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The Journal of the Allentown Hospital is published each February, May, August, and November by the Staff and Trustees of the Allentown Hospital. Original papers are invited from all members of the Staff and will be reviewed for publication by the Editorial Committee. Papers submitted must be typewritten, double-spaced, on one side of the page only. Illustrations must be original drawings or black and white glossy photographs. References should be listed alphabetically and should conform to the Quarterly Cumulative Index Medicus: author's name and initials, journal, volume, initial pages and year. Papers should be submitted to the chairman of the committee.
CONGENITAL POLYPOSIS OF THE COLON
BRYON D. WILKINS, M.D.

It is the purpose of this paper to present a case of congenital polyposis of the colon and emphasize the problems presented and mistakes made in both the diagnosis and treatment of this condition.

This condition has been generally known as multiple polyposis which, as Buie\(^1\) states, is fundamentally incorrect. On the other hand, multiple polypoid disease seems debatable as polypoid means resembling a polypus. Inasmuch as polyposis is defined as "the development of multiple polypi on a part",\(^2\) the addition of multiple to polyposis is repetitious. Therefore, I have termed this condition "congenital polyposis of the large bowel."

The term polypi has been defined as "a tumor arising from mucous membrane and attached to that mucous membrane by a pedicle." In this report the term polyp will include not only those pedunculated tumors arising from the wall of the large bowel but also those sessile tumors arising from mucous membrane which have no demonstrable pedicle.

It is generally conceded that this condition is congenital and this case supports that opinion. Erdman and Morriss\(^3\) classified all cases as either congenital or acquired. The reported observation of the development of multiple polyp-like structures in cases of chronic ulcerative colitis suggest the acquired nature of some of these tumors. Swinton and Warren\(^4\) report, however, as follows: "From a microscopic study of a large series of intestines from patients with chronic ulcerative colitis, both specimens removed surgically at varying lengths of time after onset of the disease and specimens obtained at autopsy, we believe that chronic ulcerative colitis is not a factor predisposing to the development of polyps." This opinion is concurred in by many others.

HISTORY

A thin 35-year-old female patient was first seen by me June 21' 1946 with a chief complaint of frequent loose bowel movements and pain in the right lower abdomen, together with weakness. The patient supplied the following history: As long as she could remember her stools always had been on the loose side, but for the previous four to five years had been more frequent but not in any degree incapacitating. During the previous year they had become very frequent, varying
from five to twelve a day, with visible blood and slime. She usually had one movement during the night if she got up to void. Her stools were more frequent when she was up and around, less frequent if she remained quiet in bed. Her best weight was 111 pounds, but a year before her visit it was 107 pounds. The pain in the right lower abdomen became noticeable about a year prior to her visit and was intermittent, gradually becoming more severe during the year. It became worse before and immediately following her menstrual period which was regular, not clotted, and of about a day and a half duration. It was during this year that her stools became more watery, were preceded by cramping abdominal pains associated at times with nausea and that she started losing weight.

She was treated during the previous year and a half to two years by several physicians by diet, liquid medicine and pills. She said that she never had a finger put in her rectum or had been advised to have a rectal examination. On June 12, 1946 the physician who was treating her at that time sent her for an X-ray of the stomach and on June 13, 1946 for a barium enema X-ray of the colon. The X-ray report of the stomach and duodenum were reported as X-ray negative for organic disease. The X-ray report of the colon was as follows: “X-ray of the colon reveals a hyperspastic sigmoid colon, a normal transverse portion, hyperspasticity involving the ascending colon; tenderness over the tip of the caecum on deep palpation, very suggestive of pathology in this area; chronic appendix suspected. Conclusion: Hyperspastic colitis with chronic pathology in the right lower quadrant.”

**CLINICAL FINDINGS**

The patient presented herself at my office one week after the X-rays were taken. Her temperature was 100 and her skin was sallow. Perianal examination revealed normal pigmentation with radial folds slightly more pronounced than normal. Digital examination revealed sphincter tone slightly hypertonic and a number of soft pedunculated masses, together with a feeling of generalized roughness. There was blood on the finger cot.

Proctoscopic examination revealed moderate to marked internal hemorrhoids in the left lateral, right postero-lateral and right antero-lateral areas with some smaller secondary ones. Above this area, starting at about 6 cm. from the anal verge, many polypi were noted of varying sizes, both pedunculated and sessile. A bloody mucopurulent discharge was noted.
Sigmoidoscopic examination was performed to about 18 cm. and revealed innumerable polyps ranging from minute sessile ones to pedunculated ones, 1.5 cm. in diameter. Several appeared eroded and bled easily.

Abdominal examination revealed a very flat thin abdominal wall thru which it was possible to palpate a sausage-like mass in the left lower and left lateral areas, although it caused pain. There was more marked tenderness in the right lower quadrant. Peristalsis was slightly hyperactive.

The patient was prepared with two ounces of castor oil and a double contrast X-ray of the colon was taken which revealed the following:

“A prominent sigmoid portion of the colon and many small well rounded filling defects in the proximal portion of the sigmoid colon. This same pathology is seen throughout the entire transverse portion of the colon. There is also evidence of this pathology in the ascending portion of the colon. Film taken after the evacuation of the barium and before the introduction of air again reveals numerous filling defects distributed throughout the various portions of the colon. After the introduction of air into the colon many areas of pathologic changes within the lumen were noted, indicative of polyposis.”

