From the Third Medical Clinic, Kyoto University Hospital
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Experimental Chronic Neurogenic Hypertension in Dogs with Piarachnitis Adhesiva Thoracolumbalis Spinalis (II)
Chronic Effect of Alumina-Cream-Induced Piarachnitis Adhesiva upon the Renal Function and the Arterial Blood Pressure

BY

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Maekawa's theory concerning the probable pathogenesis of essential hypertension proposed in 1954 stands as follows:
(1) The arteriolar vasospasm in essential hypertension is ascribable to disturbances in ATP-ATPase system of blood vessels including the kidneys.
(2) These disturbances in ATP-ATPase system in blood vessels are due to functional derangement in the kidneys which occur as the result of renal ischemia.
(3) This renal ischemia is due to the increased tonic sympathetic discharge to the renal vascular beds from the renal vasomotor center of the spinal cord.
(4) This increased tonic sympathetic discharge from the spinal renal vasomotor center is brought into existence by the activity of subclinical adhesive arachnoiditis localized to the region of the spinal renal vasomotor center.

This hypothesis has been considerably substantiated by the author's previous experiment in dogs, in which it was shown that hypertension of 13 to 16 week duration occurred, after a latent period of 1 to 3 weeks, following the intrathecal injection with laminectomy of alumina cream fluid at the level of the 1st lumbar vertebra, an experimental procedure to produce localized spinal adhesive arachnoiditis at the level of the injection.

The present experiment, an extension of the previous one, aims to test the presence or absence of the postulated renal ischemia in such an experimental hypertension.

For this purpose alumina-cream-induced-experimental adhesive arachnoiditis was produced in dogs and these dogs were examined at frequent intervals for variations in the renal blood flow and other clearance data and for variations in blood pressure, so that it will be possible to see whether there is the diminution in renal blood flow concomitantly with the rise in the blood pressure.

METHODS

(1) Animals
Fifteen mongrel dogs have been used. Those dogs with pre-existing hypertension or abnormal electrocardiograms and those restless during blood pressure recording have been excluded from the series.

(2) Blood Pressure Measurements
Dogs, unanesthetized, were fixed on a table in supine position and blood pressures were read off from U-shaped mercury manometer connected to the femoral artery. The needle used for puncture was measured 0.7 mm. in diameter internally and physiological saline solution with heparin was used for preventing the blood from clotting inside the recording system. Pressures were taken each time after the dog has been fixed on the table and lain quite with stabilized blood pressure for more than 5 minutes.

(3) Renal Function Tests
Eight dogs have been subjected to renal function tests. Fixed unanesthetized to the table in supine position, the dogs were examined for thiosulfate and PAH clearance data by the method of constant intravenous infusion. "Sampling" has been made two or three times at intervals of 15 minutes and the glomerular filtration rate (GFR), the effective renal plasma flow (RPF), and, from hematocrit value, the renal blood flow (RBF) were obtained as the average of 2 or more stabilized values.

(4) Alumina Cream
One volume of 1% alum ammoniacal water was
added to one volume of 1% ammonium water and the resulting precipitate was washed throughly with centrifugation (3000 RPM for 5 minutes) with aseptic distilled water (4). This final precipitate was used each time after diluting it with 3 to 10 volumes of physiological saline solution.

(5) Intrathecal Injection of Alumina Cream

Seven dogs, which have been subjected to consecutive determinations of blood pressure and renal clearance, were injected, after laminectomy, with one ml. each of alumina cream fluid at different concentrations under sodium amobarbital anesthesia intraperitoneally in dose of 50 mg. per kg. body weight; at 1:3 dilution in 3 dogs, at 1:10 dilution in 4 dogs in various combinations of the dilution and the level of the injection, as indicated in Table I, e.g. at the vertebral levels of T4, T9, L1 and L4 in 1, 1, 4 and 1 dog, respectively.

