Rationale for Transthoracic Esophageal Transection for Bleeding Varices

YASUO IDEZUKI, M.D.,** MITSUO SUGIURA, M.D., KEISUKE SAKAMOTO, M.D.,
HIDECHI ABE, M.D., TSUYOSHI MIURA, M.D., SHIGERU HATANO, M.D., PH.D†
AND SEIJI KIMOTO, M.D., PH.D.‡
Tokyo, Japan

PORTAL-SYSTEMIC SHUNTS HAVE LONG been considered the operation of choice for bleeding from esophageal varices associated with portal hypertension, although impossible or inadvisable in many patients. It may be impossible because there is no suitable patent vein in the portal circulation to anastomose to a systemic vein, or inadvisable because the liver function is too seriously impaired for this organ to overcome the temporary strain on its metabolism which a portacaval shunt imposes or because of the risk of subsequent portal systemic encephalopathy may be too great.

Since attempts to stop bleeding in these patients by conservative measures were disappointing, some definite operative measures other than shunt surgery should be performed.

Attempts to stop bleeding by operations directed to the esophageal varices range from trans-esophageal injection of varices as a minimal operation to total removal of the thoracic esophagus with replacement by jejenum. The results of these operations so far reported have been discouraging.

In 1950, Walker7 successfully treated a patient with bleeding esophageal varices by dividing the esophagus at the level of the hiatus and its reconstruction with resuture. Among the operations directed to the esophageal varices, this procedure seems to be the most complete and drastic for the interruption of the collateral blood flow to the esophageal varices and hence the most reasonable operation. This investigation was performed to re-evaluate the efficacy of Walker's operation and to clarify the rationale for it.

EXPERIMENTAL MATERIAL AND METHODS

Sixty-seven mongrel dogs weighing between 8 and 18 kilograms were divided into three groups. Intravenous anesthesia with pentobarbital sodium (Nembutal) 25 mg per kilogram of body weight was used both for abdominal and thoracic operations and intubation with controlled room-air respiration using a positive pressure automatic respirator was added during the intrathoracic procedures.

The first group of 17 dogs served as the control for the other groups. Gradual occlusion of the portal vein was made to produce portal hypertension and to study the development of collateral circulation and its possible change in passage of time.

After the abdominal cavity was entered through a midline incision, the portal vein was constricted to approximately 50 per cent of its circumference with reactive celosponge tape above its highest tributary.

Portal pressure was measured with a water manometer after cannulating a branch of the inferior mesenteric vein, before and immediately after the constriction of the portal vein. Splenoportography using 25 to 30 ml of 76 per cent meglumine and sodium diatrizoate (Urografin) was performed after the constriction of the portal vein and was repeated at approximately three-week intervals up to nine weeks to follow-up the development of collateral venous circulation. Portal vein and collaterals were carefully revised by necropsy at the time of animal death or sacrifice.

Group 2 (28 dogs). This series was designed to study mainly the effects of eso-
phageal transection on the established collateral circulation and its regeneration after this procedure.

Gradual occlusion of the portal vein was made by the same method as in group 1. Two to five weeks later, when the development of the venous collateral circulation was confirmed by splenoportography, transthoracic esophageal transection was performed. A left 7th intercostal space thoracotomy was made and the lower esophagus was dissected free. The esophagus was then transected 1 to 2 cm above the esophagocardiac junction. Both right and left periesophageal veins and the vagus nerves were doubly ligated and interrupted at the level of the transection of the esophagus. The esophagus was reconstructed with continued silk sutures in two layers. The chest was closed in layers with a catheter left in place to assure adequate inflation of the lung at the end of the procedure.

Follow-up splenoportograms were taken after the esophageal transection at different intervals until 125 days later to observe the changes in the collateral circulation and to study the regeneration of collaterals in the esophageal region. Portal pressures were measured in six dogs during the operation to study whether the pressure might increase by esophageal transection or not.

Group 3 (22 dogs). In this group, the two procedures were performed in the reversed order, i.e., transthoracic esophageal transection at first and constriction of the portal vein as the second procedure, in an attempt to observe the development of collateral circulation across the scar tissue at the suture line of the esophageal reconstruction. Intervals between the two procedures were 21 to 139 days.