The patient’s condition was explained to her and an attempt was made to have her parents present themselves for examination, which they refused to do. The patient’s two children were later examined and found to have polyps. The patient was advised that one or more operations were necessary and a permanent small bowel opening on the abdomen would result. It was explained to her that due to the innumerable polyps in the rectum, some of which were broken down, it was unwise to attempt to treat them and save the rectum. As I was leaving for the American Proctologic Society meeting on the west coast, it was decided to have her enter the hospital for blood studies and possible blood transfusion before I left and then have her reenter for surgery on my return.

**LABORATORY FINDINGS**

Laboratory findings on June 22, 1946 were as follows:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb (%)</td>
<td>67%</td>
</tr>
<tr>
<td>RBC</td>
<td>3840000</td>
</tr>
<tr>
<td>WBC</td>
<td>9100</td>
</tr>
<tr>
<td>Polys</td>
<td>74</td>
</tr>
<tr>
<td>Lymphs</td>
<td>26</td>
</tr>
<tr>
<td>Serum Protein</td>
<td>7.5</td>
</tr>
<tr>
<td>A-G ratio</td>
<td>3.4/1</td>
</tr>
<tr>
<td>Plasma Chlorides</td>
<td>580</td>
</tr>
<tr>
<td>Sedimentation rate (normal 10)</td>
<td></td>
</tr>
<tr>
<td>Intake</td>
<td>1560 cc</td>
</tr>
<tr>
<td>Output</td>
<td>240 cc</td>
</tr>
</tbody>
</table>
The patient was given a low residue diet, blood transfusion of 500 cc. and multiple vitamins by mouth. She was sent home June 26, 1946.

PROGRESS OF CASE

I saw her again when I returned July 9, 1946. At that time she weighed 94 pounds and her temperature was 99. She was still having 4 to 6 bowel movements a day, although she had been spending most of her time in bed.

She was very undecided about going through with the surgical treatment advised but I refused anything less radical. As she wanted more time to consider, I saw her at weekly intervals during July. Her condition remained the same. On July 25, 1946 she weighed 98 pounds and was losing ground. She then decided to consult someone else.

The following progress of the case was obtained from the family and from hospital records.

During August, September and October of 1946 she underwent fulguration treatments of the polyps in the rectum. On October 31, 1946 she began to vomit and had more severe abdominal pains. Her bowels were moving 6 to 8 times a day. She was admitted to the hospital November 4, 1946.

After a week of preoperative preparation she had a partial colectomy on November 11, 1946, with an ileostomy. The laboratory report of the specimen was as follows: Gross:- Specimen is a piece of colon 30cm long at one end of which a normal appendix appears. The entire mucosa shows multitudinous polypoid growths throughout the entire specimen except for a few cms. at the ileocecal end. These polyps vary from 4-5 mm. to 1.5 cm. in diameter. They appear to continue at the distal end. Diagnosis:- Multiple polyposis.
Microscopic Examinations:—Section shows mucosa normally arranged with marked evidence of congestion present everywhere. Leucocytes appear among the glands. Here and there gland like sub-mucous areas are noted. They are composed of round cells, cells of mucous glands and regional polyps show irregular nuclei with polarity distorted.

Diagnosis:—Intestinal polyps with infection and beginning malignancy.

On December 6th the second stage of the colectomy was performed. This was to the recto-sigmoid which stump was inverted. The laboratory report of this specimen was as follows:

Gross Examination:—Specimen is a piece of intestine 38 cm. in length. The mucosa is everywhere studded with innumerable polyps of varying size ranging from 1.5 cm to 2.5 cm in diameter. Some have long and some have short pedicles.

Diagnosis:—Multiple polyposis.

Microscopic Examination:—Section shows tufts of cells of mucosa arranged along fibrous stalks. Cells are numerous and tufts hyperplastic. Malignant changes are not noted among these cells.

The patient was discharged from the hospital December 28, 1946, with an ileostomy. For some months she appeared to be improving and then began to fail. She expired in November 1947.

COMMENT

1. This type of condition, especially in a young person, is one of the most difficult we are called upon to treat. The seriousness, even to the probability of developing cancer, must be explained to the patient since the cure necessitates such a radical thing as permanent ileostomy and a patient is not likely to consent unless aware of the dangers involved in refusing.

2. Here again we have a case with bleeding from the rectum, diarrhoea, and abdominal pain treated for a long period of time without as much as a digital examination.

3. In a case of diarrhoea and/or bleeding from the rectum the X-ray should always be preceded by a digital examination and a sigmoidoscopic examination. In fact, any case calling for an X-ray of the colon should always be preceded by a digital and sigmoidoscopic examination.

4. X-rays of the colon can miss such conditions and are definitely misleading unless the patient is prepared properly and the double
contrast method or method of Pugh, expelling the barium and then X-raying, is used. Too often patients are sent for X-rays without any information to guide the roentgenologist.

5. In a case with innumerable polyps, especially with visible degenerative changes, it is the concensus of opinion of most men that fulguration, with the idea of saving the rectum, is contraindicated.