The injection, which was made through a small incision to the dura mater, was facilitated by a vinyl-tubing connected to a small reservoir, shown in Fig. 1., which supplied alumina cream fluid during the slow injection and was then buried in situ beneath the fascia. The tip of tubing thus remained permanently within the pia-arachnoid space. This device of the reservoir is that of Natori.

Fig. 1. Schematic representation of the relationships between the spinal cord, dura and the indwelling alumina-cream-filled vinyl reservoir with tubing.

Seven dogs have been used as controls. As shown in Table I they were laminectomized at the same levels with the dogs of the previous group, e.g. at the vertebral level of T9, L1, L4, but were injected, not with alumina cream, but with sodium dicetyl phosphate suspension in dose of 1 ml. (No. 514, No. 534, No. 502) or with physiological saline solution in dose of 1 ml. (No. 520, No. 536, No. 466, No. 504), all with the aid of a vinyl tubing and reservoir which were then buried in situ. The rest one dog (No. 493) simply received laminectomy at the vertebral level of T9 and no intrathecal injection was made.

(6) Post-operative Observations

Following the operation, the dogs were reexamined at various intervals for blood pressure, urine, electrocardiogram and general conditions, and, in some dogs for renal clearance, for the period of approximately 3 months, whereupon some of them were sacrificed and various organs were studied for histological alterations with hematoxylin-eosine staining and Weigert van Gieson elastic staining.

RESULTS

(1) General Conditions

Following the operation some of the dogs exhibited temporary, slight motor disturbances of the hind limbs, but recovered soon, excepting one or two dogs which exhibited motor disturbances of the hind limbs till the time of writing of this paper. Around the end of two or three post-operative months some of dogs, well tempered previously, became somewhat rough and excitable, but this change subsided soon, and no dogs showed any convulsive fits. There were no changes in appetite and the dogs remained essentially quite healthy.

(2) Electrocardiograms

Electrocardiograms remained essentially unchanged between the pre-operative and the 2nd post-operative month.

(3) Urine

Slight albuminuria appeared in occasional dogs following the operation, which subsided soon. There was no instance of microscopic hematuria and the urine essentially remained unchanged before and after the operation.

(4) Mean Blood Pressure

Pressure readings, obtained by U-shaped mercury manometer, in pre-operative and post-operative observations are listed in Table I (A). Inspection of the table indicates that there was a rise in the mean blood pressure by 15 mm.Hg or more in 2 of the 3 dogs treated with 1.0 ml. of alumina cream fluid at 1:3 dilution injected intrathecally (No. 474, No. 477, No. 513).

Of this group, dog No. 474 developed a rise in blood pressure by 15 mm.Hg in one week, which continued on the 2nd week where the rise was by 21 mm.Hg, followed by a fall in 4th and 5th week down to a level lower than the pre-operative level, again followed by a rise in the 6th week by 15 to 17 mm.Hg. In the 7th week the blood pressure dropped again, followed again by a rise in the 8th week to a 27 mm.Hg.
### Table 1. (A) Response of Blood Pressure in mmHg

<table>
<thead>
<tr>
<th>Substance</th>
<th>Dilution</th>
<th>Volume</th>
<th>Site of Operation</th>
<th>Initial B. P.</th>
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<tbody>
<tr>
<td>AC</td>
<td>AC</td>
<td>1:3</td>
<td>L1</td>
<td>125</td>
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<td>L1</td>
<td>134</td>
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<td>L1</td>
<td>130</td>
</tr>
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<td></td>
<td>AC</td>
<td>1:3</td>
<td>T9</td>
<td>105</td>
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<td>125</td>
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<td></td>
<td>AC</td>
<td>1:3</td>
<td>L1</td>
<td>135</td>
</tr>
<tr>
<td></td>
<td>SDP</td>
<td>1:10</td>
<td>T4</td>
<td>128</td>
</tr>
<tr>
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<td>SDP</td>
<td>1:10</td>
<td>L4</td>
<td>133</td>
</tr>
<tr>
<td></td>
<td>SDP</td>
<td>1:10</td>
<td>L4</td>
<td>122</td>
</tr>
<tr>
<td></td>
<td>SS</td>
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<td>SS</td>
<td>1:1</td>
<td>L4</td>
<td>140</td>
</tr>
<tr>
<td></td>
<td>none</td>
<td></td>
<td>T9</td>
<td>139</td>
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1. week  
2. week  
3. week  
4. week  
5. week  
6. week  
7. week  
8. week  
9. week