Venous collateral circulation was studied by splenoportograms made 13 to 37 days later and was revised by necropsy at the time of animal death or sacrifice.

**Experimental Results**

Two dogs of Group 1 died soon after the operation because the portal constriction was too tight. The remaining 15 were available for the long-term study. Average rise in portal pressure immediately after the portal constriction was 37 per cent of the preconstriction level. The portal pressure usually tended to decrease slowly after it had reached its maximum several weeks later but it remained elevated throughout the observation period in 14 dogs. In one dog, in which the portal pressure had not risen 11 days later, cellophane tape was found to be dislocated from the portal vein.

Splenoportograms made immediately after the portal constriction showed the engorgement of the spleen, but abnormal collaterals were not demonstrated. By splenoportogram three weeks later, the complete occlusion of the portal vein was revealed in 12 dogs and very narrow constriction in two. In the 14 dogs in which the portal constriction was successful, rich collateral channels were found between the portal and systemic venous systems both in the abdominal and thoracic cavities.

The abnormal anastomotic channels were found and classified at necropsy in the following areas: 1) in and around the esophagus (hepatofugal); 2) in the hepatoduodenal and hepatogastric ligaments (hepatopetal); 3) in the lienocolic ligament (hepatofugal); 4) in the duodenocolic ligament (hepatofugal). Since a great number of vessels were superimposed in the splenoportogram, it was impossible to distinguish the two hepatofugal collaterals in the abdominal cavity separately by splenoportography. However, it was possible to distinguish the following three collaterals in the splenoportogram: i.e.: 1) esophageal (hepatofugal); 2) periportal (hepatopetal), and 3) mesenterial and retroperitoneal (hepatofugal).

Esophageal collaterals were always demonstrated and were the most prominent in ten dogs. These collaterals remained almost unchanged throughout the observation period in spite of the tendency to decrease in portal pressure after it had reached its maximum.

Of the 21 dogs in group 2 which survived the gradual occlusion of the portal vein and underwent the transthoracic
esophageal transection, only ten survived more than two weeks and were subjected to further study.

Periesophageal veins and veins in the adventitia of the esophagus were always found to be dilated and tortuous at the time of esophageal transection, but submucosal varices were not found. Portal pressure was high in all six at the time of esophageal transection. However, no significant changes in portal pressures occurred by the transection of the esophagus.

Splenoportography before the esophageal transection always revealed development of abundant collaterals in the same areas as in Group 1 (Fig. 1). After the esophageal transection, all the venous collaterals above the diaphragm disappeared. The portal collaterals below the diaphragm usually became more numerous and abundant, and the opacification of the abdominal vena cava intensified (Fig. 2). These findings show that the pariesophageal collaterals were interrupted completely by esophageal transection and that the blood flow formerly directed to these channels began to flow through the collaterals already developed in the abdomen to the abdominal vena cava. These findings remained almost unchanged throughout the observation period, and the regeneration of the collaterals across the diaphragm to the esophagus could no longer be demonstrated in nine dogs (Fig. 3), although a few very fine vessels were seen to regenerate across the diaphragm in one after 16 weeks.

Of the 15 dogs in group 3 which survived the esophageal transection and then underwent the gradual occlusion of the portal vein, nine survived more than three weeks, the period shown to be sufficient for the development of collateral circulation.

---

**FIGURE 1:** Splenoportogram made 12 days after portal constriction (Group 2, Dog No. 29) shows complete occlusion of portal vein and development of abundant collateral circulation in the periportal (hepato-petal), esophageal, mesenterial, and retroperitoneal regions (hepatofugal).

**FIGURE 2:** Splenoportogram made immediately after esophageal transection (Group 2, Dog No. 29). Collaterals in the esophageal region have completely disappeared.

