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Major Advisor

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Dean
STUDIES OF BLOOD LEUKOCYTES AND PERITONEAL FLUIDS
IN SOME ACUTE ABDOMINAL CONDITIONS AND IN SHOCK

BY

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A THESIS

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## Part II.

Table I. Traumatic Shock


Table II. Thermal Shock

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A. Traumatic Shock
B. Shock with Tourniquet.

Table IV. Thermal Shock: Blood Pressure and Cell Volume

## Part III.

Table I. Four Inch Strangulated Loop
Acute leukopenia produced by the injection of foreign protein substances was noted in 1886 by Wyssokowitsch, and was attributed at first to destruction of white cells (Lowit -1892) and later to withdrawal of leukocytes from the peripheral circulation into vessels of the viscera. In substance these are the two theories that prevail today.

There is much conflicting evidence on the mechanism of leukopenia. Some of it is favorable to either or the two theories mentioned, some to neither or to both. The leukopenia occurring with damage to bone marrow by a chemical or toxic agent (agranulocytosis) will not be considered here.

Incidental to investigations into the toxicity of the contents of strangulated jejunum, Stoner and Hill found that a profound leukopenia followed within five minutes after the intravenous injection of such material into dogs. They found the leukopenia preceded the clinical symptoms of such injections by as much as thirty minutes, but in some instances leukopenia was present with no clinical symptoms. Leukopenia also followed injection of the content of normal bowel, of bowel content from simple obstruction, and of the transudate collected in a bag around an obstructed and strangulated loop. In their hands Whipple
precipitates" of such contents proved less toxic and less depressing to white cells than contents not so treated.

Stoner and Hill believe that leukopenia produced in this manner is a manifestation of some type of shock, and that the symptoms produced (hyperpnea, prostration, tremor, vomiting) by injection of intestinal contents are probably signs of the same phenomenon. Jung in 1931 noted a parallelism between leukopenia and low blood pressure and postulated the theory that in peptone shock the fall in blood pressure may be the principle cause of the leukopenia. The consensus at present, however, seems to be that shock and hemoconcentration are accompanied by leukocytosis.

Instances in which splenic, liver or other tissue substances (Goldsheider and Jacob), peptones (Webb), nucleins (Doane), intestinal contents (Hill) are suddenly introduced into the blood by natural process must be rare, but the invasion of such substances into the peritoneal cavity might occur. The possibility that leukopenias similar to those produced by intravenous injections might result from intraperitoneal injections deserved investigation. Clinically, the occurrence of leukopenia concurrent with rupture of viscera has often been noted but seldom been appreciated. Such leukopenias in the past have only served to confuse
the observers and because of the firm belief that leukocytosis should occur in these cases, leukocyte counts have been questioned.

Such speculation stimulated investigation of the reaction of the white blood cells to injections of these substances intraperitoneally. Contents of normal and obstructed intestine were as the material to be injected partly because such contents were made accessible by contemporary experiments upon simple and strangulated obstructions in dogs, and partly because, in certain pathological conditions, such peritoneal soilage could happen. Accordingly two dogs were injected intraperitoneally with 15 to 20 cubic centimeters of intestinal contents of normal dogs. The injection of 15 cubic centimeters caused the white cell count to drop from 17,100 to 15,500 in five minutes, while in another dog, receiving 20 cubic centimeters, the white blood count dropped from 8,700 to 7,850 in five minutes, and to 6,800 in fifteen minutes. Neither dog appeared very ill, both recovered.

A slight drop in the number of leukocytes of this degree could easily be due to a local peritoneal hyperleukocytosis as suggested by Lawrence. However, Steinberg in his exhaustive study on peritoneal exudates found that leukocytosis could and did appear both in the peritoneal exudates and the peripheral blood stream. He found that following the intraperitoneal injection of formalin-killed Escherichia coli suspended in a saline-tragacanth-aleuronate solution, the peripheral white count rose on an average from
from 10,600 to 12,800 in one hour. In three hours the white count of the peritoneal exudate was 91,000, while the peripheral blood showed a count of 15,600. In 72 hours the white blood count of the exudate was 240,000 and the white blood count stood at 21,450. Corwin found a similar, though not as dramatic, picture upon injection of various other substances intraperitoneally.
EXPERIMENTAL WORK

Further study of leukopenia following intraperitoneal injections was divided into the following four groups.

GROUP I

In order to obtain the material injected into the peritoneal cavity of the four dogs studied in this group, it was necessary that four other dogs be sacrificed. Ether was administered by intratrachial intubation after induction in an ether box. No preliminary medication was given. Intestinal strangulation was produced by ligation of intestine, mesentery and vessels with one-half inch twilled cotton tape under aseptic conditions. The incision was closed in three layers (peritoneum, and muscle, subcutaneous tissue, skin) with No. 8 cotton thread. The loop was removed sixteen hours after strangulation to forstall rupture and loss of the contents. These contents, usually 100 cubic centimeters of bloody, foul smelling fluid, were removed from an intact strangulated gangrenous loop of jejunum about two feet in length.

After injection of intestinal contents all four dogs had a lowered leukocyte count within fifteen minutes. Immediately upon injection the dogs became very ill; trembled, whined, and vomited. When released one hour after injection they were weak,
nauseated, and dejected and all but one died in about eight hours. Five dogs were then injected with a coarsely filtered saline suspension of contents of normal bowel. In each case the content of the whole small intestine, which was milked out immediately after the death of an animal killed with ether, was mixed with saline and filtered through gauze. This material was then injected intraperitoneally. These dogs did not appear as ill as the former group and when released appeared normal. The leukocyte drop following injection in this group (Table I-B) was as definite at fifteen minutes as in the former group (Table I-A), but a significantly greater proportion recovered.
### TABLE I

**INTRAPERITONEAL INJECTION OF INTESTINAL CONTENTS**

#### A. Contents of Strangulated Loop

<table>
<thead>
<tr>
<th>Dog #</th>
<th>Normal Amt. injected</th>
<th>W.B.C. 5 min.</th>
<th>W.B.C. 15 min.</th>
<th>W.B.C. 30 min.</th>
<th>W.B.C. 1 hr.</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>581</td>
<td>12400 20 cc.</td>
<td>12700</td>
<td>11000</td>
<td>3600</td>
<td>7500</td>
<td>Recovered</td>
</tr>
<tr>
<td>584</td>
<td>18100 100 cc.</td>
<td>12600</td>
<td>14950</td>
<td>10750</td>
<td></td>
<td>Died in 15 hours</td>
</tr>
<tr>
<td>574</td>
<td>15150 50 cc.</td>
<td>14200</td>
<td>11650</td>
<td>7900</td>
<td>6500</td>
<td>Became very ill upon injection. Died during night.</td>
</tr>
<tr>
<td>576</td>
<td>16800 50 cc.</td>
<td>12800</td>
<td>9650</td>
<td>7800</td>
<td>5150</td>
<td></td>
</tr>
</tbody>
</table>

