Gas in the Portal Vein: A Report of Two Cases

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Gas in the portal venous system was detected on plain roentgenograms of the abdomen in two women aged 61 and 72 years, respectively. Both patients had intestinal necrosis, due in one instance to a small bowel volvulus around a mesenteric band, and in the second instance to occlusion of the celiac axis, superior and inferior mesenteric arteries. In the first patient, the portal venous gas was detected before surgery, and in the second case the gas was observed at laparotomy and was visualized on radiographs of the abdomen taken shortly after death. Both patients died. Portal venous gas can be distinguished radiologically from air in the bile ducts by its characteristic slender branching gas pattern in the periphery of the liver substance. The presence of portal gas in the adult indicates intestinal necrosis in the majority of cases and should lead to early operative intervention.

On a décelé du gaz dans le système porte sur des radiographies simples de l'abdomen de deux femmes âgées respectivement de 61 et de 72 ans. On constatait chez les deux patientes de la nécrose intestinale relevant, dans un cas, d'un volvulus du grêle situé autour d'une adhérence mésentérique et, dans l'autre cas, d'une occlusion du tronc cœliaque et des artères mésentériques supérieure et inférieure. Chez la première patiente, on a décelé la présence de gas veineux avant l'intervention et, dans le second cas, au moment de la laparotomie et le gas a pu être vu sur les radiographies de l'abdomen prises peu de temps après la mort. Les deux patientes sont mortes. Au point de vue radiologique, on peut distinguer l'apparence effilée caractéristique du gaz à la périphérie du parenchyme hépatique. La présence de gaz dans la veine porte signifie une nécrose intestinale dans la majorité des cas et doit imposer une intervention précoce.

GAS in the portal venous system can be recognized on plain roentgenograms of the abdomen. This radiological sign was first described in infants by Wolfe and Evans[1] in 1955, and was first reported in adults by Susman and Senturia[2] in 1960. Undoubtedly this sign occurs more frequently than it is described, and unfortunately occurs more often than it is recognized. Since the medical and surgical literature contains relatively little on this subject and since the recognition of this entity is of considerable clinical significance, our recent experience with two patients who had gas in the portal vein warrants description.

These cases were encountered within a period of several months at the Jewish General Hospital, Montreal, Quebec.

CASE 1.—M.S., a 61-year-old white woman, was admitted to the Jewish General Hospital on July 2, 1964, with a two-day history of intermittent, generalized, crampy abdominal pain. She had no vomiting or diarrhea. The past history was pertinent in that one year before admission she had a perforated duodenal ulcer, which had been closed by suture and reinforced with an omental patch.

On physical examination, she had a blood pressure of 60/40 mm. Hg, with a regular pulse of 80/min. and a respiratory rate of 36/min. The chest was clear. The heart sounds were poorly heard, and there were no murmurs. The abdomen was markedly distended, with generalized tenderness and muscle guarding. No abdominal masses were palpable and no bowel sounds were audible.

She had a hemoglobin (Hb.) of 8.4 g. %. Urinalysis showed a 1+ proteinuria, 0-2 WBC per high-power field and 2-4 RBC per high-power field. Serum potassium was 5.4 mEq./l.; serum sodium, 129 mEq./l.; serum chloride, 82 mEq./l., and the CO_2 combining power, 14 mEq./l.

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Plain films of the abdomen taken shortly after admission showed questionable free air and grossly dilated small and large bowel loops. In the right upper quadrant of the abdomen a slender, branching gas pattern was visible. This was interpreted as gas within the intrahepatic portal venous radicles, and on this basis a presumptive diagnosis of intestinal infarction was made (Figs. 1a and 1b).

The patient was prepared for surgery with whole blood transfusions and electrolyte replacement solutions. Polyvalent gas gangrene antitoxin, antibiotics and hydrocortisone were administered. After several hours of intensive therapy, the patient's blood pressure was 90/70 mm. Hg and her condition sufficiently improved so that surgery could be undertaken.

