Filtration of the Portal Venous Circulation
For Hepatosplenic Schistosomiasis

Jim Kersting, B.G.S., C.C.P.
Department of Cardiac Surgery
William Beaumont Hospital
3601 W. 13 Mile Rd.
Royal Oak, Michigan 48072

_Schistosoma mansoni_, a 10–25 mm long trematode endemic to Middle Eastern and Nile Delta regions, causes chronic schistosomiasis by inhabiting the intestinal venule circulation and producing eggs which deposit in the portal tracts (and less frequently in the liver) via the portal venous circulation. Portal fibrosis attendant upon the egg load may then result in presinusoidal portal hypertension.

While the eggs, requiring an intermediary snail host, do not mature in the infected human, each female intestinal parasite may deposit approximately three hundred eggs daily for up to thirty years.

In advanced stages of the disease splenectomy is indicated in view of ensuing splenomegaly and portal hypertension.

Since the patient at this stage is in necessity of surgical treatment, the opportunity presents itself for an attempt at surgical removal of the adult _Schistosoma_ worms. This procedure was first applied and reported by Goldsmith and Kean in 1966.1

Our patient was a 33 year old Yemen male who had migrated to the United States in 1970. He was first diagnosed as having Schistosomiasis in 1974, at which time he was treated with twenty injections of Stibophen (Fuadin), intramuscular. In 1975 he was found to have an enlarged spleen upon examination following an automobile accident. He was admitted to this hospital in December 1976 for an acute upper gastrointestinal bleed. A rectal biopsy revealed the presence of schistosomal eggs within the tissue; no treatment for schistosomiasis had been received since the 1974 pharmacotherapy (a regimen of acknowledged limited efficacy).

Because of his known history and confirmation of schistosomiasis, and persistent hypersplenism secondary to splenomegaly, it was elected to perform a splenectomy and a portal blood filtration.

In the operating room the patient underwent spinal anesthesia with supplemental Innovar, and later, nitrous oxide general anesthesia. Following the splenectomy, the 50kg. patient received 130 milligrams (13,000 units) of Liquaemine Sodium Heparin. The stump of the splenic vein was cannulated with a USCI vena caval cannula, threaded down to the junction of the superior mesenteric vein. This cannula and outflow tubing led to a transparent 27 micron pore Bentley PF-427 blood filter, to a roller pump, into a Sarns 6302 perfusion chamber reservoir and bubble trap, to a 40 micron pore Pall EC3840 blood filter; with the extracorporeal circuit finally returning through an Argyle THI perfusion cannula into the left saphenous vein at its junction with the left femoral vein (Figure 1).
Priming solution for the pump circuit consisted of 900ml of Ringer’s Lactate plus 20 milligrams (2,000 units) of Liquaemin Sodium Heparin.

Flow was initiated and a mean flow rate of 800 ml/min at a mean reservoir pressure of 110mm Hg was established. Activated clotting times were maintained at over 400 seconds.

At fifteen minutes after initiation of the extracorporeal portal circulation an initial dose of 100 milligrams (2mg/kg) of antimony potassium tartrate (tartar emetic) was administered intravenously as an attempt to mobilize the worms from the venous circulation of the intestinal tract toward the diverted portal circulation. After thirty minutes, when no worms were visualized in either of the filters, an additional half-dose of 50 milligrams of tartar emetic was administered.

After a total of sixty minutes of extracorporeal filtration following the initial dose of tartar emetic, the pumping was terminated. No worms were visualized, either at this time, or at dump disassembly. One hundred milligrams of Protamine Sulfate was
administered intravenously to yield a post-reversal activated clotting time of 88 seconds.

Extracorporeal filtration to filter *Schistosoma* worms is documented as an effective treatment for advanced schistosomiasis.\(^2\,^3\,^4\) Failure to recover worms in this case may be due to: (1) malpositioning or migration of the proximal cannula, (2) resistance to the antimony, (3) ineffective migration of the worms due to longstanding habitation prior or to desensitization from previous drug therapy. The latter is the likelier; lack of ova in stool examinations also postulates the absence by this time of any live adult worms.

This document remains only the second unsuccessful reported clinical hemofiltration for schistosomiasis. The procedure is uncomplicated and safe, and continues to be an appropriate therapy for advanced stages of the disease.

REFERENCES