With the advent of the Dragstedt Operation and the Rutzen Bag, ileostomies, while not a thing to wish for, are at least much more satisfactory to live with than previously.

In this case there was hardly a spot in the rectum that wasn’t involved with a pedunculated or a small sessile polyp.

SUMMARY

A case of congenital polyposis of the colon is presented with some points on diagnosis and treatment which may well cause us to pause and reflect. It certainly proves the necessity for careful early and complete examination together with cooperation between the general practitioner, roentgenologist and proctologist.

It also points toward the advisability for more radical, early surgery in such involved cases.

REFERENCES:

(2) American Illustrated Medical Dictionary, Dorland, W. B. Saunders Co.
EMORRHAGE from esophageal varices secondary to a portal bed block is a condition which has baffled the medical profession for many years. Recent work by Whipple, Lord and Blakemore, and Phemister has stimulated a renewed interest in the subject.

The basic cause of bleeding gastro-esophageal varices is a portal hypertension resulting from a number of conditions. These conditions for a long time were all included under the term of Banti’s disease. Portal hypertension, however, has been found without any demonstrable sign in the portal, hepatic or splenic veins.

Portal hypertension may be divided into extra-hepatic and intra-hepatic factors. It varies in individuals and in accordance with the type of obstruction, and the site. Each case is different as to diagnosis, treatment and prognosis.

In the intrahepatic type of obstruction cirrhosis is found, varying in individuals with the same type and degree of cirrhosis.

The extra-hepatic type of obstruction may result from a number of lesions such as thrombophlebitis from regional or distant infections involving the splenic vein or other parts of the portal system such as the superior mesenteric vein. Omphalitis is another lesion which in early life can cause thrombi in the abdomen. This frequently is a contributing factor in the neonatal period since the organism can travel along the round ligament and get to the portal system early. A history of antecedent pancreatitis or severe trauma to the epigastrium suggest thrombosis of the splenic vein. Another factor is occlusion of the portal vein resulting from a continued obliteration of the umbilical vein and ductus venosus which may occur in the young. A final factor is a cavernomatous transformation of the portal vein, possibly by an organized thrombus with canalization, telangiectatic granulation tissue or a congenital anomalous neoplasm or angioma.

It is not always possible to diagnose the cause preoperatively or even postoperatively. It has been impossible to determine the site of the block at the time of splenectomy in over 50% of the cases.

With splenomegaly, hemorrhage, anemia, leucopenia, thrombocytopenia and normal liver studies and normal blood studies, the cause is
usually extrahepatic. If the liver function tests, such as the cephalin-
cholesterol flocculation are positive, with a high retention of bromsulfa-
lein, positive hippuric acid test and reversal of the albumin globulin
ratio, the presence of cirrhosis with intra-hepatic block is fairly certain.
The mechanism is probably a fibrous constriction on the venous
vessels, or possibly abnormal anastomoses between the hepatic artery
and the portal system. The coronary vein may enter the splenic
vein rather than the portal vein and hide the definite site of obstruction.
Esophageal varices do not usually occur in the splenic vein thromboses
when the coronary vein enters the portal vein.

Aids such as manometer readings on the various veins of the portal
system are used in determining the site of the obstruction. If the
pressure is elevated in the superior mesenteric vein, there is evidence
of block in the superior mesenteric vein, portal vein, or intra-hepatic
block. If there is a normal superior mesenteric vein pressure with an
increased coronary vein pressure there is indication of block in the
superior mesenteric vein with the coronary vein originating from the
splenic vein distal to the site of obstruction. With congestive spleno-
megaly, a normal superior mesenteric and coronary vein pressure and
an increased splenic vein pressure, one is inclined to do a splenectomy
only. Diodrast venograms may be used at the time of splenectomy.

**Anatomy**

Since a comprehensive knowledge of the portal system is imperative
a review of the anatomy will be discussed.

It is formed by the portal vein, superior mesenteric vein, inferior
mesenteric vein and their tributaries from the whole of the abdominal
and pelvic parts of the alimentary canal except the anal (terminal)
canal, pancreas, spleen, gall bladder and the paraumbilical vein which
passes through the round ligament of the liver. The inferior mesenteric
vein joins the splenic vein which joins the superior mesenteric vein to
form the portal vein. The portal vein is three inches long and is behind
the pancreas as well as left and anterior to the inferior vena cava. It
travels up into the lesser peritoneal sac, enters the lesser omentum,
leads up behind the common bile duct and hepatic duct and to the right
end of the porta hepatis and ends by dividing into a right and left
branch which enter the liver. The right branch receives the cystic
vein. The right and left divide with every branching of the hepatic
artery and bile ducts and the three run through the liver substance
forming the central, sublobular veins and pass to form the hepatic vein
which empties into the inferior vena cava. The round ligament passes
from the umbilicus to the left branch of the portal vein (the remnants of the fetal umbilical vein).