### Table 1. (B) Response of Renal Blood Flow in Per Cent

<table>
<thead>
<tr>
<th>Initial Value</th>
<th>451</th>
<th>382</th>
<th>247</th>
<th>316</th>
<th>352</th>
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<td>19</td>
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<td>6</td>
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<td>3</td>
<td>1</td>
<td>7</td>
<td>2</td>
</tr>
<tr>
<td>6. week</td>
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<td>12</td>
<td>28</td>
<td>9</td>
<td>15</td>
<td>5</td>
<td>11</td>
<td>17</td>
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<tr>
<td>7. week</td>
<td>7</td>
<td>8</td>
<td>15</td>
<td>15</td>
<td>1</td>
<td>7</td>
<td>8</td>
<td>3</td>
</tr>
<tr>
<td>8. week</td>
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<td>6</td>
<td>5</td>
<td>2</td>
<td>8</td>
<td>9</td>
<td>7</td>
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### Table 1. (C) Response of Renal Vascular Resistance in Per Cent

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<th>Initial Value</th>
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<th>354</th>
<th>543</th>
<th>332</th>
<th>336</th>
<th>741</th>
<th>424</th>
<th>362</th>
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<td>2. week</td>
<td>6</td>
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<td>16</td>
<td>8</td>
<td>6</td>
<td>26</td>
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<td>6</td>
</tr>
<tr>
<td>3. week</td>
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<td>-2</td>
<td>36</td>
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<td>1</td>
<td>4</td>
<td>13</td>
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<td>18</td>
<td>8</td>
<td>3</td>
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<td>12</td>
<td>9</td>
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<td>45</td>
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<td>9</td>
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<td>-4</td>
<td>2</td>
<td>17</td>
<td>6</td>
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### Table 1. (D) Response of Glomerular Filtration Rate in Per Cent

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<th>Initial Value</th>
<th>62.2</th>
<th>101</th>
<th>42.6</th>
<th>99.3</th>
<th>117</th>
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<tr>
<td>2. week</td>
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<td>13</td>
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<td>4</td>
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<td>2</td>
</tr>
<tr>
<td>3. week</td>
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<td>-4</td>
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### Table 1. (E) Response of Filtration Fraction in Per Cent

<table>
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<tr>
<th>Initial Value</th>
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<th>.414</th>
<th>.266</th>
<th>.482</th>
<th>.480</th>
<th>.532</th>
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<td>27</td>
<td>10</td>
<td>2</td>
<td>8</td>
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<td>21</td>
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<td>14</td>
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<tr>
<td>3. week</td>
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<td>8</td>
<td>7</td>
<td>21</td>
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<td>14</td>
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<tr>
<td>4. week</td>
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<td>10</td>
<td>16</td>
<td>21</td>
<td>13</td>
<td>14</td>
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AC: Alumina Cream, SDP: Sodium Dicyethyl Phosphate, SS: Physiological Saline Solution

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level, followed again by a fall. The over-all variations in the blood pressures during this 8 week period are characterized by 3 peaks and 3 depressions in blood pressure. In the dog No. 513 blood pressure began to rise to a +20 mm.Hg level in the 5th week, which continued on to the 6th week (+24 mm.Hg) and to the 7th week (+15 + 18 mm.Hg) and the pressure returned nearly to the previous level in the 8th week.

In the group of 4 dogs treated with 1.0 ml. of alumina cream at 1:10 dilution intratheceally, the rise of 15 mm.Hg or more occurred only in one dog (No. 542, No. 544, No. 541, No. 521). Of the total of 7 alumina-cream-treated dogs described above, recorded peak blood pressure exceeded 150 mm.Hg only in two dogs, No. 474 injected with 1:3 dilution alumina cream at L1-level, and No. 513 injected with 1:3 dilution alumina cream at T9-level.

