Mallory-Weiss Syndrome:

Review of 20 Cases and Literature Review

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IN 1929 Mallory and Weiss [24] described a syndrome consisting of an alcoholic debauch, followed by persistent nausea, vomiting and retching, followed in turn by massive upper gastro-intestinal hemorrhage. In four autopsies they found longitudinal lacerations of the mucosa and submucosa at the esophagogastric junction to be the source of hemorrhage. In the original and a later report,[36] the same authors presented 15 patients with clinical histories of this syndrome but without surgical or pathologic confirmation. Nothing further relevant to this syndrome appeared in the literature until 1952 when Palmer [28] reported seven clinically suspected cases among 121 patients with gastro-intestinal bleeding. In 1953 Decker et al.[9] reported 11 autopsied cases.

The surgical significance of this syndrome became apparent in 1955 when Whiting and Barron [37] reported the first patient diagnosed and successfully treated by operation. Although Decker et al.[9] suggested that the lacerations should be visible with the gastroscope, the first endoscopic observation of the lesion by Hardy [17] was not reported until 1956. To date the medical literature contains reports of 101 cases of Mallory-Weiss syndrome in which the diagnosis was proved by endoscopy, operation or autopsy (Table 1).

In 1956 Scott and Newton [31] reported the first Mallory-Weiss laceration from Brooke General Hospital. Within the past 3½ years 20 patients with this diagnosis were admitted to the surgical or the gastroenterology services of this hospital. This apparent increase in incidence probably reflects only increased awareness of the lesion and diligent efforts to make the diagnosis.

Clinical Material and Management

This is a study of 20 patients admitted to Brooke General Hospital with the diagnosis of Mallory-Weiss syndrome between February 1962 and July 1965 (Table 2). Six were active-duty military personnel and 14 were either retired military personnel or veterans. The policy at this hospital is to admit to the general surgery service those patients with upper gastro-intestinal bleeding who are in shock or who have fresh blood in their stomachs at arrival in the emergency room, whereas all other patients with gastro-intestinal bleeding go to the gastroenterology section.

All patients with upper gastro-intestinal hemorrhage undergo the diagnostic procedures described by Palmer.[28, 30] On the surgical service two large intravenous cannulae are inserted; recently one of these is also used as a central venous pressure monitor. The stomach is emptied by vigorous lavage with iced saline, and blood lost by hemorrhage is replaced as rapidly as

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TABLE I. *Mallory-Weiss Syndrome: Review of Literature*

Reference	Num-of Cases	Age	Fe-male	Male	Diagnostic Method	Etiology of Tear	Treatment	Result	Other
Mallory, Weiss,[24] 1929	4	35, 31, 56, 61	1	4	Autopsy—4	Vomiting—4 After alc.—4	Conservat.—4		Fatty metamorphosis liver—1 Acute toxic hepatitis—1 Prior GI hemorrhage—2
Weiss, Mallory,[36] 1932	2	30, 44		2	Autopsy—2	Vomiting—2 After alc.—2	Conservat.—1 Neg. surg. explor.—1		Laceration ruptured with mediastinitis in 1 Prior GI hemorrhage—1
Decker *et al.*,[9] 1953	11	52, 50, 80, 83, 68, 65, 50, 75, 69, 44, 57	1	10	Autopsy—11	Vomiting—11 After alc.—1	Conservat.—10 Neg. surg. explor.—1		Cerebral thrombosis—1 Perforated ulcer—1 Pancreatitis—1 Cholecystitis—3 Prior GI hemorrhage—1 Cirrhosis—3 Alcoholism—4 Chronic gastritis—8
Kellogg, Black-burn,[21] 1954	3	72, 42, 64		3	Autopsy—3	Vomiting—3	Conservat.—3		Acute myocardial infarct—1 Renal cell carcinoma—1 Rupt. abd. aortic aneurysm—1 2 did not have hematem.
Whiting, Barron,[37] 1955	1	79	1		Surgery	Vomiting	Surgery, oversew	Recovery, cereb. thromb. postop.	Histus hernia Pyloric stenosis
Fleischner,[13] 1956	1	54	1		Autopsy	Vomiting	Neg. surg. explor.		Hiatus hernia
Hardy,[17] 1956	1	21		1	G-scope	Vomiting after alc.	Conservat.	Recovery	Duodenal ulcer
Small, Ellis,[33] 1958	1	44		1	Surgery	Vomiting	Surgery, oversew	Recovery	Postop. hemorrhoidectomy
Kelley,[20] 1958	1	32		1	Surgery	Vomiting after alc.	Surgery-subtot. gastrect. and oversew	Recovery	Acute & chronic gastritis, E-G scope neg. Quest. if tear was due to scope
Scott, Newton,[31] 1958	1	26		1	Surgery	Vomiting after alc.	Surgery, oversew	Recovery	E-scope normal. G-scope not used.
Shuttle-worth, Hutt,[32] 1958	1	53	1		Surgery	Vomiting	Subtotal gastrect. then total gastrect. for uncontrolled bleeding	Recovery	Prior GI hemorrhage. Lacerations found in surg. specimen. M-A balloon failed. Gastritis.
McPhed-ran,[25] 1958	1	40		1	Surgery	Vomiting	Surgery, oversew opened esophagus	Recovery	Alcoholic, Blakemore tube failed
Mishkel, Jeremy,[27] 1960	1	68		1	Autopsy	Vomiting	Conservat.		Myocardial infarction
Etheredge,[12] 1960	2	33, 83		2	Surgery—2	Vomiting	Surgery, oversew—2	Recovery—1 Death—1	Prior GI hemorrhage—1 Migraine headache—1 Emotional upset—1 Sengstaken tube failed—1