**FIGURE 3:** Splenoportogram made 20 days after esophageal transection (Group 2, Dog No. 29). Collaterals in the esophageal region have no longer been demonstrated. Note the development of abundant and numerous collaterals in the mesenterial and retroperitoneal regions, and the intense opacification of the abdominal vena cava.
Splenoportograms performed later than 21 days revealed the development of rich collateral circulation in the abdomen. In contrast to the findings in group 1, collateral channels across the diaphragm were not prominent. Esophageal collaterals could not be demonstrated at all in four dogs, and were only very scarcely observed in five. Abundant collaterals below the diaphragm, possibly the tributaries of the left gastric vein, were demonstrated as in the former two groups, but communication with the esophageal veins from these vessels could not be found in most of the animals. In seven dogs, these vessels were seen to anastomose directly with the left branch of the intrahepatic portal vein becoming an effective hepatopetal collateral channel (Fig. 4).

These findings probably indicate that anastomoses are unlikely to occur across the tight scar tissue of the suture line of the esophageal reconstruction.

**Clinical Material and Methods**

Since July, 1964, transthoracic esophageal transection has been carried out in 15 patients, 12 men and three women. All patients had the history of more than one hemorrhage from the esophageal varices and were radiologically proved to have prominent esophageal varices on admission to the hospital.

Shunt surgery was impossible or inadvisable because in three there were no suitable patent veins for portal systemic anastomosis, in ten liver function was intolerably impaired for shunt surgery, and in two, shunt surgery had already been performed elsewhere but hemorrhage recurred. The operation was performed as an elective measure in 14, and as an emergency operation during massive bleeding in one.

**Operation: Operative technique is almost the same as described by Walker.** The patient is placed on his right side and the chest is entered through the left 7th or 8th intercostal thoracotomy. The mediastinal pleura is incised over the lower esophagus, which is mobilized by finger dissection, and the tape is passed around it. The lower esophagus is then freed up to 7 to 8 cm from the hiatus. At this stage, both or one vagus nerves are dissected free and so preserved when the esophagus is cut across. A clamp is placed on the esophagus as low as possible and the second clamp is placed 4 or 5 cm above the first clamp. The esophagus is divided transversely between the two clamps, 3 to 4 cm above the esophagocardiac junction (Fig. 5). It is important to divide the mucosa and submucosa, in which large varices lie, completely across.

We agree with Walker's opinion that all of the muscle layer need not always be cut off. Usually we preserved the right half of the circumference of the muscle layer in-
tact to avoid the accidental severance of the right vagus nerve, and to diminish the possibility of the post-anastomotic stricture. The esophagus is repaired with interrupted 4-0 silk sutures in two layers. The mediastinal pleura is brought together with a few stitches and the chest closed after insertion of intercostal drain.

In eight patients, transabdominal splenectomy was performed at the same time for coexisting splenomegaly and hypersplenism.

**Investigations**

Portal pressure: In six of eight patients in whom laparotomy was performed for splenectomy, pressures were recorded during transthoracic esophageal transection. A No. 8 Cournand Teflon catheter was introduced into the portal vein via a branch of the superior mesenteric vein and was connected to a water manometer. The base line of the water manometer was level with the porta hepatis.

Esophageal varices: Esophageal varices were followed radiologically. Esophagoscopy was also performed after the operation in six. Observation of the x-ray film and endoscopy were done by more than two doctors in order to avoid the possibility of error in the interpretation of the findings.

Collaterals: Changes of the portal collateral circulation were studied by portography in one patient, and by selective celiacography in seven. Portography was performed using 30 ml of 76 per cent meglumine and sodium diatrizoate (Urografin) by splenic puncture before the operation and by cannulation into a branch of the superior mesenteric vein after splenectomy. The percutaneous technique of the selective celiacography was the same as described by Seldinger. The venous phase of the selective celiacography was utilized for the observation of both the portal vein and the collateral circulation. Contrast dye used was 30 ml of either 76 per cent meglumine and sodium diatrizoate (Urografin) or meglumine iothalamate (Conray).