At laparotomy under general anesthesia, a volvulus of a large segment of the small bowel around a mesenteric band was found. This had resulted in patchy necrosis of the entire small bowel except for the proximal 18 in. of jejunum and the distal 18 in. of ileum. Inspection of the vessels of the small bowel mesentery revealed focal areas where the arteries were actively pulsating and other areas where pulsations were absent. Most of the mesenteric veins appeared to be occluded with fresh thrombus. No note was made at operation of air within the mesenteric vessels. In addition to the small bowel findings, there was a large perforation in the anterior wall of the duodenum measuring approximately 3½ x 2½ in. in size. This perforation communicated with a large abscess cavity which was sealed off from the general peritoneal cavity by omentum. The duodenal perforation was sutured and the necrotic small bowel resected. Intestinal continuity was reestablished by side-to-side jejunoileostomy.

Postoperatively the patient's condition deteriorated progressively. Her blood pressure was extremely difficult to maintain and she died 24 hours after operation. At autopsy there was evidence of generalized peritonitis. The postmortem blood culture was positive for *Pseudomonas aeruginosa*.

CASE 2.—B.M., a 72-year-old white woman, was admitted to the Jewish General Hospital on April 5,

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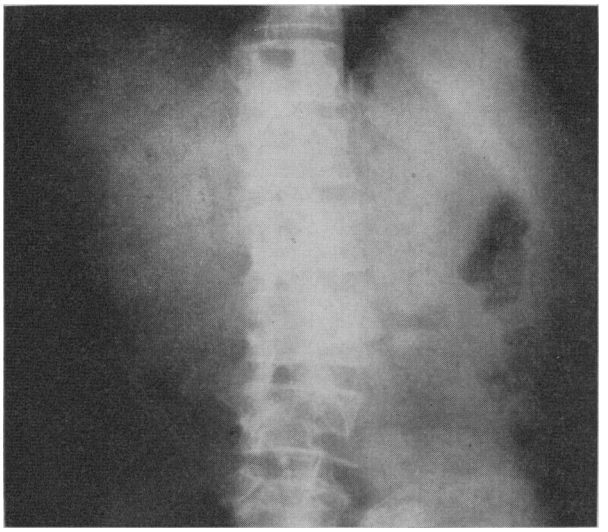

Fig. 1a.—Case 1.—A slender branching pattern characteristic of portal venous gas is noted in the right upper quadrant of the abdomen.

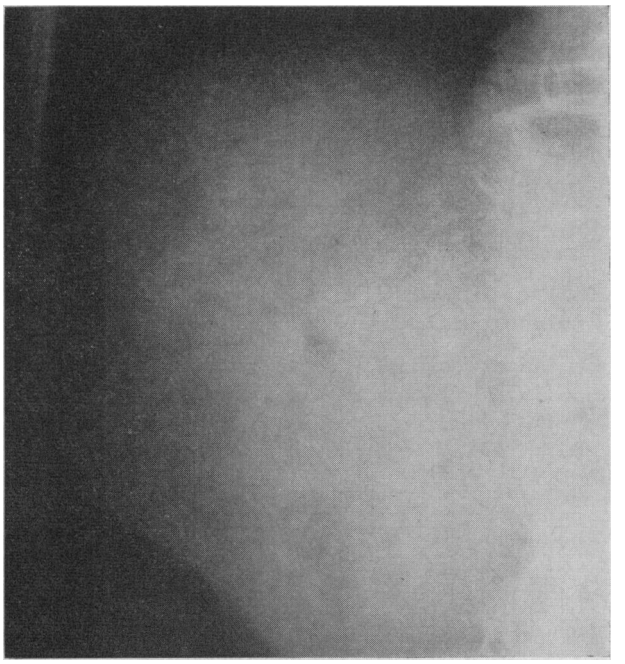

Fig. 1b.—An enlarged view of the right upper quadrant shows the branching pattern in greater detail.

