with as little tapping as possible to insure the normal functional capacity of the harvested neutrophils.

John Milton Mishler IV, D. Phil., Chief
Blood Resources and Transplantation Branch
Division of Blood Diseases and Resources
National Heart, Lung, and Blood Institute
National Institutes of Health
Federal Building, Room SA08
Bethesda, Md. 20205

To the Editor:

Chediak et al. observed lower levels of factor VIII coagulant activity in carriers of classic hemophilia who inherited the faulty gene from their mothers than in the carriers who inherited the gene from their mothers and postulated that the paternal X-chromosome might be selected preferentially over the maternal X-chromosomes in female embryos. If this hypothesis is true, one would expect to find a similar bias in factor IX levels among carriers of hemophilia B, which is also a sex-linked genetic disorder.

I reviewed data on carriers of hemophilia B reported earlier, excluding women who had more than one son with hemophilia but had no antecedent relative with hemophilia because such women cannot be classified clearly as maternal or paternal carriers. Levels of factor IX coagulant activity were compared in maternal and paternal carriers of all ages, and in smaller carrier groups with comparable age distributions (Table I). Levels of factor IX antigen were compared in maternal and paternal carriers from kindreds with hemophilia B+ and hemophilia B—. No significant differences between values for maternal and paternal carriers were found.

Our data, on a limited number of women, do not support the hypothesis of Chediak et al.

Carol K. Kasper, M.D.
Orthopaedic Hospital and University of Southern California School of Medicine Los Angeles, Calif.

REFERENCES


We have counseled 174 women from hemophilia A families during the past 5 yr, 29 of whom were obligatory carriers. Fourteen were maternal carriers, 15 were paternal carriers. Their ages and VIII:C levels are shown in Table 1.

Table 1.

<table>
<thead>
<tr>
<th>Type of Carrier</th>
<th>Mean ± SEM</th>
<th>Range</th>
<th>Mean ± SEM</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>14 Maternal</td>
<td>38.5 ± 2.4</td>
<td>25–49</td>
<td>79.7 ± 9.9</td>
<td>28–167</td>
</tr>
<tr>
<td>15 Paternal</td>
<td>17.1 ± 2.1</td>
<td>3–35</td>
<td>69.7 ± 10.7</td>
<td>22–167</td>
</tr>
</tbody>
</table>

A significant difference in age was both expected and observed between the groups, since an obligatory carrier of the maternal type by definition must have borne at least one affected male (or carrier female); on the other hand a majority of our paternal carriers were unmarried, 6 being under 15 yr of age. The mean VIII:C level of the paternal carriers was slightly but not significantly lower than the maternal carriers (t = 0.683, 27 df, p = 0.50). Thus, our data do not support the findings of Chediak et al. I urge other centers to examine their data in a similar manner and publish them.

John B. Graham, M.D.
Professor of Pathology
University of North Carolina
Chapel Hill, N.C.

REFERENCE
Factor VIII in hemophilia [letter]