**Clinical Results**

The results are shown in Table 1. One patient in whom the esophageal transection was performed as the emergency procedure died a few hours later, although the bleeding had been stopped by esophageal transection. This patient was in the precoma-

![Diagram](http://example.com/diagram.png)

**Figure 5:** Schematic drawing of transthoracic esophageal transection. All of the collaterals to esophageal varices are divided 4-5 cm above the esophagocardiac junction.
<table>
<thead>
<tr>
<th>No.</th>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Diagnosis</th>
<th>Previous Surgery</th>
<th>Esophageal Varices</th>
<th>Recurrent Hemorrhage</th>
<th>Complications</th>
<th>Follow-up Period (Mo.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>S.S.</td>
<td>22</td>
<td>M</td>
<td>Congenital malformation of portal vein</td>
<td>Splenectomy + S-R shunt (12 years ago)</td>
<td>++++</td>
<td>-</td>
<td>None</td>
<td>Stricture of esophagus</td>
</tr>
<tr>
<td>2</td>
<td>M.N.</td>
<td>22</td>
<td>M</td>
<td>Liver cirrhosis</td>
<td>Splenectomy (5 years ago)</td>
<td>++</td>
<td>-</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>3</td>
<td>K.F.*</td>
<td>49</td>
<td>M</td>
<td>Liver cirrhosis</td>
<td></td>
<td>++</td>
<td>-</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>4</td>
<td>K.F.*</td>
<td>48</td>
<td>F</td>
<td>Banti's syndrome</td>
<td></td>
<td>++++</td>
<td>-</td>
<td>None</td>
<td>Operative death</td>
</tr>
<tr>
<td>5</td>
<td>C.S.</td>
<td>46</td>
<td>M</td>
<td>Liver cirrhosis</td>
<td></td>
<td>++++</td>
<td>-</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>6</td>
<td>S.I.*</td>
<td>60</td>
<td>M</td>
<td>Banti's syndrome</td>
<td></td>
<td>++++</td>
<td>-</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>7</td>
<td>K.K.</td>
<td>40</td>
<td>M</td>
<td>Liver cirrhosis</td>
<td></td>
<td>++</td>
<td>-</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>8</td>
<td>T.K.</td>
<td>15</td>
<td>M</td>
<td>Congenital malformation of portal vein</td>
<td>Splenectomy (10 years ago)</td>
<td>++</td>
<td>-</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>9</td>
<td>H.K.*</td>
<td>49</td>
<td>M</td>
<td>Liver cirrhosis</td>
<td></td>
<td>++++</td>
<td>-</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>10</td>
<td>R.H.*</td>
<td>53</td>
<td>F</td>
<td>Liver cirrhosis</td>
<td></td>
<td>++++</td>
<td>-</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>11</td>
<td>M.Y.*</td>
<td>21</td>
<td>M</td>
<td>Liver cirrhosis</td>
<td></td>
<td>++++</td>
<td>-</td>
<td>None</td>
<td>Pneumonia</td>
</tr>
<tr>
<td>12</td>
<td>K.A.*</td>
<td>16</td>
<td>M</td>
<td>Congenital malformation of portal vein</td>
<td></td>
<td>++</td>
<td>-</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>13</td>
<td>K.K.</td>
<td>23</td>
<td>F</td>
<td>Congenital malformation of portal vein</td>
<td>Splenectomy (7 years ago)</td>
<td>++++</td>
<td>-</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>14</td>
<td>K.W.*</td>
<td>10</td>
<td>M</td>
<td>Congenital malformation of portal vein</td>
<td></td>
<td>++++</td>
<td>-</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>15</td>
<td>K.Y.</td>
<td>27</td>
<td>M</td>
<td>Congenital malformation of portal vein</td>
<td>Splenectomy (10 years ago)</td>
<td>++++</td>
<td>-</td>
<td>None</td>
<td>None</td>
</tr>
</tbody>
</table>

* Esophageal transection and splenectomy.
tous state of liver failure and it is reflected
if any of the operation had been justifiable
in this patient. The other 14 patients re-
covered uneventfully from the operation
and had no further bleeding.

Moderate post-anastomotic stricture of
the esophagus was observed as a late com-
plication in one patient, but it was treated
successfully by endoscopic dilatation.

Portal pressure: Portal pressure changes
in six patients are shown in Fig. 6. Portal
pressure increased slightly in one but rather
decreased in five (Fig. 6).

Esophageal varices: Before the esopha-
geal transection, varices were always very
prominent. Varices disappeared completely
after the operation and could no longer be
demonstrated radiologically in 14 (Fig. 7).
X-ray observations were confirmed by
esophagoscopy in six.