The paraumbilical veins accompany the round ligament and connect the left branch of the portal vein with superficial veins about the umbilicus. The ligamentum venosum connects the left branch of the portal vein with the upper inferior vena cava. It is the remnant of the fetal ductus venosus where the blood from the placenta went by the left umbilical vein to the inferior vena cava without going to the liver. Tributaries such as the left gastric vein cover both stomach surfaces. The left gastric vein travels along the lesser curvature to the esophagus from which it receives tributaries. The right gastric vein supplies the upper pyloric area and travels along the lesser curvature to the portal vein. The cystic vein travels to the right portal vein. The superior mesenteric vein begins in the right iliac fossa and ends behind the pancreas where it unites with the splenic vein. The communications between the portal and systemic systems should be noted: in the lower esophagus, branches of the left gastric and esophageal veins which join the azygos system; the rectal plexus veins, between the superior rectal vein (Sigmoid) and middle and inferior veins going to the internal iliac veins (here hemorrhoids occur); the communications of the superficial veins about the umbilicus and the paraumbilical veins which course to the left branch of the portal vein along the round ligament. Obstruction of blood through the liver results in enlarged paraumbilical veins giving a caput medussa.

The portal vein carries approximately 75% of the blood emptying into the liver and carries the nutrient material from the gastrointestinal tract and insulin from the pancreas to be altered, detoxified and utilized by the liver. It carries very little oxygen. The normal pressure in the portal vein is 60 to 104 mm. of water. A sudden complete portal obstruction results in death or atrophy of the parts draining into it. A gradual occlusion may be satisfactory.

**Pathology**

The pathology found is various. The varices in the lower esophagus may be demonstrated by X-ray. In the stomach, the coronary vein on the lesser curvature is enlarged. Evidence may be found in the form of hemorrhoids or the caput medussa at the umbilicus. In the stomach, the mucosa may have a few petechiae. Microscopically large submucosal dilated veins especially on the lesser curvature near the esophagus may be seen. The bleeding might occur from minute surface erosions connected with the submucosa by capillaries. The absence of
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gross gastric pathology frequently explains the lack of emphasis on
gastric hemorrhage associated with esophageal hemorrhage. The
probable sources of bleeding are through tiny ruptures in the veno
capillary stomata, although in cirrhosis and Banti's disease, most
bleeding is esophageal.

The spleen is enlarged. Microscopically, fibrosis, thickened capsule,
vascular adhesions and dilated tortuous veins are found. The spleen
decreases in size during or immediately after a hemorrhage and may
not be palpable. In the ensuing few days, it increases in size and stays
so until another hemorrhage occurs, the bleeding temporarily decreasing
the portal engorgement and the spleen decreasing in size. Typical
findings of cirrhosis may be evident in the liver.

Treatment

Various methods of treatment are being used for the relief of portal
hypertension and congestive or primary splenomegaly. In the poor
risk patients, extensive procedures cannot be tolerated even though
they would be the most effective. The operation of choice in these
poor risk patients is ligation of the splenic vein, the artery being ligated
twice through the gastro-colic omentum in the lesser peritoneal cavity,
and on the superior border of the pancreas. Splenectomy has been and
may be used with best results in splenic vein thromboses, schistosomi-
asis and early Banti's Disease. Approximately 20% of the blood of the
portal bed is removed which may ameliorate for a variable period of
from 12 to 25 years before recurrence or signs of cirrhosis develop. The
blood picture and the symptoms improve greatly. If done early before
esophageal varices occur, bleeding would be prevented in a large per-
centage of cases. Patients with splenectomy with no gross hemorrhage
before operation had 30% less bleeding than those who had gross hem-
orrhages. Esophageal varices do not usually occur in splenic vein
thrombi where the coronary vein enters the portal vein. Therefore very
good results occur with splenectomy. If the coronary vein is distal
to the thrombus it is satisfactory. If it is not, a splenorenal shunt should
be performed. In late Banti's disease with splenomegaly and cirrhosis,
splenectomy is useful in relieving abdominal discomfort with decrease
to some extent of esophageal congestion. It may relieve ascites re-
accumulation by decreasing congestion in the portal system. It is
best to combine omentopexy with division of the esophageal branches
of the coronary vein. One may get relief for a number of years. In
Banti's disease with a normal liver and frequent post operative hemorr-
hages, extensive obliteration with sodium morrhuate may give much
relief with fewer hemorrhages. Other cases of Banti's disease with a normal liver and splenectomy may still have severe hemorrhages especially from the stomach. Here an esophago-gastric resection may result in much decreased bleeding.

Postoperative hazards such as bleeding from the operative site, increasing platelets and a thrombosis may occur the first week postoperatively. Here heparin would be indicated. A splenectomy should not be done when prepared to do a spleno-renal anastomosis.

The procedure of omentopexy has been used whereby the omentum is placed in contact with an abraded liver surface or by suturing the omentum in contact with the split rectus muscle. Its efficacy is questionable.

Sclerosing solutions such as sodium morrhuate by repeated injection to obliterate the varices is also used. This throws more load on the remaining routes of the collateral circulation. It may be justified because they do not bleed or bleed infrequently. It does little good if gastric varices are present at the same time. Moersch of the Mayo Clinic has had 50% of the cases with no further bleeding during a four to six years follow-up. Follow-up esophagoscopy examinations reveal the lining to be thickened. Bleeding, if it occurs, may pass by the rectum rather than by emesis. Its best use probably is in follow-up splenectomies which continue to bleed with no gastric varices present.