#### B. Contents of Normal Dog's Intestine

<table>
<thead>
<tr>
<th>Dog #</th>
<th>Normal Amt. injected</th>
<th>W.B.C. 5 min.</th>
<th>W.B.C. 15 min.</th>
<th>W.B.C. 30 min.</th>
<th>W.B.C. 1 hr.</th>
<th>W.B.C. 1½ hr.</th>
<th>W.B.C. 24 hr.</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>575</td>
<td>17100 15 cc.</td>
<td>15400</td>
<td>16000</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Recovered</td>
</tr>
<tr>
<td>576</td>
<td>8700 20 cc.</td>
<td>7850</td>
<td>6800</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Recovered</td>
</tr>
<tr>
<td>577</td>
<td>31500 20 cc.</td>
<td>26500</td>
<td>18500</td>
<td>14600</td>
<td></td>
<td></td>
<td></td>
<td>26050 Recovered</td>
</tr>
<tr>
<td>597</td>
<td>7750 50 cc.</td>
<td>5350</td>
<td>6150</td>
<td>6650</td>
<td>3250</td>
<td>1300</td>
<td></td>
<td>Died</td>
</tr>
<tr>
<td>598</td>
<td>8650 50 cc.</td>
<td>6650</td>
<td>4550</td>
<td>2950</td>
<td>1600</td>
<td>1000</td>
<td></td>
<td>Died</td>
</tr>
</tbody>
</table>

---
Table I - Comment and conclusions.

When the contents of strangulated bowel are injected intraperitoneally into another animal there is a marked systemic reaction which usually results in death. About thirty minutes after the injection there is a pronounced drop in the leukocyte count in the peripheral blood.

When the contents of normal intestine are injected in the same manner, the systemic reaction is less severe and the animal usually recovers. The fall in leukocytes is not as constant or as pronounced but usually occurs.
GROUP II

In order to determine if leukopenia might occur following rupture of a viscus, we procured gastric contents by lavage of a dog's stomach one-half hour after eating white bread, (Dogs No. 605 and 594) and, by washing a stomach (removed from dog under ether anesthetic for one hour) with saline. (Dog No. 606).

White bread was used as a stimulating meal to avoid the side effects which would probably follow intraperitoneal injection of the meat extract test meal usually used. However, because white bread is such a poor secretagogue, it was decided that histamine diphosphate (2.08 mg. intramuscularly) should be used in the remainder of the experiments in Group II. Gastric contents were removed one-half hour after injection of histamine and injected into dogs 715, 717, 716, 718, and 719 after treatment as indicated in "Remarks". Results of such injections can be seen in Table II. All dogs in this group lived with the exception noted in Table II.
TABLE II

INTRAPERITONEAL INJECTION OF GASTRIC CONTENTS

<table>
<thead>
<tr>
<th>Dogs</th>
<th>W.B.C.</th>
<th>Normal</th>
<th>8450</th>
</tr>
</thead>
<tbody>
<tr>
<td>#</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>605</td>
<td>15000</td>
<td>13000</td>
<td>11050</td>
</tr>
<tr>
<td>606</td>
<td>13000</td>
<td>11050</td>
<td>10800</td>
</tr>
<tr>
<td>594</td>
<td>11050</td>
<td>10800</td>
<td>10600</td>
</tr>
<tr>
<td>715</td>
<td>10800</td>
<td>10600</td>
<td>10400</td>
</tr>
<tr>
<td>716</td>
<td>10600</td>
<td>10400</td>
<td>10200</td>
</tr>
<tr>
<td>718</td>
<td>10400</td>
<td>10200</td>
<td>10000</td>
</tr>
<tr>
<td>717</td>
<td>10200</td>
<td>10000</td>
<td>9800</td>
</tr>
<tr>
<td>719</td>
<td>10000</td>
<td>9800</td>
<td>9600</td>
</tr>
</tbody>
</table>

GASTRIC CONTENTS INJECTED

<p>| | | | | | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>15 min.</td>
<td>8150</td>
<td>13100</td>
<td>11150</td>
<td>10300</td>
<td>10300</td>
<td>14300</td>
<td>3150</td>
<td>10700</td>
<td></td>
</tr>
<tr>
<td>30 min.</td>
<td>6500</td>
<td>8600</td>
<td>11350</td>
<td>11900</td>
<td>11450</td>
<td>11800</td>
<td>3000</td>
<td>11300</td>
<td></td>
</tr>
<tr>
<td>60 min.</td>
<td>6000</td>
<td>12700</td>
<td>5700</td>
<td>8250</td>
<td>13950</td>
<td>12500</td>
<td>3100</td>
<td>10600</td>
<td></td>
</tr>
<tr>
<td>1½ hrs.</td>
<td>6700</td>
<td>12700</td>
<td>3850</td>
<td>6850</td>
<td>11150</td>
<td>14700</td>
<td>3900</td>
<td>4950</td>
<td></td>
</tr>
<tr>
<td>2 hrs.</td>
<td>6550</td>
<td>11650</td>
<td>5750</td>
<td>7800</td>
<td>10950</td>
<td>11950</td>
<td>2700</td>
<td>8400</td>
<td></td>
</tr>
<tr>
<td>2½ hrs.</td>
<td>6550</td>
<td>11400</td>
<td>2450</td>
<td>6800</td>
<td>11200</td>
<td>16500</td>
<td>4100</td>
<td>6400</td>
<td></td>
</tr>
<tr>
<td>3 hrs.</td>
<td>7850</td>
<td>7400</td>
<td>4400</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 hrs.</td>
<td>7850</td>
<td>11700</td>
<td>7400</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 hrs.</td>
<td>7850</td>
<td>14750</td>
<td>7400</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>21 hrs.</td>
<td>20150</td>
<td>21550</td>
<td>29200</td>
<td>45300</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

died in 49 hrs.
REMARKS:

#605. Given 20 cubic centimeters unfiltered gastric contents
removed from healthy dogs after meal of white bread.

#606. Given 20 cubic centimeters filtered saline suspension
of contents of stomach removed from dog under ether
anesthesia.

#715. Given 50 cubic centimeters of unfiltered gastric con-
tents removed one-half hour after histamine stimulation.
Stomach was here lavaged with 300 cubic centimeters
of distilled water before injection of histamine and
removal of contents.

#716. Given 25 cubic centimeters of filtered gastric content.
Stomach was not lavaged. Otherwise same as #715 and #716.

#717. Given 50 cubic centimeters of unfiltered contents from
unlavaged stomach removed one-half hour after histamine
stimulation. This dog, a female Scottie, in estrus,
died 49 hours after the injection.

Table II- Comment and conclusions.