TABLE I.—(*Continued*)

Reference	Number of Cases	Age	Female	Male	Diagnostic Method	Etiology of Tear	Treatment	Result	Other
Stahlgren, Ling,[35] 1960	1	50		1	Surgery	Vomiting after alc.	Surgery, oversew	Recovery	Alcoholic, Sengstaken tube failed
Abrams,[1] 1960	1	75		1	Autopsy	Vomiting	Conservat.		Alcoholic; considerable coughing
Labram,[22] 1960	1	82, 55		2	Autopsy—2	No vomit.			
Case Records,[7] MGH, 1961	1	61		1	Surgery	Vomiting	1st oper.—drained pancr. Source of bleeding found in hiatus hernia at 2nd oper.	Death	Histus hernia. Fatty liver. E-scope normal. Acute hemorr. pancreatitis. Esophagitis. Cholelithiasis.
Glotzer, Elias,[15] 1961	1	55	1		Surgery	Vomiting	Surgery, oversew	Recovery	
Palmer,[29] 1961	2	Not given	2		E-G Scope	Vomiting	Conservat.—2	Recovery—2	Pregnant—2
Zeifer,[38] 1961	1	50		1	Surgery	Vomiting	Surgery-opened esophagus and oversew	Recovery	Alcoholic, Sengstaken failed
Hollender, Adloff,[18] 1961	1	Not given		1	Surgery	Vomiting	Surgery, oversew	Recovery	
Atkinson *et al.*,[2] 1961	11	74, 83, 55, 49, 66, 59 74, 72, 75, 84, 75	5	6	Autopsy—7 Surgery—2 G-scope—2	Vomiting—6 Coughing—1 Epilepsy—1 Status asthmaticus—1 Undetermined—2	Surgery, oversew—2 Conservat.—2	Recovery—4 Death—7	Hiatus hernia—4 Myocardial infarction—1 Pancreatitis—1 Duodenal ulcer—1 Mucosal atrophy—1
Boyce,[4] 1962	1	70	1		G-scope	Blunt trauma	Surgery, oversew	Recovery	Hiatus hernia
Smith,[34] 1962	1	55		1	E-G scope	Coughing and retching	Conservat.	Recovery	Hiatus hernia Cricopharyngeal spasm
Lion-Cachet,[23] 1962	1	38	1		Surgery	Vomiting	Surgery, subtotal gastrect. and oversew	Recovery	Gastric ulcer
Dobbins,[11] 1963	3	82, 55, 35		3	Surgery—2 G-scope—1	Vomiting—3 After alc.—1	Surgery—hiatus herniorrhaphy, vagotomy, pyloroplasty, oversew—2 Conservat.—1	Recovery—3	Hiatus hernia—3 E-scope negative—2 G-scope negative—1 Alcoholic—1
Baue,[3] 1963	3	74, 49, 34	1	2	Surgery—3	Vomiting—3 After alc.—1	Surgery, oversew—3 Distal gastrect.—2	Recovery—3	Hiatus hernia—1 Gastritis—3