Of 8 control dogs, none exhibited a rise in blood pressure by 13 mm.Hg or more (No. 514 No. 534, No. 502, No. 520, No. 536, No. 466, No. 504, No. 493).

(5) Renal Blood Flow
Renal functions were tested in 6 of 7 alumina-cream-injected dogs and 2 of 4 saline-solution-injected control dogs. The differences between the pre- and post-operative determinations, converted in per cent of the pre-operative value, are given in Table I (B). Inspection of the table indicates that there was decrease by 20 % or more in RBF in all the 3 dogs injected with alumina cream at 1:3 dilution and 1 of the 3 dogs injected with alumina cream at 1:10 dilution.

In No. 542 (injected with 1 ml. alumina cream at 1:10 dilution at T4-level) there was a decrease in blood flow by 23 % in the 2nd post-operative week. In No. 513 (injected with 1 ml. alumina cream at 1:3 dilution at T9-level) there was a decrease by 22 % in blood flow in the 5th post-operative week, but an increase by +45 % in the 7th post-operative week. Dog No. 477 (injected with 1 ml. alumina cream at 1:3 dilution at L1-level) was associated with decreases in blood flow by 31, 26 and 28 % at the 3rd, 4th and 8th post-operative week, respectively, while in the meantime there was a return to the pre-operative level at the 6th week. Dog No. 474 (injected with 1 ml. alumina cream at 1:3 dilution at L1-level) was associated with decreases in blood flow by 23 and 22 % at the 6th and 8th post-operative week, while there was a return to the pre-operative level at the 5th and 7th week. The overall picture of variation in the 4 dogs described above was characterized by alternating diminution and recovery in the blood flow at intervals of 1 to several weeks. Such characteristics were absent in the control two dogs (No. 536, No. 504).

(6) Renal Vascular Resistance
Renal vascular resistances were calculated from the observed data, dividing blood pressures by renal blood flow. The pre- and post-operative differences in the renal vascular resistance, converted in per cent of the pre-operative value, are given in Table I (C).

This table indicated that all the three dogs injected with 1:3 dilution alumina cream and one of those injected with 1:10 dilution alumina cream developed increases in the renal vascular resistance ranging between 33 and 55 per cent. A closer observation of No. 474 reveals that changes in blood pressure were associated with simultaneous changes in the renal blood flow in such way that increase in the blood pressure was associated with decrease in the renal blood flow and vice versa, the calculated renal vascular resistance also changing simultaneously with the two parameters with directional parallelism with the blood pressure. Dog No. 474 was associated with increase in the renal vascular resistance by 42 and 33 per cent at the 6th and 8th post-operative week, but decrease by 11 and 2 per cent at the 5th and 7th post-operative week; dog No. 477 was associated with increases by 55, 44 and 43 at the 3rd, 4th and 8th post-operative week, while variations were small in the 5th-6th-7th week period, vascular resistances being -2, +14 and +6 per cent, respectively; dog No. 513 was associated with increase by 36 per cent at the 5th post-operative week and dog No. 542 with increase by 54 % in the second week, but their resistances remained not significantly altered in the rest observation period. It may be said generally that there were alternating increase and recovery in the renal vascular resistance, as was the case with the renal blood flow, at intervals of one to several weeks.

Such findings were absent from the 2 control dogs observed (No. 536, No. 504).

(7) Variations in Glomerular Filtration Rate
The pre-and post-operative differences in glomerular filtration rate, expressed in per cent of the pre-operative value, are listed in Table I-(E), which indicates that there were no definite directional changes in GFR correlatable to specific injection-level or material, except for No. 477 which was associated with persistent decrease in GFR.