When gastric contents are injected intraperitoneally a drop
in leukocyte count occurs in over half the animals. This drop
is not constant in degree or time of occurrence. The systemic
reactions in this group were not as severe as in Group I. All
animals in this group lived.
The appendices of five dogs were ligated in such a way that venous return was stopped. This operation was done aseptically with intratracheal ether anesthesia after induction in an ether box, and with no previous medication. A cutting suture was fixed to the appendix in the distal one-third. At six hours the appendix was swollen to three times its former size and was black and gangrenous. At this time these dogs had a leukocytosis. Rupture of the appendix (produced by pulling the cutting suture) resulted in a drop in white blood count in all but one, and an increase in signs of illness. In all cases the dogs died, and in all cases the first drop in white blood count was followed by a rise in two to three hours. This rise was transitory, however, and soon a severe leukopenia occurred, followed by death. In the one exception (dog #604) noted in Table III, perforation at surgery occurred.
<table>
<thead>
<tr>
<th>Dog #</th>
<th>601</th>
<th>602</th>
<th>604</th>
<th>607</th>
<th>608</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>9450</td>
<td>9100</td>
<td></td>
<td></td>
<td>13500</td>
</tr>
<tr>
<td>Appendix freed from mesentery, ligated and cutting suture affixed</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 hr. P.O.</td>
<td>18700</td>
<td>15650</td>
<td>18400</td>
<td>19000</td>
<td>29200</td>
</tr>
<tr>
<td>Cutting suture removed ---, appendix ruptured</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 min.</td>
<td>16350</td>
<td>16250</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15 min.</td>
<td>16100</td>
<td>14250</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30 min.</td>
<td>15150</td>
<td>21200</td>
<td>10550</td>
<td>29050</td>
<td></td>
</tr>
<tr>
<td>60 min.</td>
<td>14800</td>
<td>13350</td>
<td>18350</td>
<td>6950</td>
<td>26000</td>
</tr>
<tr>
<td>1½ hrs.</td>
<td>8350</td>
<td>15950</td>
<td>1850</td>
<td>26050</td>
<td></td>
</tr>
<tr>
<td>2 hrs.</td>
<td>6600</td>
<td>19400</td>
<td>900</td>
<td>18000</td>
<td></td>
</tr>
<tr>
<td>2½ hrs.</td>
<td>7500</td>
<td>16400</td>
<td>1900</td>
<td>17500</td>
<td></td>
</tr>
<tr>
<td>3 hrs.</td>
<td>4650</td>
<td>21400</td>
<td>1950</td>
<td>18300</td>
<td></td>
</tr>
<tr>
<td>3½ hrs.</td>
<td>3950</td>
<td>18900</td>
<td>3250</td>
<td>17500</td>
<td></td>
</tr>
<tr>
<td>4 hrs.</td>
<td>4000</td>
<td>17500</td>
<td>3650</td>
<td>16650</td>
<td></td>
</tr>
<tr>
<td>4½ hrs.</td>
<td>1400</td>
<td></td>
<td>2200</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 hrs.</td>
<td>2050</td>
<td>20560</td>
<td>Died</td>
<td>19150</td>
<td></td>
</tr>
<tr>
<td>5½ hrs.</td>
<td></td>
<td></td>
<td></td>
<td>24650</td>
<td></td>
</tr>
<tr>
<td>6 hrs.</td>
<td></td>
<td>17150</td>
<td></td>
<td>23650</td>
<td></td>
</tr>
<tr>
<td>6½ hrs.</td>
<td></td>
<td>21850</td>
<td></td>
<td>22400</td>
<td></td>
</tr>
<tr>
<td>16700</td>
<td>Died</td>
<td>2250</td>
<td></td>
<td>14600</td>
<td></td>
</tr>
</tbody>
</table>

at 22 hrs. 20 hrs. at 16 hrs. at 24 hrs.
Table III - Comment and Conclusions.

When a gangrenous appendix is made to rupture by the use of a cutting suture the leukocyte count falls. It is only rarely that it returns or goes higher than it was before rupture. The drop in the leukocyte count is not constant in degree or time of occurrence but occasionally a marked leukopenia develops.

It might be expected that the blood picture following rupture of a gangrenous strangulated appendix would be similar to that produced by injection of contents of a strangulated jejunal loop. However, symptoms appeared more rapidly in the latter, the leukopenia appeared more quickly and the length of life was much shorter.

Dogs in the first (Table I-A) and the last (Table III) of these experiments seemed prostrate. They shivered, vomited, and their became cold. They were acutely ill.
GROUP IV

In those experiments involving intraperitoneal injection of various suspensions, normal saline taken from stock was used as the vehicle. The possibility that normal saline so injected could cause a white cell response does exist. In order to evaluate the influence of this factor on leukocyte counts, two control experiments were done.

Fasting dogs were placed on an animal table as in Groups I, II, and III, and 25 cubic centimeters of autoclaved stock normal saline was injected intraperitoneally. These dogs showed some distress, but none of them vomited or gave symptoms similar to those in the other groups. Leukocyte counts were made and are tabulated in Table IV-A.

A second control experiment attempting to ascertain the effect, if any, of restraint and blood counts on the dogs gave results as illustrated in Table IV-B. These dogs were ones which had never before been used experimentally.

These results probably lie within the margin allowed for errors in counting. However, the trend toward a lower white count upon saline injection should be considered. The amount of fluid used could not reasonably account for the lowered count by simple dilution of blood elements by rapid absorption from the peritoneum. Neither should the minimal peritoneal irritation upon injection of normal saline cause a local collection of leukocytes.
TABLE IV

CONTROL EXPERIMENTS

A. Injection of Normal Stock Saline Intraperitoneally

<table>
<thead>
<tr>
<th>Dog #</th>
<th>Normal W.B.C.</th>
<th>1/2 hr.</th>
<th>1 hr.</th>
<th>1 1/2 hr.</th>
<th>2 hr.</th>
<th>2 1/2 hr.</th>
<th>Nervous State</th>
</tr>
</thead>
<tbody>
<tr>
<td>708</td>
<td>6250</td>
<td>5700</td>
<td>7550</td>
<td>4700</td>
<td>5700</td>
<td>6250</td>
<td>Quiet</td>
</tr>
<tr>
<td>709</td>
<td>8500</td>
<td>10100</td>
<td>7550</td>
<td>8350</td>
<td>7950</td>
<td>8750</td>
<td>Apprehensive</td>
</tr>
<tr>
<td>710</td>
<td>11800</td>
<td>12500</td>
<td>11300</td>
<td>13400</td>
<td>12000</td>
<td>14500</td>
<td>Very excited</td>
</tr>
</tbody>
</table>

B. Counts with Restraint Alone

<table>
<thead>
<tr>
<th>Dog #</th>
<th>Normal W.B.C.</th>
<th>1/2 hr.</th>
<th>1 hr.</th>
<th>1 1/2 hr.</th>
<th>2 hr.</th>
<th>2 1/2 hr.</th>
<th>Nervous State</th>
</tr>
</thead>
<tbody>
<tr>
<td>712</td>
<td>5950</td>
<td>4800</td>
<td>6250</td>
<td>5450</td>
<td>4550</td>
<td>6550</td>
<td>Apprehensive</td>
</tr>
<tr>
<td>800</td>
<td>8750</td>
<td>9100</td>
<td>8000</td>
<td>8250</td>
<td>8150</td>
<td>8850</td>
<td></td>
</tr>
</tbody>
</table>
Other physiologic variations were kept constant by using healthy dogs of approximately the same weight, and by starting the experiments at approximately the same time each day. Technical variations were kept constant by using the same counting techniques, chambers, pipettes, and technicians in all experiments. Several of the series of counts were counted in duplicate and the average recorded. In these the discrepancies between counters was seldom over 500 cells per cubic millimeter and averaged 300 cells per cubic millimeter.