Collaterals: Preoperative splenoporto-
gram of case 9 revealed the opacification
of the large and tortuous left gastric and
paraumbilical veins, which were the effec-
tive outflow tracts in the portal circulation
in this patient. After the esophageal tran-
section, the left gastric vein was no longer
opacified, showing that the blood flow to
the esophageal varices had been completely
interrupted by this procedure. The post-
operative portogram also demonstrated the
regurgitation of the dye into the inferior
mesenteric vein and showed the possibility
for this vein to become an effective out-
flow tract after the operation (Fig. 8). No
significant change in the portal pressure
was observed in this patient.

Preoperative selective celiacography re-
vealed prominent portal collaterals in the
areas of both left gastric and short gastric
veins in two of the seven cases. These col-
laterals disappeared completely after the
operation.

DISCUSSION

Direct application of the experiments of
portal hypertension in animals to human

![Figure 6: Portal pressure changes in six patients. (E. T.: esophageal transection).](image-url)
patients is sometimes open to criticism because of some differences in portal hypertension among various mammalian species.

Essential differences hitherto reported in literature\textsuperscript{1,14,16} are: 1) true esophageal varices in the submucous layer do not develop in the dog and hence no bleeding occurs from esophageal varices; 2) persistence of the portal hypertension is limited and it depends on the balance between the degree of obstruction of venous outflow tract and adequacy of the collateral channels; 3) liver function is not impaired usually in the dog with the extrahepatic portal obstruction.

Present investigations in dogs support these observations.

However, there are also some similar-

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure7.png}
\caption{Barium varicography of Case 1 (SS, 22-year-old man). (A) before esophageal transection; (B) 16 months after esophageal transection.}
\end{figure}

\textit{FIGURE 7}
The third group was designed to study the development of collateral circulation across the established scar tissue of the suture line of transection.

By analyzing the results of these three groups separately and then summarizing, the following conclusions may be obtained without much controversy.

1. Esophageal transection extirpates all the collaterals in the esophageal region for certain periods, and during this period, we may expect the collaterals in the abdomen to become more numerous and abundant and for these collaterals to work as the more effective outflow tract in the portal circulation after esophageal transection.

2. Regeneration of the anastomotic channels in the reconstructed esophagus is unlikely to occur because of the tight scar tissue at the suture line, and this is particularly true when the effective collaterals are already established in the abdomen.

3. Portal pressure will not be raised any more than pre-transection level as long as these intra-abdominal collaterals work as an effective out-flow tract of portal circulation and compensate for the extirpation of the periesophageal channels.

Supported by these experimental backgrounds, we have applied this operation to human patients. The results in patients so far attained are satisfactory. Pre- and post-operative observations by splenoportography and by selective celiacography demonstrated similar changes in collateral circulation as in the animal experiments. Portal pressure recordings showed little variation during the esophageal transection and indirectly showed the compensation for the extirpated collaterals in the esophagus with other collaterals in the abdomen.

Disappearance of varices after the operation were common in all patients. However, the ultimate evaluation of the esophageal transection should be postponed for some time because the follow-up period of our cases is yet insufficiently long.

Walker7 stressed the importance of complete elimination of esophageal varices in the thoracic cavity, which ensued by di-

![Figure 8A](image1.png)  
**Figure 8A:** Portography of Case 9 (H.K., 49-year-old man). (A) before esophageal transection and, (B) immediately after esophageal transection.
viding the mucosa and submucosa of the esophagus at the hiatus level. Esophageal transection has the advantage over Crile’s operation (transesophageal suture of esophageal varices) in the completeness of the elimination of the varices, and has advantages over esophagocardiac resection or total removal of the esophagus in the simplicity of the operative technique and in the preservation of the cardiac sphincter mechanism, which is important to prevent reflux esophagitis.

Development or regeneration of small anastomotic channels across the suture line may occur after esophagocardiac resection as well as after the esophageal transection, but the operative risk is apparently smaller in the latter.

Walker reported the follow-up results of transthoracic esophageal transection in 53 patients in 1964. More than 60 per cent of his patients have had no recurrent hemorrhages after the operation, and liver failure or portal systemic encephalopathy did not ensue from the operation.