An additional measure to consider when splenectomy and sclerosing solutions fail or if for other reasons they cannot be supplied, is resection of the bleeding segment. This is especially true since transthoracic esophago-gastric resections and total gastrectomy yield a mortality of less than 15% and at least a fair state of health can be maintained with improvement of nutrition and only a mild anemia. This eliminates that portion of varicose veins, divides the vessels of the lesser curvature and variable parts of the greater curvature. Therefore a decreased gastric blood supply and a more or less barrier in the esophago-gastric venous channels at the line of anastomosis is established.

When a total gastrectomy, esophago-jejunostomy, jejuno-jejunostomy is performed, one gets rid of the bleeding points when the hemorrhage is predominantly from the stomach and establishes a connection between the esophagus and jejunum free of varices. Therefore resection would appear worthy of trial when control of hemorrhage by splenectomy, porto-caval shunt, or injection fails or if the portocaval shunt on exploration is found to be impossible to perform. Blakemore states that at the site of the anastomosis, the venous drainages are different but yet will inosculate with inevitable subsequent varices.
Phemister feels that it is so but that they rarely if ever bleed. It should be remembered that all the operative procedures are directed at combating the hemorrhage and possibly preventing further cirrhosis or delaying it. They are not a cure.

In the performance of a spleno-renal anastomosis, the splenectomy which is performed eliminates 20 to 40% of the total circulating blood volume. The shunt is capable of handling a large volume of blood, decreasing the tendency of gastro-intestinal hemorrhage and decreasing or preventing the formation of ascites. An end-to-end anastomosis, sacrificing the left kidney, or an end-to-side anastomosis without sacrificing the kidney is preferable. The full length of the splenic vein should be preserved during the splenectomy with minimum trauma. The spleen is mobilized by cutting through the gastrocolic and phrenicocolic ligaments and the tail of the pancreas is separated from the spleen pedicle permitting freedom of movement for better examination of the splenic vein. The splenic vein is ligated at its branching close to the spleen. The kidney is mobilized and the renal artery clamped by applying the vitallium tube without twisting the anastomosis. With the vitallium tube an intima-to-intima anastomosis is obtained. Anastomosing the end of the splenic vein to the side of the renal vein is better than to the end of the renal vein; it is more likely to remain open due to the difference of the pressure gradient. Suturing is extremely difficult to do perfectly. One of two misplaced sutures may ruin the operation. The time factor is important here also since serious kidney damage may result with prolonged blockage of the vein. The longest blockage usually is thirty-five minutes.

The results are so outstanding as to justify continuation of the operation. The mortality thus far by the surgeons previously mentioned has been close to zero. None have bled from the gastro-intestinal tract except one who had a Levine tube inserted by mistake. This operation is used mainly in cases with splenic vein thromboses with the coronary artery distal to the thrombus. It is used also in cases with cavernomatous transformation or atresia of the portal vein.

Nicolai Eck, a Russian physiologist, first successfully performed an anastomosis of the portal vein and the inferior vena cava in 1877. A porto-caval anastomosis is ideal in a number of ways. It reduces the elevated pressure and decreases the tendency to hemorrhage of the varices. It relieves the passive congestion of all structures drained by the portal system. A decreased tendency toward ascites formation results. There is a better chance for the anastomosis to remain open. The porto-caval anastomosis is impossible to perform in some cases with fibrous or cavernomatous transformation. It is better than the
spleno-renal anastomosis because of the high portal pressure. It also conducts more blood.

This type procedure is used especially with cirrhosis. The end-to-side type of anastomosis is superior to the side-to-side. The end of the portal vein is anastomosed to the side of the inferior vena cava. It is easier and need not occlude the inferior vena cava completely and is more likely to remain open while the side-to-side promptly closes.

The technique: The portal vein is slightly posterior and medial to the common duct. It is mobilized from the point of bifurcation at the liver to the origin of the splenic vein. A spot on the inferior vena cava is selected with no angulation or compression. The inferior vena cava is mobilized down to the entrance of the left renal vein. The portal vein is clamped, cut and irrigated with saline and the end is cuffed over a proper sized vitallium tube if this type of procedure is decided upon. Two separate purse string sutures, deknatel No. 3, well vaselinized, are passed through the inferior vena cava. A partial or complete occlusion is performed. A cruciate incision is made and the vitallium tube is placed in the opening with the purse string sutures tied about the cuff of the vitallium tube. A resulting mucosa-to-mucosa anastomosis is thus obtained.

The operation is quite difficult and requires one who has excelled in anatomical knowledge and who is familiar with the field. It requires a team. The combatment of anemia, shock, hypoproteinemia and improvement of the blood clotting mechanism has yielded good results, with avoidance of thromboses. A marked improvement in liver function, and disappearance of ascites or hemorrhage has resulted. The suturing type of anastomosis is believed superior to the use of the vitallium tube type.

Disadvantages are also present. Shunting the blood from the liver with its nutritive factors may aid in further degeneration.