Table IV - Comment and conclusions:

The injection of normal saline intraperitoneally and the excitement produced in the animal by restraint do not effect the leukocyte count.
DISCUSSION

Those who first had occasion to inject various animal substances and to observe the white blood cells noted a leukocytosis. However, in 1894, Goldschieter and Jacobs upon intravenous injection of suspensions of spleen, thymus, and marrow found a drop in white count, which they attributed to a chemotactic storage of white blood cells in lungs, spleen and liver. Endothelial swelling, stimulated by the autonomic nervous system, causing stagnation of these blood elements cannot entirely explain this drop in white blood count, although autonomic stimuli can be demonstrated to be at least coincidental with the onset of the leukopenia. Mueller has shown that intracutaneous injections of peptones as well as other stimuli to skin, cause peripheral leukopenia and visceral leukocytosis. Section of the autonomic nerves to the part stopped this "autonomic reflex leukopenia". However, Ewing observed similar endothelial swelling in normal livers from animals with no leukopenia. Cowie and Calhoun after giving typhoid vaccine intravenously to a series of ten humans, concluded that the leukopenia is a characteristic part of the "foreign protein reaction", and that the leukocytes accumulate in the visceral circulation. Lawrence has observed a redistribution of white cells within the vessels following intravenous injection of hydrophilic colloids (typhoid vaccine, gelatin, globulin and fibrinogen). In these cases the neutrophiles assume a marginal position in
the venules of liver, lungs, spleen and omentum during the peripheral neutropenia.

Many experiments have been done on this problem with rabbits as the subjects. Wells found that in normal rabbits the white blood count in lung and liver approximated closely the peripheral count, while in blood from splenic parenchyma it was several times greater. In less than 10 minutes after intravenous injection of killed bacteria (streptococcus, staphylococcus, typhoid bacillus) there occurred a marked drop in white blood cells in the peripheral blood and a great increase in white blood cells in visceral blood. Leukocytosis followed in four to fifteen hours. He believes that a positive chemotaxis acts on the polymorphnuclear leukocytes attracting them to spleen, liver, and other organs in which injected organisms have been filtered from the blood. Indeed these bacteria have been shown to lodge in the liver and spleen in about 10 minutes or at the onset of Wells'leukopenia. Other workers (Doane, Zwecker, Hill) have offered contradictory evidence to this idea of refugee leukocytes seeking sanctuary in the visceral organs, while still others have supported the concept.

Doane, Zerfas, Warren, and Ames excepted all but the spleen as storage points for white blood cells during leukopenia. They also pointed out the fallacy in counts made near, at, or after death, and showed convincingly that these were worthless when compared with a leukopenia in an otherwise healthy animal. They
showed that a drop in white cell count commensurate with the peripheral drop exists in liver and lung on intravenous injection of sodium nucleinate (one gram), and that there is a tremendous rise in polymorphonuclear neutrophils in the spleen. Following splenectomy they showed no drop in white count but a more rapid and lasting leukocytosis. They believe, therefore, that the spleen is a reservoir of the white cells during leukopenia. To prevent circulatory collapse they administered Caffiene Sodium Benzoate.

The use of sodium nucleinate was a logical development from Lowit's theory that lysis of leukocytes must occur before leukocytosis and from the fact that the greatest portion of the destroyed leukocytes was to be nuclein. The leukopenia following the intravenous injection of peptone they considered to be different in nature from that following the injection of sodium nucleinate or the nucleotides, adenine and guanine. Intravenous nucleotides gave responses similar to that following splenectomy, i.e., a rapid and prolonged leukocytosis.

However, Zwecker found no such phenomenon using one-tenth gram sodium nucleinate or B. coli vaccine. Using one gram he induced a shock-like state in rabbits. This difference in technique he concluded to have been the reason Doane, et al found their results. Further, the use of Caffiene Sodium Benzoate to combat the shock-like state would upset the vascular system, thereby adding more confusing factors. Zwecker
found a leukopenia in splenectomized animals comparable to that in normal animals. There was also a fall in blood pressure of fifty to seventy millimeters of mercury.

The experiments of Wells, Zwecker, and Doane, et al on rabbits cannot be applied without reservation to other animals. Other factors, emotional, physiological, individual, and chronological must be considered. Emotional leukopenia (31% drop) in rabbits has been shown by Nice and Katz to occur in both normal and splenectomized rabbits, while cats regularly showed leukocytosis. Mora, Amtman and Hoffman found an emotional leukocytosis in both cats and dogs. Hourly and diurnal, as well as postprandial variations must be considered. The narcotising action of anesthetics upon the leukocytes has been shown in vitro but concentrations used in animals experiments would not effect the leukocytes as such.

Mueller's experiments showing a neurogenic migration of leukocytes seem quite convincing. However, when Hill repeated the experiments with a slightly different technique he found little evidence supporting Mueller's work. The usual neutropenia following intravenous injection of peptone (ten cubic centimeters of twenty per cent peptone solution) Mueller attributed to accumulation of the leukocytes in vessels of the liver. Blocking these nerves with atropine forstalls the leukopenia following peptone injection. The work of Cooper
and others support these interpretations in part. Mueller and Myers were also able to block the leukopenia following intravenous albumen or alkaline salvarsan with atropine. Here white blood counts before and after death were again compared, thus vitiating in our opinion the value of the experiment.

Dogs and cats with denervated livers were found by Hill to have a fall in white count upon intravenous injection of peptone solution similar to those in dogs with intact nervous systems. Exercise and other methods of producing vascular changes causing variations in white blood count were found to be less effective after sympathectomy by Garrey and Butler. Vascular tone soon returns and then usual variations are again seen. Leriche and Fontaine observed a leukopenia and subsequent leukocytosis following periarterial sympathectomy, as did Shaw upon causing vasodilation with heat and friction. However, in these cases the differential counts were not altered, nor were there any changes in the red cell counts. No doubt there are changes in the leukocytes dependent upon nervous or vasotonic conditions. Perhaps they play a large part in complicating the general picture of leukopenia, but they cannot serve as complete answers to the questions posed by leukopenia.
CONCLUSIONS

1. Leukopenia similar to that produced by intravenous injection can be produced by intraperitoneal injection of intestinal contents from normal and obstructed loops of intestine.

2. Less severe leukopenia follows the intraperitoneal injection of gastric contents.

3. Severe rapid leukopenia is produced by rupture of a gangrenous appendix by means of a cutting suture.

4. The mechanism of leukopenia is not well understood. Perusal of the literature on leukopenia reveals only that a multiplicity of theories exist, none of which have been firmly established.

