CALCIFICATION OF THE SUPRARENAL GLANDS OF CATS.

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PLATE 11.

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It is for the purpose of calling attention to a little known but apparently common disease of the suprarenal glands of cats that the following observations are published.

The only extensive paper on calcification of the suprarenal glands of cats is that of Brüschweiler (1) who reported finding 15 instances in 303 cats examined at Berne. He thought that the incidence of the disease increased with age and that it was probably dependent upon some disturbance of calcium metabolism secondary to chronic inflammations of the intestines. Kruse (2) in reporting an instance of bone formation in the suprarenal medulla of a monkey mentions that Wislocki showed him a calcified suprarenal of a cat.

During the past 5 years we have observed 64 cases of calcification of the suprarenals in 257 cats—approximately 25 per cent. Of the 257 cats, 6 were born and reared in the laboratory, but in none of these was calcification found. The remaining 251 were street cats whose average stay in the laboratory was less than 1 month. All of the 64 instances of calcification were, therefore, found in the street cats. As regards sex, 32 were females, 31 males, and in 1 the sex was not recorded. As regards age, we grouped them as follows: old adults, 7; adults, 21; young adults, 23; kittens, 13. While the ages are only approximate the groups are relatively accurate. Only those with milk teeth present were called kittens. Old adults included those with most of the incisors and canine teeth absent. Young adults included those with complete second sets of clean, white teeth, and adults those with one or more incisor teeth absent and more or less yellowish discoloration of those present.

We have seen this form of calcification only in cats. In a series of 328 dogs and approximately 2300 rabbits whose suprarenals were
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examined microscopically, no cases were observed. Through the
courtesy of Dr. C. H. Lenhart, I have examined his preparations of
150 pig, 200 sheep, and 125 cattle suprarenals obtained from the
slaughter house without finding a case of this kind of calcification
(calcification of the suprarenals in tuberculous and other local inflam-
matory conditions while very common in these animals has no relation
to the present subject). A few cases of calcification of the suprarenals
have been recorded in man. Those reported by Newsam (3) and Vic-
tor (4) may be referred to. Newsam's case, a child of 2 years and 7
months, apparently healthy, suddenly became unconscious and died
in 14 hours. Autopsy showed calcification of the fascicular and retic-
ular zones with true bone formation in the medulla. Victor's case
was similar except that there was no ossification and the calcification
was confined to the two inner layers of the cortex.

Clinical Manifestations.

The disease seems always associated with distemper. It is there-
fore more common in the young, and during the fall and spring months.
Only a portion of the cats having distemper develop calcification,
and in the cases of distemper ending fatally, it is often absent. It
usually develops 2 to 3 weeks after the infection is manifest, and while
recovery from distemper is usual, the calcification remains, and we
have formed the impression that though extensive it may remain for
years with the animal in good health. The condition may be sus-
pected in its severe forms if the cat after 2 or 3 weeks of distemper
fails to improve, loses weight, becomes weak and ataxic, the fur
becomes rough, and it shows little desire for food—symptoms which
resemble those seen in cats surviving double suprarenalectomy for 2
or 3 weeks.

Pathological Anatomy.

The most characteristic feature is the presence of areas of calcifica-
tion in the fascicular zone (see Figs. 1 to 3). There are all degrees
from the smallest areas, detectable only with the microscope, to almost
complete calcification of the entire cortex. The process begins in the
middle zone as small foci of degenerating cells at first without calcium
deposition. Then as the lesion advances, calcium is deposited and in
the severest cases it extends through the glomerular and reticular layers. In the more advanced cases, it is readily detected on gross examination as smaller or larger pale yellowish areas on the surface of the gland. When the calcification is generalized, the gland may be fractured between the fingers in the same way as a calcified artery wall.

