a secular trend toward an earlier age of menarche in Europe and the United States in the past century. There has been recent controversy on whether such a change has taken place. We have reviewed 218 reports on age of menarche in Europe from 1795 to 1981, covering 220,037 individuals. The historical European data are mainly from Backman’s extensive collation.

Figure 1 and Table 1 show that in Europe the age of menarche has become earlier by two to three months per decade in the past century and a half. The greatest rate of decline, 3.2 months per decade, has been observed in the Scandinavian countries. The smallest rate of decline, 1.1 month per decade, has been observed in France.

Although the European historical data are undoubtedly variable in quality and method of collection, the overall trend is statistically significant (Table 1) and consistent with the well-documented acceleration in height and weight of girls and boys in the past century. When the rate of growth levels off with optimal conditions, the age of menarche also levels off. Also consistent with the data on growth is the disappearance of rural—urban differences in age of menarche and of differences associated with social class, occurring as socioeconomic conditions became more equitable in 20th-century populations.

The data from the United States also indicate a secular trend in age of menarche of about two months per decade in the past century. The average age of menarche was 14.73 years in Bowditch’s pioneer study in 1877, about 14 years at the turn of the century, and 12.8 years in 1947. The downward trend apparently leveled off at about 12.8 years; recent ages of menarche are still the same (Fig. 1). Between 1900 and 1945 in the United States, a downward trend

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in age of menarche of about three months per decade occurred in mothers of twins or of singletons, correlating with a more rapid gain in body weight (Wyshak G: unpublished data).

The findings in Europe and the United States are consistent with the fact that menarche is delayed by undernutrition \(^5,\,8,\,14\) and strenuous physical exercise. \(^15,\,17\) For example, the age of menarche is still relatively late, about 15 years, among poor girls in developing countries \(^5,\,18\) and among dancers \(^15,\,16\) and athletes \(^17\) in affluent countries. Menarche may also be delayed in girls living at high altitudes, in association

### Table 1. Secular Trend in Age of Menarche in Europe.

<table>
<thead>
<tr>
<th>Country</th>
<th>No. of Samples (No. of Individuals)</th>
<th>Time Period</th>
<th>Regression Equation *</th>
<th>Decline in Months/Decade</th>
<th>Correlation Coefficient (r)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Europe †</td>
<td>218 (220,037)</td>
<td>1795–1981</td>
<td>Y = 51.74 – 0.0192±0.0015X</td>
<td>-2.3</td>
<td>0.658</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Europe excluding France</td>
<td>170 (192,178)</td>
<td>1795–1981</td>
<td>Y = 62.38 – 0.024±0.0014X</td>
<td>-3.0</td>
<td>0.801</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Scandinavia</td>
<td>68 (83,957)</td>
<td>1839–1972</td>
<td>Y = 66.88 – 0.0270±0.0018X</td>
<td>-3.2</td>
<td>0.879</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Germany</td>
<td>63 (76,800)</td>
<td>1795–1939</td>
<td>Y = 48.65 – 0.0172±0.0028X</td>
<td>-2.0</td>
<td>0.619</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>France</td>
<td>47 (26,959)</td>
<td>1830–1967</td>
<td>Y = 32.50 – 0.0094±0.0020X</td>
<td>-1.1</td>
<td>0.570</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>England §</td>
<td>19 (29,427)</td>
<td>1832–1981</td>
<td>Y = 45.54 – 0.0162±0.0022X</td>
<td>-1.9</td>
<td>0.870</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

*Y denotes age of menarche, and X calendar year.
†Historical data are from Backman;\(^7\) and data on Holland and England (1981)\(^8\) are included. Recent ages of menarche for Belgium, Czechoslovakia, Hungary, Italy, Poland, Romania, Russia, Spain, and Yugoslavia (symbol \(\circ\) in Figure 1), are from Eretele and Tanne;\(^4\) (no numbers given). If the 17 samples from these nine countries are omitted from the total for Europe, the regression equation is: \(Y = 43.01 – 0.015(0.0017)X\); the decline is 1.9 months per decade; \(r = 0.544; P < 0.001\).
with slower rates of physical growth\textsuperscript{5, 8} (Fig. 1). In Bangladesh, unfortunately, there has recently been a trend toward a later age of menarche accompanying adverse economic conditions and a deterioration in the diet.\textsuperscript{10} In evaluating the age of menarche of a population or an individual girl, it is important to consider these environmental factors.

\textbf{REFERENCES}


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\textbf{founded by RICHARD C. CABOT}

\textbf{ROBERT E. SCULLY, M.D., Editor}
\textbf{EUGENE J. MARK, M.D., Associate Editor}
\textbf{BETTY U. McNEELY, Assistant Editor}

\textbf{CASE 17-1982}

\textbf{Presentation of Case}

A 77-year-old man was admitted to the hospital because of fever and subcutaneous masses.

He was well until three months previously, when a dry cough developed. Two months before admission the temperature rose to 38.9°C, accompanied by chills, pleuritic pain, and anorexia. Erythromycin was administered for two weeks, without improvement. He was admitted to another hospital. X-ray films of the chest showed small bilateral pleural effusions. A tuberculosis skin test was markedly positive. Repeated blood cultures and cultures of urine and pleural fluid yielded no microorganisms. Cytologic examination of the pleural fluid and microscopical examination of a pleural-biopsy specimen were negative; no acid-fast bacilli were observed. An intravenous urographic examination disclosed a large, irregular mass invading the base of the bladder, especially on the right side; there was slight dilatation of both ureters, and a small diverticulum projected from the right wall of the bladder; the upper urinary tracts and kidneys appeared normal. A computed tomographic (CT) scan of the abdomen showed a solid mass involving the right posterolateral portion of the bladder wall and considerable enlargement of the prostate gland; a diverticulum of the bladder was observed on the right side; the pancreas and retroperitoneum appeared normal; there was no evidence of an abscess or lymphadenopathy. The fever subsided, and the pleural effusions resolved. The patient left the hospital before a barium-enema examination and upper gastrointestinal series were accomplished. Soon after discharge fever recurred. One or two weeks before entry tender masses appeared in the left thigh and the right gluteal region. One week before admission he was first seen at this hospital. An intravenous urographic examination disclosed probable calcification in the retropublic area and degenerative changes in the lower lumbar spine; there was prompt symmetric opacification of the collecting systems, and nephrotomographic examination disclosed normal renal parenchyma and outlines; the ureters appeared normal; the bladder was trabeculated, with a diverticulum on its right side and an irregular impression on the bladder base; there was a moderate postvoiding residuum of urine. On follow-up examination one week later fever persisted, and the patient was admitted to this hospital.