5. Low leukocyte counts in patients with ruptured appendix or perforated gastric ulcer are occasionally seen. Indeed, from the experiments here described they are often to be expected.
Although the following paper is a natural sequel to the preceding one its development was due largely to the friendly encouragement of Dr. C.J. Wilhelmj, who is greatly interested in the physiology of shock. The observations upon shock caused by burns are published with the permission of Dr. Nicholas Dietz who was at the same time investigating the physiology of that type of shock.
PART II

THE WHITE BLOOD COUNT IN TRAUMATIC
AND IN THERMAL SHOCK
INTRODUCTION

In an earlier communication it was shown that a decrease occurred in the white blood count following various intra-peritoneal injections and/or rupture of the appendix in dogs, similar to that found following intravenous injections of the contents of strangulated intestinal loops. These dogs showed many signs of shock. Huer and Andrus found a "toxic shock" manifested by low blood pressure, hemococoncentration, and slowing of the circulation following injection of the contents of closed intestinal loops. Cannon mentions shock produced by intravenous injections of muscle tissue suspensions as evidence for the existence of a toxic factor in shock. The arguments for and against the toxic theory of shock will not be discussed here. Moon observed the characteristic signs of shock after intraperitoneal injection of a minced muscle suspension, after intravenous injection of killed bacterial cultures, and with the onset of experimental peritonitis. These authors strengthened the impression that the leukopenia that had been studied by us was but another manifestation of shock.
GROUP I

TRAUMATIC SHOCK

In this group of seven dogs shock was induced by trauma to both hind limbs or to both fore limbs under ether anesthesia. The amount of trauma was not the same in all experiments. The muscular parts of the extremities were pounded with a hammer for approximately ten minutes. Neither skin nor bones were broken. Blood pressure was determined by means of a cannula in the carotid artery connected to a mercury manometer. Three cubic centimeters of blood were taken from the jugular vein every half hour for hematocrit determination and white blood counts. The removal of this amount of blood alone would not produce shock although it may have materially hastened its onset in these cases. Ether anesthesia may have contributed to the rapid onset of shock too, although Schweitzer does not consider it of great significance.

Four of the dogs in this group (Table I-A) showed a marked fall in white blood cells. Three showed slight or variable drops. In some of these experiments the drop in white blood count preceded the drop in blood pressure to the "critical level" by as much as two hours. Those dogs that showed a slight drop and variable white blood count attained the "critical level" of blood pressure earlier in the experiment.
than the balance of the animals in this group. It seemed that the critical level was usually reached by a precipitous drop in blood pressure after removal of the blood sample for counting and hematocrit determinations.

It is known that a sharp drop in blood pressure follows release of a tourniquet on a traumatized limb or on an untraumatized limb that has been constricted in a tourniquet for several hours. To follow the effect, if any, of this rapid change in blood pressure upon the total leukocyte count, three more experiments were done. (Table I-B) Tourniquets were tied about both hind extremities of two dogs after they had been etherized and cannulated. They were then traumatized about five minutes. Upon releasing the tourniquets the blood pressure dropped 20 mm. of mercury in one dog, 10 mm. of mercury in the other; the former dog also showed an appreciable drop in white count. The third dog, No. 706, which had received no trauma, had a leukocytosis until release of the tourniquet precipitated a drop in blood pressure and a temporary drop in white count. Peculiarly, this dog, 706, was the only one in this whole group that had hemoconcentration. It was not traumatized. Dog No. 714 had distemper.

The relationship between blood pressure and white blood count was noted earlier by Jung, who believed that a fall
in blood pressure could cause leukopenia. The experiments described above confirm this relationship but indicate that they are more a parallel than a causal relation because in most instances the leukopenia is detected before change in blood pressure or red blood cell concentration can be found. Schweitzer likewise held that the leukopenia was caused by low blood pressure during and following surgical procedures. He found the fall to be general, and not a redistribution of white cells, although he found some differences between counts on peripheral and visceral blood samples.

Allergic shock (probably a very different mechanism) is also characterized by a leukopenia. Waldbott, Ascher, and Rosenzweig found a significant drop in white count within fifteen minutes after the injection of pollen. In these cases the white count returned to normal in about two hours.

Harlow and Selye found a leukopenia following severe "alarm-stimuli". Their alarming stimuli were "acute, non-specific, damaging influences such as cold, surgical injuries, and intoxication with sub lethal doses of drugs." Their findings were similar to those in Tables I and II in that the white count returned to normal or above normal in those dogs that lived while continued leukopenia indicated impending death.
### Table I.


<table>
<thead>
<tr>
<th>Dog #</th>
<th>W.B.C.</th>
<th>Etherization</th>
<th>0.5 hr.</th>
<th>1 hr.</th>
<th>1.5 hr.</th>
<th>2 hr.</th>
<th>2.5 hr.</th>
<th>3 hr.</th>
<th>4 hr.</th>
<th>5 hr.</th>
<th>6 hr.</th>
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<tr>
<td>580</td>
<td>7800</td>
<td>10100</td>
<td>7700</td>
<td>7650</td>
<td>7600</td>
<td>2250</td>
<td>1150</td>
<td>heart sample at death: 6900</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>605</td>
<td>11400</td>
<td>15040</td>
<td>8900</td>
<td>8350</td>
<td>4800</td>
<td>6650</td>
<td>8250</td>
<td>heart sample at death: 7700</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>701</td>
<td>11300</td>
<td>10750</td>
<td>8150</td>
<td>6550</td>
<td>5250</td>
<td>5550</td>
<td>4650</td>
<td>6850</td>
<td>5500</td>
<td>4550</td>
<td>5680</td>
</tr>
<tr>
<td>705</td>
<td>16200</td>
<td>17450</td>
<td>12300</td>
<td>6500</td>
<td>8175</td>
<td>died</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>713</td>
<td>31700</td>
<td>29200</td>
<td>33500</td>
<td>29400</td>
<td>31300</td>
<td>32500</td>
<td>23500</td>
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<td>24350</td>
<td>20450</td>
<td></td>
</tr>
<tr>
<td>703</td>
<td>11100</td>
<td>12000</td>
<td>13750</td>
<td>14150</td>
<td>12450</td>
<td>14150</td>
<td>18050</td>
<td>15650</td>
<td>death sample: 25700</td>
<td></td>
<td></td>
</tr>
<tr>
<td>704</td>
<td>31150</td>
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<td>44400</td>
<td>43400</td>
<td>30450</td>
<td>20200</td>
<td>48750</td>
<td>43950</td>
<td>37250</td>
<td>36300</td>
<td></td>
</tr>
</tbody>
</table>

#### B. White Blood Count following release of tourniquet.

<table>
<thead>
<tr>
<th>Dog #</th>
<th>W.B.C.</th>
<th>Etherization</th>
<th>No.Hrs.</th>
<th>Trauma Before removal of Tourniquet</th>
<th>W.B.C. 1 hr.</th>
<th>2 hr.</th>
<th>3 hr.</th>
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</thead>
<tbody>
<tr>
<td>706</td>
<td>14700</td>
<td>17700</td>
<td>5 hrs.</td>
<td>None</td>
<td>14750</td>
<td>20100</td>
<td>14450</td>
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<tr>
<td>707</td>
<td>12200</td>
<td>11300</td>
<td>2½ hrs.</td>
<td>5 min.</td>
<td>8900</td>
<td>5000</td>
<td>6500</td>
</tr>
<tr>
<td>714</td>
<td>12700</td>
<td>12700</td>
<td>4½ hrs.</td>
<td>5 min.</td>
<td>13800</td>
<td>12600</td>
<td>18450</td>
</tr>
</tbody>
</table>
GROUP II

THERMAL SHOCK

This group of six dogs showed a marked drop in white count shortly after being burned. In all cases the count began to rise after about three hours only to fall again if the dog was to die. The animals that recovered had counts that continued to rise over an eight hour period. In this group the first white count after burning was uniformly lower than the first count after trauma or after the onset of declining blood pressure and white count in Group I. More dogs survived in this group than in Group I, but any doubts regarding the presence of shock were dispelled by evidence from blood pressure and red cell volume determinations.