(8) Variations in Filtration Fraction

The pre- and post-operative differences in filtration fraction, expressed in per cent of the pre-operative value, are listed in Table I-(E). Increases over 20% were recorded in 2 of the 3 dogs injected with 1:3 dilution alumina cream. Dog No. 474 (injected with 1:3 dilution alumina cream at L1-level) was associated with increases by 37, 25 and 46 per cent in the 3rd, 4th and 6th post-operative week; dog No. 513 (injected with 1:3 dilution alumina cream at T9-level) was associated with increases by 20 and 41 per cent in the 4th and 5th post-operative week, while filtration fraction remained unchanged from the pre-operative value in the rest period. With 1:10 dilution alumina cream, all the 3 dogs failed to present any significant variation in the filtration fraction.

Control dogs did not exhibit any large variations in filtration fraction.

(9) Histological Pictures

Autopsy was performed on 2 dogs (No. 474, No. 541) at the end of 3 post-operative months, when the rest dogs remained healthy.

*Kidneys*—— Characteristic histological changes in the 2 dogs consisted of endothelial proliferation of vas afferens glomeruli.

*Spinal Cord*—— In the 2 dogs there were thickening of the dura and cellular proliferation in the piaarachnoid space, as shown in Fig. 2, where one can note the contents of the indwelling vinyl tube, which was removed at the time of the autopsy, nearly at the center of the area of cellular proliferation.

Appearance at the time of autopsy of the indwelling vinyl reservoir, riding over the spinal cord, is shown in Fig. 3.

Intramedullarily, there were neither cavitation nor enlargement of the central canal.

![Fig. 2](image1.png)  
No. 474. The spinal cord. There are thickening of the dura and cellular proliferation in the piaarachnoid space. One can note the contents of the indwelling vinyl tube, which was removed at the time of autopsy, nearly at the center of the area of cellular proliferation. Weigert vanGieson elastica staining.

![Fig. 3](image2.png)  
No. 474. Appearance at the time of autopsy of the indwelling vinyl reservoir (R), riding over the spinal cord (S).
DISCUSSION

To summarize the main features of the present experiment, 7 dogs were injected after laminectomy with alumina cream at 1:3 or 1:10 dilution in dose of 1.0 ml. at varying levels (T4, T9, L1, L4) with the aid of a vinyl tubing introduced intrathecally through a small incision applied to the dura mater and connected to an indwelling, alumina-cream-filled reservoir.

Following the operation, there was a rise in blood pressure by 15 to 27 mm.Hg in 4 of the 7 dogs. In 6 of the 7 dogs renal blood flow determinations were made before and after the operation. All of the 4 dogs made hypertensive following the operation exhibited decreases in renal blood flow by 22 to 26 per cent. Eight dogs, serving as controls, have been injected after laminectomy, not with alumina cream, but with sodium dicetyl phosphate, physiological saline solution in the same doses or no material at all, at varying levels (T9, L1, L4) similarly with the aid of indwelling vinyl reservoir and tubing. Following the operation, however, there was no rise in blood pressure in excess of 12 mm.Hg, and the post-operative diminution in renal blood flow was insignificant in the 2 dogs tested.

Back to the clinical and theoretical aspects of the problem of essential hypertension pertinent to this experiment, some salient points must be mentioned here briefly.

As regards clinical "facts" in essential hypertension, as were discussed in detail in the previous paper, suggestive evidences have been presented by Maekawa and his students indicating the participation of the spinal factor in the initiation and/or maintenance of the essential hypertension. They are: (a) high frequency of incidence of myelographically suggested subclinical adhesive arachnoiditis in the lower thoracic segments, (b) extensive involvement in degeneration of the lateral horn cells as revealed in 3 autopsied cases of essential or malignant hypertension, (c) reduced cold pressor responses from the sole in essential hypertension which was taken to indicate increased tonic sympathetic activity in the distal half of the spinal cord, and (d) preliminary observation with petechiodermmography suggesting the increased vasoconstrictive tone in the lower thoracic segments in occasional hypertensive patients.