TABLE I.—(*Continued*)

Reference	Number of Cases	Age	Female	Male	Diagnostic Method	Etiology of Tear	Treatment	Result	Other
Freeark *et al.*,[14] 1964	13	64, 67, 68, 45, 39, 53, 48, 50, 38, 51, 34, 33, 26	2	11	Surgery—12 Autopsy—1	Vomiting—13 After alc.—10	Surgery—13 Oversew—12 with vagotomy—5 with antrectomy—1 Subtotal gastrect.—1	Recovery—7 Death—6	Sengstaken-Blakemore tube failed—2 Hepatic failure—2 Delirium Tremens—1 Gastrotomy leak—2
Grimes,[16] 1964	11	42, 41, 58, 78, 38, 49, 54, 23, 36, 47, 42	2	9	E-scope—2 Surgery—4 Autopsy—5	Not given—7 Vomiting—4	Surgery—7 Oversew—5 Blind gastrect.—1 with later oversew Laparotomy—1 Conservat.—4	Recovery—6 Death—5	Pancreatitis—5 Duodenal ulcer—4 Pyloric hypertrophy—1 Several alcoholics
Meyerowitz, Rosenthal,[26] 1964	1	38		1	Surgery	Vomiting after alc.	Surgery, oversew	Death—postop.	
Byrne, Moran,[6] 1965	3	56, 44, 81	1	2	G-scope—1 Surgery—2	Vomiting—3 After alc.—1	Surgery, oversew	Recovery—3	Gastric sarcoma—1
Degradi *et al.*,[10] 1965	12	Not given	Not given		G-scope—12	Not given	Conservat.—8 Surgery—4	Recovery	Hiatus hernia—12 Many alcoholics
Present series, 1965	20	17, 42, 36, 61, 46, 47, 56, 38, 68, 45, 44, 44, 38, 33, 73, 71, 57, 36, 53, 53		20	E-G scope—17 Surgery—2 Autopsy—1	Vomiting—20 After alc.—16	Surgery, oversew—8 Conservat.—12	Recovery—17 Death—3	Hiatus hernia—4 Cirrhosis—4 Alcoholic—6 Cholecystitis—1 Prior GI hemorrhage—8 Sengstaken tube failed—3

possible. Lactated Ringer's solution or dextran partially replace lost blood volume while type-specific blood is being prepared. Appropriate laboratory tests are done, including complete blood count, liver function tests, serum amylase, blood urea nitrogen, blood glucose determinations, and clotting studies. As soon as the pulse rate decreases to about 100, or is at least decreasing, esophagogastroscopy is performed and is followed immediately by upper gastro-intestinal x-rays. Patients are then returned to the intensive care ward where gastric lavage is resumed. Patients on the surgical service receive vitamin K, premarin and calcium glucoheptonate intravenously. Atropine is given in doses of 1.3 mg. every 4 hours, and the patient is sedated with intramuscular barbiturates.

In those patients in whom bleeding ceases or slows markedly, nasogastric suction is maintained for 24 hours. The nasogastric tube is then removed, and the patient is given a liquid diet with hourly supplements of antacid or milk and cream mixtures. Vital signs, hemoglobin and hematocrit are followed closely over the next few days.

Patients with the diagnosis of Mallory-Weiss laceration who do not stop bleeding within the first few hours or those who have received more than three or four units of blood and continue to bleed are operated upon. Control of hemorrhage by the

TABLE 2. *Mallory-Weiss Syndrome, Brooke General Hospital, February 1962–July 1965*