The procedure used in Group II is as follows: The evening before the experiment the dog was shaved and weighed. Early the next morning, the normal blood sample was taken and the dog was given 0.25 mg. sodium amytal per kilogram of body weight intravenously. In twenty minutes it was quite deeply anesthetized. The lower extremities and trunk of the animal were then immersed in vigorously boiling water. The surface of the animal exposed and the time of exposure to the boiling water was approximately the same in all instances. The carotid artery was then cannulated and connected to a mercury manometer, recording blood
pressures upon a smoked drum. The blood pressures taken at this stage are given in Table III-B, (Group II) as "normal".

In one-half hour the smoked drum was marked and 3 cc. of blood was withdrawn from the jugular vein for a white blood count and hematocrit determination. Blood samples were taken every one and one-half hours until the fourth hour, then at longer intervals.
### GROUP II

#### TABLE II

**WHITE BLOOD COUNT IN THERMAL SHOCK**

<table>
<thead>
<tr>
<th>Dog #</th>
<th>Normal W.B.C.</th>
<th>W.B.C. Anesthetized-burned</th>
<th>1½ hr.</th>
<th>3 hr.</th>
<th>4½ hr.</th>
<th>6 hr.</th>
<th>7½ hr.</th>
<th>9 hr.</th>
<th>Remarks</th>
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<tr>
<td>739</td>
<td>25200</td>
<td></td>
<td>3100</td>
<td>5350</td>
<td>7850</td>
<td>14650</td>
<td>19250</td>
<td>12850</td>
<td>10350-Died</td>
</tr>
<tr>
<td>743</td>
<td>10700</td>
<td></td>
<td>1750</td>
<td>5600</td>
<td>10500</td>
<td>8250</td>
<td>8200</td>
<td></td>
<td>died-death sample - 6000</td>
</tr>
<tr>
<td>722</td>
<td>34400 histaminase intravenousy</td>
<td></td>
<td>6100</td>
<td>8200</td>
<td>17900</td>
<td>40000</td>
<td></td>
<td>38500</td>
<td>Recovered</td>
</tr>
<tr>
<td>730</td>
<td>27400</td>
<td></td>
<td>4550</td>
<td>10550</td>
<td>13500</td>
<td>24500</td>
<td>38300</td>
<td></td>
<td></td>
</tr>
<tr>
<td>744</td>
<td>24700</td>
<td></td>
<td>38100</td>
<td>39600</td>
<td>40000</td>
<td>52500</td>
<td>40600</td>
<td>54300</td>
<td>Carotid Art. not cannulated.</td>
</tr>
<tr>
<td>745</td>
<td>12800</td>
<td></td>
<td>8700</td>
<td>6500</td>
<td>12800</td>
<td>18600</td>
<td>25500</td>
<td>31900</td>
<td></td>
</tr>
</tbody>
</table>

**Note:** Torantil brand of histaminase (Winthrop T.360N) was given intramuscularly the night before the experiment and intravenously (100 M) after the burning to Dogs 722, 730 and 744. This was given in connection with another problem that has little if any bearing upon this experiment.
DISCUSSION

Were the conditions studied here true shock? In those animals subjected to trauma, the standard method of Blalock was used with only one modification (use of ether instead of barbital anesthesia). The burned animals were anesthetized with sodium amytal with essentially the same results as were found under ether anesthetic. The animals shortly showed an accelerated pulse, hyperpnea, and often slightly increased blood pressure, later followed by a sharp drop in blood pressure. All these signs are typical and there is general agreement upon their essentiality to the shock picture. Many authors, and particularly Virgil Moon, hold that hemococoncentration is a necessary part of shock. Bordeen (quoted by Cannon) and Cannon believe that a concentration of white cells accompanied the concentration of red cells.

These last attributes of shock were not found uniformly in these experiments. Indeed, red cell concentration was not found in any of our cases of traumatic shock (Group I, Table III), and leukocytosis was found in none of the experiments here listed. Concentration of red cells (Group II, Table IV) was found in all cases of burn shock and in that produced by a long application of tourniquet without trauma. Swingle, Parker, Taylor and Hays
concluded that hemoconcentration was not a constant finding in
shock after a large series of experiments upon dogs in traumatic
17
shock. Since specific gravity of whole blood, although a more
sensitive sign than hemoconcentration, is but a manifestation
of hemoconcentration, the only reliable indication of the presence
of shock is a blood pressure below the "critical level."
GROUP I

TABLE III

A. Traumatic Shock: Blood Pressure and Cell Volume

<table>
<thead>
<tr>
<th>Dog #</th>
<th>Normal B.P.</th>
<th>C.V.</th>
<th>1/2 hr.</th>
<th>1 hr.</th>
<th>1 1/2 hr.</th>
<th>2 hr.</th>
<th>2 1/2 hr.</th>
<th>3 hr.</th>
<th>4 hr.</th>
<th>5 hr.</th>
<th>6 hr.</th>
</tr>
</thead>
<tbody>
<tr>
<td>605</td>
<td>102 40</td>
<td></td>
<td>116 43</td>
<td>108 43</td>
<td>80 42</td>
<td>64 43</td>
<td>Died</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>701</td>
<td>104 42</td>
<td></td>
<td>88 44</td>
<td>86 14</td>
<td>96 45</td>
<td>96 43</td>
<td>66 44</td>
<td>60 43</td>
<td>44 42</td>
<td>41 32</td>
<td>40 22</td>
</tr>
<tr>
<td>703</td>
<td>98 32</td>
<td></td>
<td>80 41</td>
<td>70 41</td>
<td>72 42</td>
<td>28 42</td>
<td>24 41</td>
<td>18 40</td>
<td>12 44</td>
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<tr>
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<td>92 38</td>
<td>60 38</td>
<td>60 39</td>
<td>36 39</td>
<td>60 39</td>
<td>52 38</td>
<td>38 32</td>
<td>38 20</td>
<td>37 8</td>
</tr>
<tr>
<td>705</td>
<td>90 41</td>
<td></td>
<td>100 42</td>
<td>92 42</td>
<td>80 43</td>
<td>Died</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