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6. Gray’s Anatomy
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8. Blair & Blair—Ligation of Splenic Artery, the operation of choice in selected cases of Portal Hypertension and Banti’s Syndrome. Annals of Surgery January, 1950
MYOCARDIAL infarction, not unlike many other clinical entities in medicine, often presents a picture which is so completely atypical and lacking in the fundamental requisites for early diagnosis and, therefore, effective treatment, as to try the most intuitive of diagnostic capabilities.

The purpose of this paper, therefore, is to point up the important necessity of increased awareness in this regard in order to prevent complacency in making a diagnosis that excludes infarction, especially in patients whose age and clinical condition presupposes its possible existence, or in whom the necessary predisposing factors are present. Furthermore, both the frequency of coronary accidents in the general population as it now exists, and the fact that increasing thousands of people will be enjoying a greater longevity in the next several decades, make the issue of prompt diagnosis paramount. The total cardiac mortality in 1940 was 292.5/100,000. Of this 25 per cent was attributable to coronary disease, most of these representing infarction and its consequent complications. Another 46 percent was attributed to chronic myocarditis and myocardial degeneration which undoubtedly contains a large per cent of acute and chronic coronary thrombosis. Master estimates that 800,000 attacks of coronary thrombosis occur annually in the U. S., which means that after the age of 40 years one man in 38 and one woman in 115 sustains a coronary occlusion each year.

Prior to discussing necrosis of the myocardium it is necessary to define the several terms: coronary thrombosis, acute coronary occlusion, and myocardial infarction. They are often incorrectly used synonymously, and their correct usage relates to the following discussion.

Coronary Thrombosis refers to an acute thrombotic occlusion of a coronary artery. It is almost always associated with and is an intrinsic stage of the process of arteriosclerosis.

Coronary Occlusion denotes the sudden obstruction of a coronary artery either by thrombus, by intimal hemorrhage with swelling of the arterial wall, or by embolus.

Myocardial Infarction signifies the necrosis or death of a portion of heart muscle because of an interruption or curtailment of its blood
Although acute coronary occlusion is the usual cause, infarction can also follow a drastic reduction in blood volume or oxygen content of coronary blood, as can exist in many circulatory or hematologic diseases.

We are not concerned in this presentation with the distinctive clinical picture of coronary thrombosis characterized usually by pain in the chest with its usual location, radiation, severity and duration; its independence of exertion; its association with shock, acute cardiac failure, fever, leukocytosis, and the typical E.C.G. changes.

We are concerned with cardiac infarction in its more unusual aspects, those cases that present a greater clinical challenge and call for a higher index of suspicion on the part of all of us. In other words, cardiac infarction that may be masked:

1. By the signs and symptoms of the underlying disease state which by virtue of its secondary effects on the dynamics of the coronary circulation leads to infarction.
2. By co-existing, but not necessarily related disease states, that may overshadow the presence of infarction.
3. By a cardiac disturbance which initiates the clinical picture and eventually, belatedly betrays itself through typical E.C.G. patterns.

If we are to sharpen our diagnostic acumen in this regard, it would seem clear, from the definitions mentioned, that the terms “acute coronary occlusion” and “myocardial infarction” are not interchangeable. Moreover, they are not invariably associated. Infarction need not ensue, following coronary occlusion, and conversely, infarction can and does develop in the absence of acute coronary occlusion. Friedberg and Horn found, in a series of 1,000 consecutive autopsies, that 31 per cent of the cases of acute myocardial infarction were not associated with a recent coronary artery occlusion. In 37 such cases, pulmonary embolism was associated in 12; calcific aortic valvular stenosis in 8; severe coronary and old occlusions with congestive failure in 6; an operative procedure in 4; a severe acute anemia in 3; malignant hypertension with a cerebral complication in 3; and occlusion of the superior mesenteric artery in one.

Therefore, it would seem wise to presume that myocardial infarction may occur whenever prolonged myocardial anoxia exists, whether due to mechanical occlusion of a coronary artery, or, to any disturbance in circulatory dynamics, such as shock-like states following surgical procedures, mesenteric thrombosis, massive pulmonary embolism, and, acute hemorrhage, all of which cause a fall in aortic blood pressure
and therefore in coronary blood pressure, leading to a decrease in coronary blood flow. The result, in either case, is coronary insufficiency.

The following cases are presented in the hope that they will illustrate the three categories of hidden myocardial infarction previously mentioned.

1. That cardiac infarction may be masked by the signs and symptoms of an underlying disease state which, by virtue of its secondary effects on the dynamics of the coronary circulation, may result in infarction. Parenthetically, it may be added that statistically hypertension is the most common condition associated with acute coronary occlusion. Levine and Brown, Master, Dack, and Jaffe, and Palmer found hypertension in one-third to three-quarters of the cases of coronary occlusion. Next to hypertension is diabetes mellitus. Other contributory clinical conditions are: familial hypercholesterolemia with xanthomatosis, myxedema, polycythemia, and diseases of the biliary tract.