### Table 1.

**Suprarenals with Calcification.**

<table>
<thead>
<tr>
<th>Cat No.</th>
<th>Weight of suprarenal (dried)</th>
<th>Calcium.</th>
<th>Remarks.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mg.</td>
<td>mg. per cent</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>54.1</td>
<td>0.11</td>
<td>0.2</td>
</tr>
<tr>
<td>2</td>
<td>67.1</td>
<td>2.76</td>
<td>4.1</td>
</tr>
<tr>
<td>3</td>
<td>73.0</td>
<td>3.81</td>
<td>5.2</td>
</tr>
<tr>
<td>4</td>
<td>32.7</td>
<td>2.77</td>
<td>8.5</td>
</tr>
<tr>
<td>5</td>
<td>92.2</td>
<td>8.68</td>
<td>9.4</td>
</tr>
</tbody>
</table>

**Suprarenals without Calcification.**

<table>
<thead>
<tr>
<th>Cat No.</th>
<th>Weight of suprarenal (dried)</th>
<th>Calcium.</th>
<th>Remarks.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mg.</td>
<td>mg. per cent</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>49.8</td>
<td>0.01</td>
<td>0.02</td>
</tr>
<tr>
<td>7</td>
<td>46.9</td>
<td>0.01</td>
<td>0.02</td>
</tr>
<tr>
<td>8</td>
<td>55.2</td>
<td>0.03</td>
<td>0.05</td>
</tr>
<tr>
<td>9</td>
<td>49.6</td>
<td>0.03</td>
<td>0.06</td>
</tr>
<tr>
<td>10</td>
<td>23.8</td>
<td>0.05</td>
<td>0.08</td>
</tr>
<tr>
<td>11</td>
<td>38.2</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Our series supports Brüschweiler's observation that it is always calcification and never ossification. We have seen very few cases where the medulla is at all involved and in these it was clearly secondary. In the early stages the calcification is soft and amorphous, while in the long standing and recovered cases the calcification is hard, crystalline, and usually such areas have a fibrous capsule. At no stage have we observed evidence of inflammatory exudate about the foci. In
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The early stages, calcium is probably deposited in the form of soap, while in the later stages it changes to carbonate and phosphate. A few of the glands have been examined for calcium; the figures are presented in Table I.

The sequence of events leading to the deposition of calcium seems to be somewhat as follows: The toxin of distemper produces a specific degeneration of groups of cells in the fascicular layer, lime salts are deposited in these foci at first combined with fatty substances, later going over to carbonate and phosphate.

EXPERIMENTAL OBSERVATIONS.

We have not succeeded in producing the lesion. Sublethal injury and necrosis of the cortex by diphtheria toxin and by arsenic have been tried. In a large series of anesthetized rabbits and cats the cortex was frozen with ethyl chloride. Partial ligation of the blood vessels and trauma causing extensive hemorrhage into the cortex were tried. All of these procedures gave negative results. In some of the cats in which these experimental injuries were produced, calcification of the suprarenals was found at autopsy but the incidence was not greater than in those not so treated. Hence the calcification was probably present before the experimental injury (see Fig. 4). It seems, therefore, that the toxin producing the initial degeneration of the cells of the fascicular zone which leads to calcification is a highly specific one. It does not occur in association with ordinary bacterial infections as for example chronic wound infection or chronic pneumonia with empyema. Likewise it does not occur following the usual types of experimental injury as mentioned above.

SUMMARY.

Calcification of the fascicular zone of the cortex has been observed in 64 of 257 cats. It is always calcification and never ossification. It is more common in young animals and in our experience is associated with distemper. In its severe forms it may be recognized clinically. The symptoms resemble those seen in cats surviving double suprarenalectomy for 2 to 3 weeks. The toxin producing the focal degeneration is clearly a very specific one since attempts to produce such lesions by several types of experimental injury have failed.
The sequence of events appears to be similar to that present in other degenerative processes associated with calcification, namely cell injury and necrosis, deposition of calcium at first as fatty compounds which later change to carbonate and phosphate. It is suggested that this lesion should be considered in interpreting experiments in which cats are used.

BIBLIOGRAPHY.


EXPLANATION OF PLATE II.

Fig. 1. Illustrates the distribution of the calcification in relation to medulla and the cortical zones.
Fig. 2. Higher magnification of two small early foci.
Fig. 3. Very early lesion showing cell degeneration and beginning of calcification.
Fig. 4. Calcification in a gland frozen with ethyl chloride 25 days before removal. The degeneration and necrosis and cholesterol spaces are the result of freezing. The calcification was probably present at the time of freezing.
(Marine Calcification of suprarenal glands of cats.)