B. Shock with Tourniquet: Blood Pressure and Cell Volume

<table>
<thead>
<tr>
<th>Dog #</th>
<th>Normal B.P.C.V.</th>
<th>No. Hours</th>
<th>Trauma</th>
<th>1/2 hr.</th>
<th>1 hr.</th>
<th>1 1/2 hr.</th>
<th>2 hr.</th>
<th>2 1/2 hr.</th>
<th>3 hr.</th>
</tr>
</thead>
<tbody>
<tr>
<td>706</td>
<td>108 48</td>
<td>5 hrs.</td>
<td>None</td>
<td>64 62</td>
<td>52 65</td>
<td>30 65</td>
<td>22 65</td>
<td>8 66</td>
<td></td>
</tr>
<tr>
<td>707</td>
<td>78 41</td>
<td>2 1/2 hrs.</td>
<td>5 min.</td>
<td>106 40</td>
<td>98 41</td>
<td>78 43</td>
<td>50 43</td>
<td>62 44</td>
<td>58 43</td>
</tr>
<tr>
<td>714</td>
<td>143 39</td>
<td>4 hrs.</td>
<td>5 min.</td>
<td>154 38</td>
<td>148 37</td>
<td>138 38</td>
<td>126 39</td>
<td>Cannula off</td>
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</tbody>
</table>


<table>
<thead>
<tr>
<th>Dog #</th>
<th>Normal</th>
<th>Anesthetized-</th>
<th>Burned</th>
<th>1½ hrs.</th>
<th>3 hrs.</th>
<th>4½ hrs.</th>
<th>6 hrs.</th>
<th>7½ hrs.</th>
<th>9 hrs.</th>
</tr>
</thead>
<tbody>
<tr>
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<td>130 37</td>
<td>130 45</td>
<td>124 48</td>
<td>122 52</td>
<td>100 53</td>
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<td>30 58</td>
<td>Died</td>
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</tr>
<tr>
<td>739</td>
<td>124 42</td>
<td>140 42</td>
<td>152 36</td>
<td>170 43</td>
<td>170 49</td>
<td>150 44</td>
<td>150 45</td>
<td>Died</td>
<td></td>
</tr>
<tr>
<td>724</td>
<td>110 41</td>
<td>107 40</td>
<td>112 47</td>
<td>115 53</td>
<td></td>
<td>96 58</td>
<td>87</td>
<td>Died</td>
<td></td>
</tr>
<tr>
<td>719*</td>
<td>120 45</td>
<td>112 42</td>
<td>133 47</td>
<td>135 57</td>
<td>125 59</td>
<td></td>
<td></td>
<td>155 56</td>
<td></td>
</tr>
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<td>109 40</td>
<td>102 46</td>
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<td></td>
<td>100 39</td>
<td></td>
</tr>
<tr>
<td>722*</td>
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<td>105 33</td>
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<td>115 36</td>
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<td></td>
<td>Recovered</td>
<td></td>
</tr>
<tr>
<td>730*</td>
<td>120 36</td>
<td>103 41</td>
<td>99 40</td>
<td>87 42</td>
<td>85 42</td>
<td>At 14 hrs. 80 44</td>
<td>Recovered</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* These four dogs were treated with Torantil brand of histaminase (Winthrop) a pig stomach wall extract.
CONCLUSIONS

1. A fall in white blood count may be found in traumatic, thermal, and probably in other types of shock.

2. Concentration of red blood cells is not a necessary part of traumatic shock; indeed it is the exception rather than the rule.
PART III

PERITONEAL ASPIRATION IN THE DIAGNOSIS
OF STRANGULATED INTESTINE
ACKNOWLEDGEMENT

This study was suggested by Dr. F. C. Hill, who has done much brilliant research in clinical and laboratory problems in intestinal obstruction. Frequent reference will be made to the earlier work of Dr. Hill and his associates. He appreciates the difficulties of the clinician in differentiating strangulated intestinal obstruction from simple obstructions of the bowel or other intra-abdominal diseases. He has found the procedures outlined in this paper of value in such a differential diagnosis.
INTRODUCTION

In the course of some experiments on dogs in which a loop of strangulated bowel was placed in a rubber bag, Stoner and Hill observed that within a few hours the bag began to fill with an exudate, profuse in amount, which had a cherry-red or pink color. This fluid appeared before the loop ruptured and the redness of its color gradually increased until after the loop ruptured, when it of course took on the appearance of the contents of strangulated bowel. They showed that this fluid is toxic when injected intravenously into another animal. In order to confirm the presence of this exudate, in the absence of the rubber bag, although it has been previously mentioned by Scott and Wangensteen who studied the fluid found in various types of obstructions in dogs, they produced strangulated loops of bowel four inches long in another series of dogs. It was found that fluid which was exactly similar to the fluid present in the bag formed in the abdominal cavity of these animals. The fluid contains blood and the erythrocytes are present in approximately the concentration which is produced when one drop of blood is placed in one cubic centimeter of water.

Richardson in 1920 called attention to this rusty, blood-stained fluid in cases of strangulation of the bowel and suggested
that the appearance of such peritoneal fluid might aid in the
diagnosis of intestinal obstruction after the abdomen had been
opened. In a series of 135 cases of intestinal obstruction
he found twenty-one in which there were strangulation of the
bowel and this typical fluid. There were also a few cases in
which, although there was interference with the mesenteric
circulation, it was definitely stated that there was no rusty
fluid. Richardson believed that these cases came to operation
before the fluid had time to form. In some cases of simple ob-
struction he noted that clear, straw-colored fluid may be present.
7
Scott and Wangensteen in their studies on various types of obstruct-
ions in dogs found that when the mesenteric veins were ligated,
50 to 250 cubic centimeters of hemorrhagic fluid having low protein
content and little if any odor, developed. In those dogs with
arterial or with both venous and arterial obstruction, 100 to
500 cubic centimeters of dark, bloody, foul-smelling fluid with
high protein content were present.
EXPERIMENTAL WORK

Because of the striking character of this fluid and its positively diagnostic nature when present, we were led to perform a series of experiments on dogs in which we attempted to aspirate fluid from the abdominal cavity at various intervals after a strangulation-obstruction had been produced. The procedures followed were the same on all animals: Under intratracheal ether anesthesia, and using the usual sterile precautions, a mid-line incision was made and a segment of jejunum four inches long and one foot below the ligament of Treitz was ligated with cotton tape. The vessels of the mesentery were included in the ligature but the tape was not pulled tightly enough to obstruct the arteries. The abdomen was then closed in layers, by the usual method. The peritoneal cavity was aspirated, using a lumbar puncture needle at one, two, four and six hour intervals after the operation.

As shown in the accompanying table fluid was obtained one hour after operation in two dogs which contained in one instance many red blood cells and white blood cells, and in the other white blood cells only. At two hours in three of four animals, fluid was obtained which under the microscope showed many red blood cells and white blood cells. Culture of this fluid showed no growth in one, and short chain streptococci and gram negative short rods
in another. At four hours, aspiration of the abdominal cavity of all four animals revealed typical reddish fluid which was diagnostic of strangulation, and culture from this time on was always positive for bacteria which we did not identify. At six hours in all animals, the same type of fluid was obtained. The animals died during the night, and in all cases the loop was found at necropsy to be ruptured.