The case is that of L.G., a 46 year old white male who, when first seen presented a complaint of an ache in the region of the Angle of Louis's radiation. He was ambulatory, without apparent acute distress. He had continued at his occupation for two hours after the onset of the ache. At the time, he complained of lassitude, some recent weight gain, and some dryness of hair and skin. An E.C.G. one year previously, because of a similar complaint of aching substernally, was completely normal. B.P. previously had been 138/94. An E.C.G. was done. It showed a typical acute posterior occlusion. Sedimentation rate was 12 mm./hr. (Westergren). B.P. was 146/98. Blood cholesterol was 216 mgm. per cent. The patient was given routine care without dicoumarolization. The sedimentation rate rose to 35 mm./hr. after 9 days, and gradually receded to normal in succeeding weeks. Upon recovery, a B.M.R. was done and was minus 33 per cent. Further blood cholesterol determinations were 349 mgm. percent, 370, 383. He was placed on thyroid extract gr. one-quarter t.i.d. He was allowed to resume his work, although he occasionally complained of substernal discomfort of short duration. The blood cholesterol was 263 mgm. per cent. Choline therapy was initiated. After four months the patient had no further substernal discomfort and has remained well to date. A subsequent B.M.R. was minus 19 per cent. As expected, the clinical symptoms of mild myxedema were alleviated.
II. That Cardiac Infarction may be masked by co-existing or concomitant, but not necessarily related, disease states that may overshadow the presence of an infarction.

Again, probably most common among these are infections of all kinds, and in any location, pneumonias, pyelitis, etc. The elevation of blood fibrinogen and the occurrence of cold agglutinins may favor the occurrence of thrombosis. Trauma to the chest wall also may produce pericardial and myocardial contusion with resultant myocardial infarction. Post-operative infarction may be hidden in the welter of confusing signs that follow in the first 72 hours. It has been pointed out that 5.6 per cent of attacks occur post-operatively.

The second case is that of E.C., a 68 year old white male with a history of long standing hypertension, with recent occasional angina of effort. On June 20, 1950 he felt a little dizzy, had some vague precordial discomfort which was also felt in the back and shoulders. It was more of an ache than a real pain. He breathed heavily. B.P. was 200/100, perhaps a little higher than it had been before. Temperature was 101.4°. W.B.C. was 11,500 with 78 per cent polys. Sedimentation rate was 54 mm. (Westergren). He complained of dysuria with increased frequency of urination and cloudy urine. A urinalysis revealed a light cloud of albumin with many pus cells. E.C.G. showed only the expected left ventricular hypertrophy.

The pyuria was treated with penicillin and sulfonamides and a tentative diagnosis was made of possible myocardial infarction. In the next 24 hours, severe frank hematuria developed, as did frequent premature ventricular contractions. The sedimentation rate rose to 100 mm./hr. The B.P. fell to 160/88. The patient was given papaverine HCL, and quinidine SO$_4$ to control the arrhythmia. The pyuria and hematuria slowly abated and the temperature became normal in the three days that followed. It was not until eight days had passed that a deepening of the T waves in the lateral precordial V leads was seen. This confirmed the original suspicion of infarction of the myocardium.

Subsequent E.C.G. revealed T wave inversions in all the precordial leads, and a pronounced Q wave in aVF, which had not previously been present. The sedimentation rate made a usual progression toward normalcy. The B.P. remained in the neighborhood of 160/85 to 140/80 for approximately six weeks when it rose to the pre-infarction level.

A prostatic smear done after recovery revealed a chronic prostatitis. The patient had not been dicoumarolized due to the hematuria, al-
though, the latter may have represented an embolic phenomenon to the kidney. The patient subsequently made a good clinical recovery.

III. That Cardiac Infarction may be masked by a cardiac disturbance which initiates the clinical picture and eventually belatedly betrays itself thru typical E.C.G. patterns.

It is common knowledge that the presence of any of the arrhythmias, supraventricular or ventricular, may cause the trail of myocardial infarction to be obscured, unless suspicion leads one to do serial electrocardiograms. The following case is presented to illustrate this fact.

E.L., a 72 year old white female, complained of rapid heart action which she had never before experienced. She was in no apparent great discomfort, but complained of having perspired a good deal at about 5 a.m. and of having had substernal discomfort two hours prior to being seen. An E.C.G. revealed auricular fibrillation. Her B.P. was 104/60. Her previous B.P. was unknown. She was given I.V. aminophyllin and also given quinidine by mouth. Digitalis was withheld because of the possibility of infarction of the myocardium. Temperature remained normal throughout the course of her illness. At no time was the sedimentation rate elevated. An E.C.G., taken 1 to 2 hours after the initial one, revealed a normal sinus rhythm with an occasional premature ventricular contraction. The precordial T waves were upright, but low. There was slight depression of the S-T segments in the lateral precordial leads. There was slight upward bowing of the S-T segment in aVR. The E.C.G. at this time was non-committal with regard to infarction. At no time following the onset was precordial discomfort noticed. Ten days after the onset, the T waves in the lead V5 and V6 became inverted, substantiating the diagnosis of cardiac infarction. Dicoumarol was started and maintained for several days but discontinued due to lack of cooperation of the patient in taking the blood samples for prothrombin time determination. She made a good clinical recovery with no further evidence of arrhythmia. Her pulse was 60 and B.P. rose to 160/104. Due to the slow pulse and a history of thyroidectomy, a blood cholesteral was done. This was well within normal limits. The patient refused a B.M.R.