1965, because of an episode of severe precordial pain one week before admission. This pain lasted for several hours and was attributed to coronary ischemia. Following this episode the patient appeared to do well until the day before admission when she complained of pain in the lumbar spine which radiated to the left side of the chest and to the left upper quadrant of the abdomen. Because of the persistence and the severity of the pain, she was admitted to hospital.

The patient's past history was pertinent in that she was a diabetic and had had hypertension for many years. Repeated bouts of congestive failure had responded to the usual therapeutic measures.

On physical examination the blood pressure was 195/120 mm. Hg. Her pulse was regular at a rate of 100/min.; her respiratory rate was 20/min. The oral temperature was 98.4° F. The chest was clear and the heart sounds were well heard. No murmurs were present. On admission the abdomen was slightly distended, with tenderness to palpation in both upper quadrants. Bowel sounds were normal.

The initial ECG revealed evidence of coronary ischemia and the patient was treated for impending myocardial infarction. She was given morphine sulfate, with moderate relief of pain.

Approximately eight hours after admission she suddenly became restless and confused, and her blood pressure dropped to 138/88 mm. Hg. Two hours later the blood pressure could not be obtained and the abdomen became markedly distended, with tenderness throughout. Bowel sounds were no longer audible, and bright red blood was aspirated from a nasogastric tube.

Intensive "antishock" therapy was instituted with rapid administration of blood, plasma and fluids. The blood pressure returned to a level of 120 mm. Hg systolic, and the patient seemed to be improved.

Portable films of the abdomen taken in bed revealed dilated loops of small and large bowel. Gas could be seen in the bowel wall but none was visible in the portal venous system. A diagnosis of superior mesenteric artery thrombosis was made on clinical grounds, and the patient was prepared for surgery.

Under general anesthesia the abdomen was explored through a midline incision. There was necrosis of the entire gastrointestinal tract extending from the gastroesophageal junction to the distal sigmoid. There was extensive necrosis of the gallbladder and patchy necrosis of the liver, spleen and pancreas. The superior and inferior mesenteric arteries and the celiac axis were individually isolated and found to be completely occluded at their origins. The patient's blood pressure at the time of exploration of these vessels was 120 mm. Hg systolic, and good pulsations were palpable in the aorta. There were no pulsations in any of the peripheral mesenteric arteries. Numerous gas bubbles were seen within the large and small branches of the mesenteric veins. No corrective surgery was possible and the abdomen was closed. The patient died six hours after surgery and autopsy permission was refused. Because the preoperative film taken approximately 10 hours before surgery had not revealed gas in the portal vein, a second film was taken immediately post mortem and this showed gas filling the portal vein and its large intrahepatic branches (Figs. 2a and 2b).

DISCUSSION

Plain films of the abdomen may reveal the presence of gas in the portal venous system. A slender, branching gas pattern seen within the liver is characteristic of gas in the intrahepatic portal venous branches. This pattern must be distinguished from air in the biliary duct system. This difference is demonstrated in Fig. 3. When gas is present in the portal vein, the branching gas pattern is observed within the smaller radicles towards the periphery of the liver. On the other hand, when air is present in the bile ducts, it is found in the common bile ducts and the major hepatic ducts but not in the peripheral radicles. The explanation for this difference appears to depend on the different flow patterns of portal venous blood and bile. Gas in the portal vein is carried with the current of blood flow

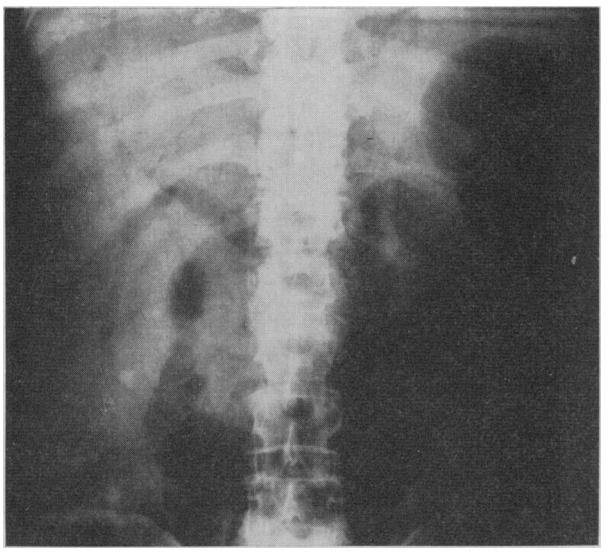

Fig. 2a.—Case 2.—The gas is visible in the portal vein and in the larger intrahepatic portal venous branches.