Maekawa presented a theory on the possible pathogenesis of essential hypertension which both accounts for the observed clinical "facts" and explain the energetics of the generalized vasoconstriction. Details of this theory have been described in the introduction to this paper and in the previous paper, so will not be reiterated here. It suffices to note that the part of Maekawa's theory where he associated localized spinal adhesive arachnoiditis with the pathogenesis of hypertension has been considerably substantiated by the author's experiment reported previously. The present investigation, originally aimed to test the presence or absence of renal ischemia simultaneous with elevation in the blood pressure in such experimental arachnoiditis-induced hypertension, ended in nearly affirmative answer: there was definite diminution in the renal blood flow associated the rise in the blood pressure, in sharp contrast to the control experiment, in the author's alumina-cream-induced lower thoracic adhesive arachnoiditis.

Pressor Responses ——— Since this experiment was carried out in hot summer season of July and August and ended within a short observation period of 8 to 9 weeks, pressor effects of the cold can be eliminated from consideration. Alumina-cream-injected dogs developed elevation in blood pressure by 15 mm.Hg or more (4 of the 7 dogs), and this contrasted sharply with the observed absence of pressor responses in the control group.

The relative efficacy of concentrated and less concentrated alumina cream fluids in producing hypertension may be assessed from the observation that 2 of the 3 dogs injected with 1:3 dilution fluid developed hypertension of 150 mm.Hg or higher mean blood pressure, while only 1 of the 4 dogs injected with 1:10 dilution fluid developed hypertension of at best 124 mm.Hg. It appears that concentrated alumina cream is more efficient in the production of hypertension than is dilute one.

Whether or not a specific level of the spinal cord must be involved in the experimental arachnoiditis for the blood pressure to be elevated significantly can not be answered by the present experiment, since the dog injected at T4-level differed from other dogs in starting with unusually low blood pressure and does not give a war-
ranted answer as to whether or not the observed subsequent traverse, at +15 to +19 plateau, may be taken to indicate a significant elevation in the blood pressure.

The question of whether the essential requirement for the elevation in blood pressure is localized adhesive arachnoiditis per se or the neuropharmacological action of aluminium ion also cannot be answered at the time of the writing this paper, since the decision rests upon the histological examination of the sodium-dicitrylphosphate-injected normotensive dogs for actual arachnoiditic processes.

Decrease in Renal Blood Flow and Increase in Renal Vascular Resistance—— As to the relative efficacy of concentrated and less concentrated alumina cream fluid in the production of hypertension, the results appear to indicate that the concentrated material is more effective than is the less concentrated one.

As to the specificity of the spinal level to be involved in arachnoiditis for the renal blood flow to be significantly reduced, again this investigation does not afford any definitive answer.

The relative importance of the adhesive arachnoiditis per se and the local aluminium ion for the reduction in renal blood flow may be questioned, but no materials are available for the assessment of this problem.

Noteworthy in this experiment was the alternating decrease and recovery of the renal blood flow in hypertensive dogs and those injected with alumina cream at 1:3 dilution at intervals of 1 to several weeks.

While the synchronicity is not clear, except for one hypertensive dog, between the cyclic variations in blood pressure and those in renal blood flow, unidirectionality of the changes in renal blood flow (all for decrease) and that in renal vascular resistance (all for increase) is quite conspicuous, and equally marked is the degree to which increase in renal vascular resistance in hypertensive dogs is capable as compared with normotensive dogs.

Increase in Filtration Fraction—— Increase in filtration fraction occurred in 2 of the dogs injected with concentrated alumina cream fluid.

Changes in the Spinal Cord—— The experimental adhesive arachnoiditis produced was localized histologically to the region exposed to the operation. In the present experiment cavitation of the type described in the previous paper was entirely absent, which may be explained by the shortness of the observation period in the present series as compared with as long a period as one year employed in the previous experiment.

It may be that presence of the alumina-cream-induced pialarthritis over 3 months or more is required for the development of cavitation.