Case Number	Age	Initial Symptom	Etiology of Tear	Method of Diagnosis	Endoscopy Findings	Surgical Findings	Treatment	Result Interval from Admission to Cessation	Other
1	17	Headache	Vomiting	E-G scope	Gastric laceration		Conservative	Stopped bleeding spontaneously on admission	Epigastric pain for 1 yr. before
2	42	Nausea	Alcoholic Vomiting	Surgery	2.5 cm. bleeding gast. ulcer	3 lacerations GE	Gastrotomy, oversew	Surgery 7.5 hr. after admission	Mild hemorrhage 2 yr. later
3	36	Nausea	Alcoholic Vomiting	E-G scope	1.5 cm. gast. laceration		None	No active bleeding on admission	1 UGI hemorrhage 2 yr. before
4	61	Vomiting	Alcoholic Vomiting	Autopsy	Varices		Conservative, S-B tube failed	Died 32 hr. after onset of bleeding	Cirrhosis, hepatic coma on admission
5	46	Vomiting	Alcoholic Vomiting	E-G scope	Distal esoph. rent		Conservative	Died 7.5 hr. after admission	Gastroesophageal laceration, aspiration pneumonia, pulmonary edema
6	47	Hematem.	Alcoholic Vomiting	E-G scope	2 cm. gast. laceration		Conservative	Died 28 hr. after admission	Cirrhosis, varices, 2 previous hemorrhages
7	56	Epigast. pain	Vomiting	E-G scope, surgery	Laceration	3 gastric lacerations	Gastrotomy, oversew, cholecystectomy	Surgery 8 hr. after admission	Acute cholecystitis
8	38	Vomiting	Alcoholic Vomiting	Surgery	Blood in stomach, norm. esoph.	1 laceration	Conservative, S-B tube failed, gastrotomy and oversew	Surgery 9 hr. after admission	Mild episode of hematem. 3 mo. later and 16 mo. previously
9	68	Melena	Alcoholic Vomiting	E-G scope, surgery	Gastric laceration	1 laceration	Conservative, gastrotomy, oversew	Surgery 5 hr. after admission	Ulcer disease for several years
10	45	Epigast. pain	Alcoholic Vomiting	E-G scope	G-E lacer., sm. varices		Conservative	Stopped spontaneously 2 hr. after admission	Had prior history of epigastric pain, on Coumadin
11	44	Epigast. pain	Alcoholic Vomiting	E-G scope	Mucosal laceration			Stopped spontaneously 5 hr. after admission	Duodenal ulcer, old, cirrhosis, hemorrhages
12	44	Nausea	Alcoholic Vomiting	E-G scope	Gastric laceration gast. ulcer		Conservative	Bleeding stopped in 24 hr.	2 previous hemorrhages with diagnosis duodenal ulcer subtotal gastrectomy 1 yr. ago
13	38	Weakness	Alcoholic Vomiting	E-G scope, surgery	Laceration sm. varices, erosive gastritis, esophagitis	Gastric laceration	Conservative, long gastrotomy, oversew	Surgery 21 hr. after admission	Undiagnosed UGI hemorrhage 1 yr. ago

TABLE 2.—(*Continued*)

Case Number	Age	Initial Symptom	Etiology of Tear	Method of Diagnosis	Endoscopy Findings	Surgical Findings	Treatment	Result Interval from Admission to Cessation	Other
14	33	Headache	Vomiting	E-G scope, surgery	Laceration	2 lacerations bleeding, 2 pyloric ulcers with obstruction	Conservative, gastrotomy, oversew, subtotal gastrectomy	Surgery 16 hr. after admission	Ulcer symptoms for 5–6 yr., severe hypertension
15	73	Nausea	Alcoholic Vomiting	E-G scope	Laceration, gastritis, hiat. hernia		Conservative	Bleeding ceased shortly after admission	Hiatus hernia
16	71	Abdomin. pain	Vomiting	E-G scope	Laceration		Conservative	Bleeding ceased after ½ hr.	Laparotomy for massive UGI hemorrhage 10 yr. ago—No findings
17	57	Vomiting	Alcoholic Vomiting	E-G scope	Laceration, esophagitis		None	Bleeding had ceased on admission	
18	36	Hematem.	Alcoholic Vomiting	E-G scope, surgery	Laceration hiatus hernia	Laceration	Conservative, gastrotomy, oversew	Surgery 24.5 hr. after admission	Undiagnosed hematem. and melena 1 yr. ago, hiatus hernia
19	53	Nausea	Alcohol Hiccough Vomiting	E-G scope	Laceration, esophagogastritis		Conservative	Bleeding ceased 24 hr. after admission	Hiatus hernia
20	53	Vomiting	Alcoholic Vomiting	E-G scope, surgery	Laceration, gastritis, hiatus hernia	Laceration	Conservative, gastrotomy, oversew, S-B tube failed	Surgery 8 hr. after admission	Cirrhosis of liver, hiatus hernia, acute necrotic hepatitis, delirium tremens