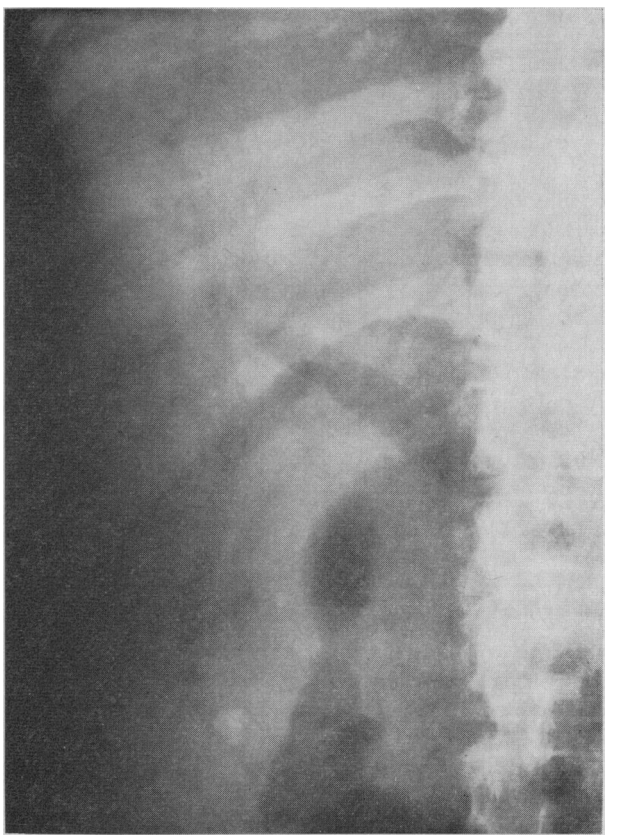

Fig. 2b.—Case 2.—Detailed view of the right upper quadrant.

to the smaller branches in the periphery of the liver, whereas air in the biliary ducts is kept within the larger biliary channels by the current of biliary flow which is from the liver to the second portion of the duodenum.

In Case 2, the gas in the postmortem film is seen in the portal vein and its large branches but not in the periphery of the liver. Since this is a postmortem film, there is no current of flow and consequently the gas has remained in the large portal branches.

The first description of gas in the portal vein was made in 1955 by Wolfe and Evans,[1] who described this sign in six infants. In four, portal gas was observed on roentgenograms of the abdomen made before death and in two the finding was made at postmortem study. The primary disease in three of these infants was erythroblastosis fetalis. In the other three, the primary process was duodenal atresia in one, imperforate anus in another, and diarrhea in the third. Pneumatosis intestinalis was a feature in three. The six infants died. The features common to all were a positive blood culture for *E. coli* and marked gaseous distension of the intestinal tract. One of the infants died seven days after the recognition of portal vein gas.

Portal vein gas was first described in the adult by Susman and Senturia[2] in 1960. Their patient, a 77-year-old white man, had extensive gangrene of the small intestine as a result of superior mesenteric artery thrombosis. The preoperative abdominal film revealed a branching pattern of gas in the hepatic area which was thought to represent air in the biliary tree, and a preoperative diagnosis of gallstone ileus was made. At laparotomy many gas bubbles were noted in the various branches of the superior mesenteric vein, and the true nature of the radiologic sign was then recognized.