From the bacteriological evidence (See Tables) it might be suspected that the bowel had been perforated in securing the four and six hour samples. The organisms found were gram negative short rods, probably colon bacilli, and the short chain streptococci which too are almost constant inhabitants of the intestinal tract. In these four cases the dogs died during the night and the loop was found to be ruptured at autopsy in the morning (twenty-three hours Post-Operative). There was no way of knowing whether or not the loop had been punctured. To make sure that this was not the case, two additional dogs were operated upon and obstructed loops two feet in length were produced. The loops were made this large to insure the greater possibility of puncturing the intestinal loop. Scott and Wangensteen found that in this type of operation strangulation occurred in from four to six hours. This agreed with our findings on aspiration so well that we accepted their estimate of the time of death
<table>
<thead>
<tr>
<th>Dog No.</th>
<th>Hours After Obstr.</th>
<th>Amount of Fluid</th>
<th>Color</th>
<th>Smear of Fluid</th>
<th>Culture of Exudate</th>
<th>Autopsy Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>547 Wt.</td>
<td>2</td>
<td>1 drop Clear</td>
<td></td>
<td>Few RBC; many WBC</td>
<td>Negative</td>
<td>Loop ruptured and sour smelling purulent fluid present. Loop, black Omentum, and intestines bound together with adhesions.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Many RBC, WBC and large fat globules.</td>
<td>Aerobic: few short chain strep. Slightly acid. Anaerobic: few gram negative short rods.</td>
<td></td>
</tr>
<tr>
<td>23 lbs.4</td>
<td>4</td>
<td>4 drops Rusty</td>
<td></td>
<td>Same as above</td>
<td>Anaerobic: pellicle forming gram negative short rods and short chain strep.</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>2 cc.</td>
<td>2 cc. Rusty</td>
<td></td>
<td>Same as above</td>
<td></td>
<td></td>
</tr>
<tr>
<td>553 Wt.</td>
<td>2</td>
<td>None</td>
<td></td>
<td>WBC</td>
<td>Aerobic: acid, no gas formed</td>
<td>Loop ruptured and sour smelling fluid present, no adhesions.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Anaerobic: Pellicle formed</td>
<td></td>
</tr>
<tr>
<td>11 lbs.4</td>
<td>1</td>
<td>1 drop Rusty</td>
<td></td>
<td>RBC and WBC</td>
<td>Aerobic: gram positive and gram negative short rods, many short chain strep. acid forming. Anaerobic: pellicle and coal gas odor, gram negative short rods</td>
<td>Ruptured loop Sour purulent fluid 100 cc.</td>
</tr>
<tr>
<td>6</td>
<td>3 drops Rusty</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>555 Wt.</td>
<td>1</td>
<td>1 drop Clear</td>
<td></td>
<td>WBC</td>
<td>Aerobic: short chain strep. Anaerobic: gram negative short rods.</td>
<td>Ruptured loop same as above.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Many RBC and WBC</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10 lbs.2</td>
<td>1</td>
<td>1 drop Rusty</td>
<td></td>
<td>WBC, many RBC</td>
<td>Aerobic: staph, strep. gram negative, rods. Anaerobic: short chain strep. gram positive rods.</td>
<td></td>
</tr>
<tr>
<td>560 Wt.</td>
<td>1</td>
<td>1 drop Clear</td>
<td></td>
<td>Many RBC and WBC</td>
<td>Aerobic: acid-forming short gram negative rods, short chain strep. Anaerobic: same as above.</td>
<td></td>
</tr>
<tr>
<td>12 lbs.2</td>
<td>10</td>
<td>10 drops Rusty</td>
<td></td>
<td>Many RBC and WBC</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>2 drops Rusty</td>
<td>WBC -90% Polys RBC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
and loop rupture (which averaged sixteen hours). In accordance
with this observation the abdomen was reopened (post mortem) at
fourteen to eighteen hours and intact loops of bowel removed.
The loops were closely examined for perforations but none found.
About forty cubic centimeters of rusty fluid was found in the
peritoneal cavity in each instance. Samples of this fluid were
removed aseptically for culture and smear, and the findings
agreed qualitatively with those made at fourth and sixth hour
punctures, although the quantity of organisms and cellular
matter was much greater. There were no purulent exudates or
adhesions present in ether case. The fluid found within the
obstructed loop was grossly different from the intraperitoneal
fluid and no further differentiation seemed necessary. It may
be assumed therefore that none of the fluid aspirated on ab-
dominal puncture came from within the obstructed loops of
bowel, and that these loops were not perforated during the
abdominal puncture.
DISCUSSION

2,1
 Danzer and Neuhoff and Cohen have previously reported on the value of peritoneal aspiration in the clinical diagnosis of various types of peritonitis and both emphasize the safety of the procedure and point out the difficulty of puncturing the intestine. The bowel moves away from the point of the needle and it takes considerable force to enter into its lumen. If the bowel should happen to be adherent to the peritoneal wall at the site of puncture, the intestine probably would be penetrated, but under these conditions the puncture would be of little importance since there would be no leakage into the peritoneal cavity. The site chosen for puncture is probably of little importance. If a mass is present it should be avoided and the needle of course should be inserted high enough above the symphysis to miss the bladder. It is not difficult to tell when the needle enters the peritoneal cavity and with ordinary care we believe that the procedure is safe. It sometimes requires aspiration for as long as a minute to obtain the fluid, according to Danzer, and we believe that if a needle with a stylet and a two-way stopcock is used, so that there will be no leakage of air into the peritoneal cavity before suction can be applied, that it would be easier to aspirate the fluid.
We are not reviewing the value of this procedure in the diagnosis of other intra-abdominal conditions, because Horsley and the authors previously quoted have already done so, but wish to emphasize that in a patient who may have a strangulated bowel and on whom for various reasons, postponement of operation is being considered, peritoneal aspiration may point to the absolute necessity of surgery and may save the patient's life. The absence of fluid on peritoneal aspiration does not rule out strangulated bowel, but one drop of the characteristic fluid proves the presence of an abdominal emergency and calls for immediate surgery. A drop of fluid is all that is necessary, for if this is placed on a slide the presence of red blood cells in a clear fluid is diagnostic.
CONCLUSIONS

1. There is a characteristic rusty fluid found in the peritoneal cavity four hours after strangulation of intestine.

2. This fluid may be obtained by simple aspiration of the peritoneal cavity, and when obtained it is diagnostic.

3. Contrary to popular opinion there is little danger of perforating the intestine when aspirating the peritoneal cavity.
REFERENCES

PART I


33. Zwecker, I. T., "Does Splenectomy Influence Leukocytosis Induced by Injection of Certain Foreign Substances?" Arch. Path. 7: 1012, 1929.
REFERENCES

PART II


REFERENCES

PART III