As was described early in this paper, Maekawa's theory on the probable mechanism in the pathogenesis of essential hypertension indicts the irritation of the spinal renal vasomotor center with charge of producing renal ischemia and its functional derangements. More particularly, he charges the subclinical adhesive arachnoiditis with the vasomotor center irritation. Considerable parts of this theory have been substantiated by the present experiment where those dogs bearing alumina-cream-induced arachnoiditis developed elevation in blood pressure by 15 mm.Hg or more, decrease over 20 per cent in the renal blood flow, and large increases in the renal vascular resistance, intermittently at intervals of one to several weeks. Detailed mechanisms whereby the renal ischemia causes or associated elevation in blood pressure and the question of synchronous or asynchronous cyclicity of these changes remain to be investigated.

Classical experimental neurogenic hypertension of moderator nerve removal type by Hering, finds no clinical counter-part in human essential hypertension. This holds also for the experimental neurogenic hypertension produced by electric stimulation of the frontal area or increase in intracranial pressure. The experimental neurogenic hypertension with renal mediation, reported by Kottke et al., depended on the continuation of the stimulation, since the elevation of pressure persisted only during the stimulation. Renal mediation of neurogenic hypertension in animals seems to be well established, because Heymans and Bouckaert found that while total sympathectomy abolishes the hypertension due to interruption of the moderator nerves, this form of hypertension persists if the sympathectomy is complete except for sparing the nerves to the kidneys. However, there have been scarcely any investigation testing the possibility of producing
neurogenic hypertension with chronic, nearly physiological irritation of the spinal cord at the level of the renal vesomotor center, until the present experiment, suggested ingeniously by Maekawa, was performed by the author. This type of nearly physiological experiment on the neurogenic hypertension with renal mediation appears to be much demanded for particularly at such critical moment as today when "Goldblatt experimental renal hypertension", as Smith puts it, "is present in a state of flux and throw no certain light on the origin of hypertensive vascular disease in man" [10].

The type of neurogenic hypertension reported in the previous and present paper appears to constitute reasonably good experimental counterpart of human essential hypertension in that lower thoracic subclinical adhesive arachnoiditis was associated with diastolic hypertension [2] and reduction in renal blood flow. Reproduction of essential features of human essential hypertension was not complete, however, in that the duration of the hypertension was short and there was poor persistence of the elevated blood pressure. These gaps call for further studies on pressor mechanisms operative in this neurogenic hypertension as well as on many aspects of the problems, methodological, hemodynamic and biochemical.

**Summary**

1. A total of 7 dogs were injected after laminectomy with small amounts of alumina cream fluid, 4 of them at the level of the 1st lumbar vertebra housing presumable renal vasomotor center, 3 of them at T4, T9 and L4 level. Reduction in the renal blood flow by 22 to 31 per cent occurred in all of the 3 dogs injected with concentrated material at one time or another during the period between the 3rd and 8th week (table I).

2. Two of the 3 dogs injected with concentrated material developed intermittent elevation in mean blood pressure (table I).

3. All the 3 dogs, 2 of them hypertensive, injected with concentrated material developed intermittent increases in calculated renal vascular resistance by 33 to 55 per cent of the original resistance value during the period of observation between the 3rd and 8th post-operative week.

4. Two dogs, one hypertensive and the other normotensive, were sacrificed at the end of 3 post-operative months and examined for histological alterations in the spinal cord (and kidneys). There was adhesive arachnoiditis localized to the spinal cord level exposed to the operation and endothelial proliferation of vas afferens glomeruli (fig. 2).

5. Eight dogs were used as the controls and injected, after laminectomy at similar levels, with sodium dicetyl phosphate, physiological saline solution or no material at all. Their mean blood pressure were followed for the period of 8 weeks and the renal plasma and blood flow, glomerular filtration rate were serially determined in 2 of them. There was no significant changes in this group in the blood pressure, renal blood flow and renal vascular resistance.

6. These results appear to substantiate, to a considerable extent, Maekawa's theory on the probable mechanisms in the pathogenesis of essential hypertension that the presence of subclinical adhesive arachnoiditis in the area of the spinal renal vasomotor center plays an important role in the production of renal ischemia and the initiation of hypertensive processes.

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