Wiot and Felson[3] were the first to diagnose the condition premortem in adults; they described four patients seen during a three-year period: all died. Of three adults, two had necrosis of bowel, due in one to a small bowel volvulus and in the other to superior mesenteric artery thrombosis. The third adult died eight hours after being admitted in coma and in shock. At autopsy there were small bowel distension and mucosal congestion but no ulceration or necrosis. The mesenteric vessels were normal. Two of these patients were diabetics; two had positive blood cultures, one for *Aerobacter aerogenes* and the other for *Proteus mirabilis, Proteus aeroganoids* and *Clostridia paraputrificans.*

Their fourth patient was an infant born with esophageal atresia. This was corrected surgically but the child died on the sixth postoperative day.

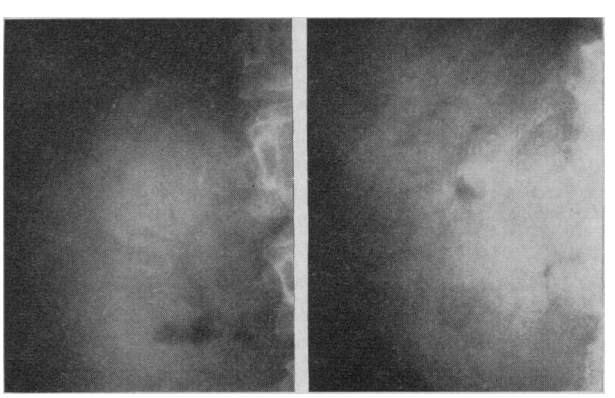

Fig. 3.—A comparison between air in the biliary ducts (left) and gas in the portal vein (right).

At autopsy gas was found within the chambers of the heart, the inferior vena cava, the portal veins and the intestinal arteries. Microscopically, numerous bacterial colonies were noted within the liver. No evidence of intestinal necrosis was present, but the blood culture was positive for *Aerobacter aerogenes.*

Sisk[4] described three cases of portal vein gas in which the gas was detected in the portal venous system on abdominal roentgenograms taken before death. In two instances there was evidence of intestinal necrosis. In the third case, that of a 19-year-old girl, it was noted at operation that many of the mesenteric vessels contained more gas than blood, and that there was marked distension of the stomach and duodenum, ending abruptly at the superior mesenteric artery. This patient died during operation, and at autopsy there was evidence of acute hemorrhagic pancreatitis but no intestinal necrosis.

Barrett[5] described two adults in whom gas was detected in the portal vein on radiographs of the abdomen made before operation. There was intestinal necrosis in both; in one it was due to occlusion of the superior mesenteric artery, and in the other to necrotizing enterocolitis. A culture taken from the portal vein during operation in the first case was positive for *Clostridium welchii.* In the second, *Clostridium welchii* was cultured from liver sections taken at autopsy.

Stewart[6] reported three cases of portal vein gas which was associated with gangrene of the small intestine in each instance. In two, the gangrene was due to superior mesenteric artery occlusion, and in the third to superior mesenteric vein occlusion.

Guyer and Grainger[7] reported two cases of portal gas in which the diagnosis was made on retrospective review of abdominal films. Both patients had extensive intestinal necrosis due to superior mesenteric artery occlusion.

Edwards, Costopoulous and Bell[8] described a 70-year-old patient with known primary macroglobulinemia who was admitted with an abdominal catastrophe and died shortly after admission. An abdominal film taken two hours before death revealed portal venous gas, and a repeat film after gastric decompression with a nasogastric tube revealed a clearer and more striking generalized portal venous gas pattern. At autopsy the bowel was distended, deep blue in colour and congested throughout. The authors did not record the presence or absence of intestinal necrosis or ulceration. There was evidence of thrombosis of the superior pancreaticoduodenal artery with rupture of this artery and resultant hemorrhage into the abdominal cavity. Postmortem culture grew *Aerobacter aerogenes, Clostridium welchii* and *E. coli* from liver, blood and peritoneal cavity.