Summary

In summary, it has been shown that infarction of the myocardium often occurs atypically, and that its signs and symptoms may be masked by (1) The signs and symptoms of the underlying disease state; (2) The signs of co-existing, but not necessarily related, disease states;
(3) The signs of cardiac disturbance, such as arrhythmias, which may initiate the clinical picture. Cases have been presented to illustrate these principles.

It is hoped that this presentation will result in greater awareness on the part of all physicians in this regard, especially because of the rapid increase in number of the aged members of the population.

BIBLIOGRAPHY:

HOSPITAL SERVICE REPORT

For the Quarter Ending November 30, 1950.

Operating services were rendered as follows:

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<tr>
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Total Medical Admissions | 1960 |
Total Births             | 578  |
A thirty-two year old white female was admitted to the plastic service on September 6 with a complaint that four months previously she became aware of a small pea sized mass on the left side of her nose in the region of the inner canthus. This mass was never painful nor did it ever become inflamed or ulcerated. She had consulted a physician only because of the cosmetic improvement to be gained and because of a cancer-phobia. She thought that the mass had increased somewhat in size since its onset but that this increase was very difficult to measure. She admitted that it might have been present for longer than four months.

**PHYSICAL EXAMINATION**

The patient was a well developed white female of Italian extraction, in no distress or discomfort and essentially normal in all respects except for a small 1 cm. mass on the left side of the root of the nose. This mass was not tender or inflamed. It was moderately freely movable and was not attached to overlying skin or to the underlying nasal bone. It seemed to have the consistency of cartilage.

**PAST MEDICAL HISTORY**

Her past medical history revealed no serious illnesses and her only operation was a rhinoplasty performed two years previously. She had been perfectly satisfied with the result and had no further difficulties with her nose until the onset of her current complaint, four months previously.

**X-RAY**

An X-Ray of the facial bones and nose was taken on the day of admission and this was reported as follows:

"X-Ray examination of the nasal bones and of the bones in and around the nasal area failed to show any X-Ray evidence of anomalies or pathologies."

**LABORATORY**

Laboratory data revealed a negative Wasserman, Kahn and Mazzini. Urinalysis was negative. Hemoglobin was reported as 83%, RBC
5,010,000, WBC 7500, polymorphonuclearcytes 70, lymphocytes 26, monocytes 2, eosinophiles 1, basophiles 1. Her coagulation time was two minutes.

OPERATION

On September 7, under local procaine anesthesia (1% with 10 drops of adrenalin to each ounce), a transverse incision one cm. long was made over the mass. The mass was dissected free from the neighboring structures with some difficulty. It was attached to the deeper layers of tissue in that area but was located above the periosteum of the nasal bones. It was removed intact and the wound was closed in the usual manner and a pressure dressing applied.

PATHOLOGY

The mass was examined in detail and found to be approximately \( \frac{1}{2} \) cm. in diameter, soft but not cystic in consistency and yellowish-white in color. The gross description by the pathologist read as follows:

"Specimen is a small cyst \( \frac{3}{8} \) of a cm. in diameter. It contains a small amount of thick mucous. It has a slightly yellow tinge and resembles vaseline.

The microscopic diagnosis:

On sectioning, a sac-like formation is noted. Lining is composed of flattened cells. Main part of tissue consists of fibrous cells. There is no evidence of inflammation or malignant changes. Diagnosis: Fibrous cyst of nose (benign)."

DISCUSSION

This case has caused some thought by those to whom it has been shown because of the fact that the mass proved to be a collection of vaseline above the nasal bones and beneath the skin, making its appearance or being noted by the patient some twenty months after a rhinoplasty. The operation performed to correct her nasal deformity was of the more usual type. An incision was made between the upper and lower lateral cartilages, under topical cocaine and local novacaine anesthesia. Using the scalpel and periosteal elevator, the periosteum was elevated from the nasal bones and the perichondrium from the upper lateral cartilages. The dorsal hump was removed with a saw and then detached from the cartilaginous septum with a blunt-edged knife. The frontal processes were infractured by the use of the angular saw inserted through a small vestibular incision with the saw engaged subperiostially under an elevator. The nasal tip was also altered but the details of this are unimportant here. Following all corrective
measures, both nostrils were packed with vaseline gauze and a Safian clamp applied. It is the usual practice to use three pieces of vaseline gauze in the post-rhinoplasty packing, each piece measuring approximately 3 by 8 inches. One piece is placed in each nostril and the third is placed in both, forming a bridge across the columella.

It is interesting to conjecture the probable pathway of a small collection of vaseline in coming to lie opposite the inner canthus. A likely possibility would be through inadvertent perforations of the mucous membrane at the tip of the saw during the sawing through of the frontal processes or through direct communication of the nasal cavity and skin as a result of separation of the nasal bone from the bony septum by means of an osteotome. Because of the location of this mass, both possibilities must be considered.

Whatever its pathway, vaseline packing in this case did not prove to be as innocuous as it is commonly regarded.

PREVENTION

Vaseline packing should not be placed in the region of the nasal bones. This is not necessary because of the hemostasis produced by the application of a clamp which automatically compresses both nasal bones.

STAFF MEETINGS

Auditorium of The Nurses Home

FEBRUARY 15 .................. Otolaryngology Department
MARCH 15 ...................... Pediatrics Department
APRIL 19 ....................... Surgical Department