Alterations in thickness of glomerular capillary basement membrane (BM) in various diseases have been histopathologically controversial. The author tried electron microscopically to evaluate the thickness in healthy subjects and analyzed the changes seen in some pathological conditions.

The width of BM was measured in 49 glomeruli of 21 individuals. In 7 of them, with 26 glomeruli studied, detailed renal function tests showed normal values. The measurements were taken between the endothelial cytoplasmic membrane, as it is attached to the BM, and the outer lining of the lamina rara externa underneath the cytoplasmic membrane of epithelial foot processes. The width was measured only in the peripheral portions, at intervals of 1μ. To avoid tangential plane of sections, portions of the capillary wall were not measured in which the attached epithelial and endothelial cytoplasmic membranes were not clearly visible or where
the endothelial pores were seen circularly. Calculating from the total number of 7670 sites of measurements, the mean width was 3146 Å, with the standard deviation of 983 Å. The range of mean width of all glomeruli varied from 2248 Å to 5000 Å, indicating differences in the BM thickness in healthy subjects. If the over-all mean plus twice the standard deviation is accepted as the upper limit of "normal", only a mean width exceeding 5112 Å must be regarded as significantly "abnormal" thickness. The results also showed that a minimum of 125 measurements was necessary to estimate a mean width within a 95 per cent confidence limit.

In diabetics the relation between the thickening of glomerular BM and the thickening of intercapillary tissue is still in controversy. Renal biopsies of 30 diabetics and sections from 2 autopsies were used for study and 45 glomeruli from them were examined in the same manner as in healthy controls. Calculation was based on a total of 7140 measurements. All of 25 cases without nodules had the mean width of their peripheral capillaries within normal limit (less than 5112 Å). On the other hand, BM of 4 of the 7 cases with nodular lesions exceeded the maximal upper limit (more than 5112 Å). The residual 3 cases with nodules were within the limit of controls. The study of this series showed that the thickening of peripheral capillary BM of diabetics occurred only in glomeruli with nodular lesions. Conversely, nodular glomerulosclerosis could occur without thickening of peripheral BM.

These data do not confirm the statements that all diabetics have thickened glomerular peripheral BM, and that the thickening of the peripheral capillary BM precedes formation of nodules.

The glomerular BM can give the appearance of thickening with some kinds of deposit around the lamina densa. This can be the case of amyloid kidney in which bundles of peculiar amyloid fibrillae are distributed focally and irregularly around the faint lamina densa. Dense, homogeneous substance may deposit between the lamina densa of BM and the endothelial or mesangial cells (variant E deposit), diffusely in cases of lupus nephritis, giving the thickened appearance of BM. The so-called "membranous" change of BM is characterized by irregular outer surface and tremendously thickened appearance, and is usually consisted of irregularly spi-
ked, towards the epithelial side, or split lamina densa with deposits of various densities (Fig. 1). The “hump”, deposit between glomerular epithelium and BM, has usually normal or minimally thinned three layers of BM and is specifically observed in cases of acute glomerulonephritis, in which the daily protein excretion is relatively small in amount (less than 2.5 g in our series studied). On the other hand, the so called variant B deposit, which is differentiated from the hump by the presence of more or less spiked lamina densa around it, giving the appearance

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**Fig. 1.**
Membranous change of glomerular basement membrane seen in a case of lupus erythematosus with nephrotic syndrome.

- **Cap**: capillary lumen
- **Ed**: endothelial cell
- **Ep**: epithelial cell
- **R**: red blood cell
- **BM**: basement membrane

Arrow indicates spike-like protrusion of lamina densa.
Variant B deposit in glomerular basement membrane.

U : urinary space       Ep : epithelial cell
B : variant B deposit   Ld : lamina densa
Ed : endothelial cell   Cap : capillary lumen

The deposit is less compact and is more or less buried in lamina densa.

of deposit buried in the epithelial side of lamina densa (Fig. 2), is seen mostly in cases with large amount of proteinuria (Table). As two cases of nephrotic syndrome with variant B deposit were confirmed to progress, one year or so later, into the membranous type of change, it is suggested that the variant B deposit might participate in development of membranous changes.

Extreme thinning or discontinuity of BM is observed often in cases of acute glomerulonephritis and lupus nephritis, and is probably participated in hematuria or proteinuria of high rate of globulin content, clinically seen in these cases.
Table. Glomerular lesions related to Variant B-deposit.

<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>Sex</th>
<th>History Protein g/24h</th>
<th>Urine Protein Alb./glob.</th>
<th>Serum Protein Cholesterol</th>
<th>Duration</th>
<th>Light Microscopy</th>
<th>Clinical Diagnosis</th>
<th>Electron Microscopy (Deposits)</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>35</td>
<td>F</td>
<td>+ 13.0</td>
<td>+</td>
<td></td>
<td>20W</td>
<td>Diffuse GNT (Lobular)</td>
<td>NTS + Nephrotic Synd.</td>
<td>A B E</td>
</tr>
<tr>
<td>2</td>
<td>17</td>
<td>F</td>
<td>+ 5-8</td>
<td>+</td>
<td></td>
<td>3W</td>
<td>&quot; ( &quot; )</td>
<td>&quot;</td>
<td>A B E</td>
</tr>
<tr>
<td>3</td>
<td>22</td>
<td>M</td>
<td>- 2.2</td>
<td>+ 2.88/3.19</td>
<td></td>
<td>30W</td>
<td>&quot;</td>
<td>Lupus Nephritis</td>
<td>A B E</td>
</tr>
<tr>
<td>4</td>
<td>37</td>
<td>M</td>
<td>- 10</td>
<td>- 1.5 /3.5</td>
<td>467</td>
<td>4W</td>
<td>Membranous GNT.</td>
<td>Nephrotic Synd.</td>
<td>A B</td>
</tr>
<tr>
<td>5</td>
<td>50</td>
<td>F</td>
<td>? 9-12</td>
<td>- 1.9 /3.88</td>
<td>395</td>
<td>?</td>
<td>Nodular Glomerulosclerosis</td>
<td>Diabetic Nephropathy</td>
<td>B</td>
</tr>
<tr>
<td>6</td>
<td>16</td>
<td>F</td>
<td>? 9</td>
<td>- 1.3 /3.1</td>
<td>612</td>
<td>10Y</td>
<td>Diffuse GNT.</td>
<td>Lupus Nephritis</td>
<td>B E</td>
</tr>
<tr>
<td>7</td>
<td>78</td>
<td>M</td>
<td>? 4-10</td>
<td>- 1.5 /2.7</td>
<td>611</td>
<td>6W</td>
<td>Focal GNT.</td>
<td>Nephrotic Synd.</td>
<td>B</td>
</tr>
<tr>
<td>8</td>
<td>36</td>
<td>M</td>
<td>(? #)</td>
<td>-</td>
<td>?</td>
<td>Membranous GNT.</td>
<td>&quot;</td>
<td>&quot;</td>
<td>B</td>
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References