Rigler and Pogue,[9] in a paper discussing the roentgen signs of intestinal necrosis, described six patients with portal venous gas. The age distribution of these patients was not given. Five of the six patients demonstrated gas in the bowel wall as well as portal venous gas and, similarly, five of the six demonstrated evidence of intestinal necrosis due, in three cases, to mesenteric thrombosis, in the fourth to strangulation obstruction, and in the fifth to necrotizing gastroenterocolitis. In the sixth instance, gas in the portal circulation and evidence of retroperitoneal gas were the result of perforation of a gastric ulcer into the lesser omental sac.

The precise significance and indeed the source of the portal gas have not been clearly delineated. The condition may have a different etiology in adults than in infants because it seems to occur under different circumstances at the two age levels. In the seven infants reported to date intestinal necrosis was not seen, although intestinal distension was a feature in each. However, six of the seven had a blood culture positive for *E. coli* and in the seventh the blood culture was positive for *Aerobacter aerogenes.* A blood culture positive for an organism capable of causing fermentation and thus producing gas may provide an important diagnostic clue.

In the majority of reported cases in adults evidence of intestinal necrosis was found. In several of these, blood cultures were positive for gas-producing organisms. In a number of cases in both adults and infants the gas was not confined to the mesenteric or portal veins but was found in systemic vessels, in the chambers of the heart, in the retroperitoneal, extraperitoneal and subcutaneous tissues, and in the wall of the intestine.

The origin of the gas in all cases remains a matter of speculation. One possible explanation is that the gas in the portal venous system originates in the lumen of the intestine, where it is under increased pressure in the distended small and large bowel, and finds its way into the vascular channels in the ischemic bowel wall. This explanation may be valid when there is frank necrosis of the intestinal wall and disruption of small vascular channels; however, it does not cover those cases in which there is no evidence of intestinal necrosis or ulceration. Even here, however, it is possible that, with marked intestinal distension, gas may find its way into the vascular channels through a mucosa that is congested, though not necrotic.

A second and perhaps more likely explanation is that portal vein gas is formed within the wall of the intestine by gas-producing organisms such as *E. coli* and Clostridium and then finds its way into vascular channels. Since three of the six infants described by Wolfe and Evans[1] demonstrated pneumatosis intestinalis, it is possible that a relationship exists between this apparently benign condition and portal venous gas. The nature of this relationship is not clear, although it was suggested by Wolfe and Evans that pneumatosis intestinalis may have been a possible source of gas in these three infants.

Systemic gas is presumably portal gas which has found its way through the portal venous system

into the hepatic sinuses, then into the hepatic veins, and from there into the inferior vena cava and the general systemic circulation. This hypothesis would not, however, explain gas found outside vascular channels. Septicemia due to gas-forming organisms of intestinal origin may perhaps account for this more diffuse distribution.

The pathophysiological effects of portal venous gas also remain a matter of speculation. This gas may produce obstruction to blood flow and thus produce portal hypertension, which may have been an important factor in the death of these patients. It is significant that none of these patients has survived, although in two instances the gas was detected as long as seven days before death.

An analysis of the reported cases suggests that in infants the presence of portal gas is an indication of septicemia with gas-forming organisms, and draws attention to the necessity of blood cultures and treatment with appropriate antibiotics. In addition, in the majority of adult cases, portal venous gas indicates intestinal necrosis and should lead to early operative intervention. Although no survivors have been reported to date, an early aggressive combined medical and surgical approach may yet be successful.

SUMMARY

Two cases of gas in the portal vein are described. In one patient the diagnosis was made before operation on plain roentgenograms of the abdomen; in the other the presence of portal gas was noted at operation and confirmed by postmortem films. Both patients had extensive intestinal necrosis, due in one instance to a volvulus of the small intestine around a mesenteric band and in the other to occlusion of the superior and inferior mesenteric arteries and celiac axis. Both patients died.

The literature on this subject is reviewed and a distinction is made between portal vein gas and biliary tract gas. The possible sources of portal gas are presented and the clinical significance of this entity in infants and adults is discussed.