Recurrent hemorrhages were apparently greater than those with portacaval shunts (Linton and associates 19 per cent, McDermott and co-workers 15 per cent, Hal lenbeck and colleagues 24 per cent), but his results were unexpectedly good among the operations of this type.

Both our clinical and experimental investigations clearly showed the possibility of the complete elimination of the esophageal varices by esophageal transection and also the probability of the persistence of the effects in some of the cases.

Transthoracic esophageal transection seems to be the operation of choice in cases of extrahepatic portal block or severe liver failure of cirrhosis in which shunt surgery is impossible or inadvisable.

**Summary**

Rationale for transthoracic esophageal transection has been clarified in animal experimentation, and the results of clinical transthoracic esophageal transection for bleeding varices in 15 patients were reported.

Esophageal transection extirpated all the collaterals to the esophageal varices and the regeneration of the collaterals in the esophageal region was much less than expected, probably because of the sufficient compensation with other collaterals and because of the tight scar tissue at the site of esophageal reconstruction.

Fourteen of the 15 patients survived the operation and had no further bleeding from esophageal varices throughout the follow-up period up to 22 months.

**RESUMEN**

Las razones básicas para la transección esofágica transtorácica han sido clarificadas en animales de experimentación. Se reportan los resultados clínicos de la transección esofágica transstorácica en casos de varices sangrantes.

La transección esofágica elimina todas las colaterales de las varices esofágicas. La regeneración de las colaterales a la región esofágica fue mucho menor de lo que se esperaba, probablemente debido a la compensación suficiente con otras colaterales y el denso tejido de cicatrización en el sitio de la reconstrucción esofágica.

Catorce de los quince pacientes sobrevivieron la operación y no sangraron más de las varices en todo el período de observación de hasta 22 meses.

**RESUMÉ**

Les rôles pour la section transthoracique de l’oesophage ont pu être précisées par l’expérience sur l’animal. L’auteur rapporte les résultats de la section tranoesophagienne-trans thoracique pour saignement de varices chez 15 malades.

La section tranaoesophagienne comporte l’ex tирраtion de toutes les collatérales des varices œsophagiennes, la régénération de leurs collatérales dans la région œsophagienne peut être considérée comme improbable étant donné la compensation suffisante par les autres collatérales et le tissu cicatriciel serré qui se constitue sur la paroi œsophagienne reconstituée.

Sur les 15 malades, 14 ont survécu après l’opération et n’eurent plus d’hémorragie provenant de varices œsophagiennes au cours d’une période de survie qui dépasse 22 mois.

**ZUSAMMENFASSUNG**

Die Gründe für eine transthorakale quere Durchtrennung der Speiseröhre wurden im Tierexperiment geklärt, und die Ergebnisse einer klinischen transthorakalen Oesophagus-Durchtrennung der Speiseröhre beseitigte alle Kollateralen zu den Varizen des Oesophagus und die Regeneration der Kollateralen in der Region des
TRANSTHORACIC ESOPHAGEAL TRANSECTION

Oesophagus war wesentlich geringer als erwartet, wahrscheinlich infolge der ausreichenden Kompen- sation mit anderen Kollateralen und weil das straffe Narbengewebe auf der Seite der oesophagealen Rekonstruktion dies verhinderte.

14 der 15 Patienten überlebten den Eingriff und beobachteten keine weitere Blutung aus ihren Speiseröhren-Varizen—während einer Gesamtbeobachtungszeit von 22 Monaten.

REFERENCES

For reprints, please write: Dr. Idezuki, Box 440, University of Minnesota Hospital, Minneapolis 55453.

WALDENSTROM'S DISEASE WITH LOCALIZATION TO STOMACH AND LUNGS

Waldenstrom's disease is a disease affecting the reticulo-endothelial system. In the majority of cases, the characteristic bone marrow disorders come to light as a result of hemorrhage or neurologic dis- orders. Sometimes, however, the predominant sympto- mptoms involve the ganglia, the liver or the spleen. The report presented demonstrates that although the disease is of protean symptomatology, the symptoms can be localized to the stomach or the lung.