REFERENCES

1. WOLFE, J. N. AND EVANS, W. A.: *Amer. J. Roentgen.*, **74**: 486, 1955.
2. SUSMAN, N. AND SENTURIA, H. R.: *Ibid.*, **83**: 847, 1960.
3. WIOT, J. F. AND FELSON, B.: *Ibid.*, **86**: 920, 1961.
4. SISK, P. B.: *Radiology*, **77**: 103, 1961.
5. BARRETT, A. F.: *Clin. Radiol.*, **13**: 92, 1962.
6. STEWART, J. O. R.: *Brit. Med. J.*, **1**: 1328, 1963.
7. GUYER, P. B. AND GRAINGER, K.: *Brit. J. Radiol.*, **36**: 379, 1963.
8. EDWARDS, A. M., COSTOPOULOUS, L. B. AND BELL, H. E.: *Canad. Med. Ass. J.*, **91**: 1300, 1964.
9. RIGLER, L. G. AND POGUE, W. L.: *Amer. J. Roentgen.*, **94**: 402, 1965.

PAGES OUT OF THE PAST: FROM THE JOURNAL OF FIFTY YEARS AGO

A TEN-DOLLAR BILL AND AN EGG UNDER MY ARM

Dr. N. Viner: One question I want to ask and that is with regard to the distinction between false croup and real croup, or laryngeal diphtheria. Under the ordinary measures a false croup will tend to disappear in 24 hours, but a very large number of cases of the so-called false croup may last for several days and are very severe. There may be no actual demand upon the accessory respiratory muscles but the stridor is very intense and the cough and speech are characteristic of laryngeal diphtheria. Now cases like that under the usual treatment with ipecac, and probably steaming with Friar's balsam, will improve or not at the end of two or three days, but in view of the fact that we have seen so many of these cases this winter and rather few of diphtheria, and knowing that the proportion of laryngeal diphtheria compared with diphtheria in general is only 10%, a lot of these must have been purely false croup recovering with the usual care and without antitoxine. A great many of these patients are extremely poor and if you consider that in a case of laryngeal diphtheria you want a large injection of serum which costs the people $10, and you cannot send it into hospital until you are sure it is diphtheria, the result is that you may be neglecting a very real case of croup. What is one to do in these cases where you cannot afford to give $10, where there is no improvement in 24 hours and you are not sure it is diphtheria?

Another side issue is in connection with cultures. I have never taken a real culture of diphtheria but I have taken an egg, boiled it, removed the shell and getting between the two soft layers of the outer coats, taken a smear and incubated it under my arm or in the oven. On two occasions I tried it in the oven, but it dried out and was no good but under my arm I obtained a fairly good specimen of the diphtheria bacillus.

Dr. W. E. Enright: I have had a number of cases of laryngeal diphtheria and in two especially I think the children owe their lives to the prompt and efficient measures which were taken at the Alexandra Hospital, both being cases of intubation. In one the child had been going about with slight cough, husky voice, no fever, no pharyngeal signs, suddenly cyanosis set in and about 15 minutes later on my arrival I found the child dying. I communicated with the hospital, gave him a large dose of strychnine and drove him to the hospital where prompt intubation saved his life, though six efforts were made before it was successful, artificial respiration being carried on and camphor and other stimulants given. The child is now a fine, healthy boy. Another difficult case was in a child who had frequently had croup and the one for which I was called in seemed scarcely different from the others. However, cyanosis appeared and there was considerable obstruction. Here, too, intubation saved the child's life.

I would like to ask as to the advisability, or otherwise, of giving serum in a case of tonsillitis with much exudate not extending beyond the tonsils and where the general condition is not severe. I have heard it said by a number of practitioners that serum in these cases has a very beneficial effect. I hope that some day the city will supply to the poor free serum which can be obtained on a doctor's order. On a number of occasions I have supplied the serum myself, but we do not always feel equal to that. It would be a good move on the part of this Society if some effort could be made to get serum quickly and as a matter of charity for certain patients where it could be proved they were unable to bear the expense.—Montreal Medico-Chirurgical Society, *Canad. Med. Ass. J.*, 6: 859, 1916.