Sengstaken-Blakemore tube is attempted prior to operation in patients with esophageal varices and in those critically ill from other diseases; if bleeding does not cease immediately with gastric and esophageal balloons inflated, operation is performed. In the absence of associated gastric or esophageal disease, through a high gastrotomy, the laceration is identified and is oversewn with 2-0 chromic catgut. In all but one of eight patients who were operated upon a high gastrotomy exposed the laceration. In one the upper end of the laceration could not be reached, but further hemorrhage was controlled by a Sengstaken-Blakemore tube placed at operation. The esophagus was mobilized in those patients in whom lacerations extended into the esophagus.

Clinical Findings

These 20 patients probably comprise about 5 per cent of all those with upper gastro-intestinal bleeding admitted to this hospital. All patients were men, ranging in age from 17 to 73 years; 19 were 40 years of age or older (Fig. 1).

All patients had a history of hematemesis. Eleven vomited prior to hematemesis, five of whom described a period of severe retching prior to bleeding. In five the initial symptom was nausea, and in four abdominal pain—an unusual finding in reported

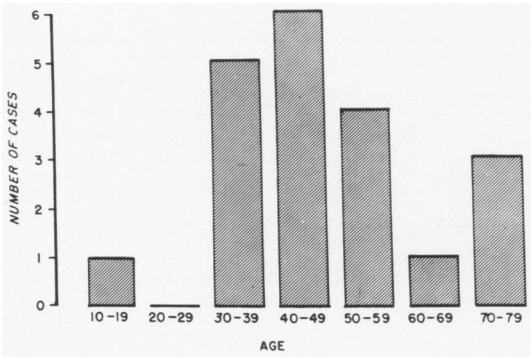

FIG. 1. Age distribution of 200 cases of Mallory-Weiss Syndrome, Brooke General Hospital, Feb. 1962 through July 1965.

cases.[7] Two hypertensive patients had headache as the initial symptom. The remaining seven had vomiting, hematemesis or melena as the first indication of trouble.

Eight patients had previous upper gastrointestinal hemorrhages, and in six hemorrhage occurred within one year prior to the current admission. In six of these eight patients no cause for the previous hemorrhage had been found, and in all bleeding subsided with conservative management. Thirteen had histories of gastric or esophageal disease.

Ingestion of alcohol was a predominant factor. Fifteen had been drinking prior to the onset of bleeding, and in most the drinking was a true alcoholic debauch. Five patients were chronic alcoholics; all three of our deaths occurred in this group. Four others were described as heavy drinkers. In four of the alcoholics cirrhosis of the liver had been diagnosed prior to this admission.

Eight patients were in shock, and ten had anemia of some degree. Five had abnormal liver function tests on admission.

Nineteen patients underwent the described diagnostic program. Esophagogastroscopy showed typical lacerations in 17 of 20 patients. Two more were diagnosed at operation; one was thought at endoscopy to have a bleeding gastric ulcer, and in the other the endoscopist described a normal esophagus but the stomach was so filled with blood that examination was unsatisfactory. One diagnosis was made at autopsy, and esophagogastroscopy in this patient disclosed only esophageal varices, which also were found at autopsy. In no patient did the endoscopist see more than one laceration, but more than one were found in five patients who were further examined either at operation or autopsy. Four patients had associated gastritis or esophagitis, and two had gastric ulcers. Three patients had esophageal varices which were minimal in two. Four had hiatal hernias. One had acute cholecystitis at operation requiring cholecystectomy. Subtotal gastrectomy was done in one patient who had pyloric obstruction caused by two pyloric ulcers.

All patients were given a trial of conservative management. In nine bleeding stopped within a few hours after institution of gastric lavage with iced saline. Attempts to control bleeding by Sengstaken-Blakemore tubes in three patients were unsuccessful.

Fifteen patients required whole blood replacement ranging in amounts from 1,000 to 9,500 cc. Eight patients who were operated upon received an average of 5,019 cc. each with an average of 3,487 cc. given preoperatively. Five patients who were not operated upon required blood transfusions averaging 2,062 cc., and in the three who died the average was 1,833 cc. None of the patients operated upon bled postoperatively.

The only complications were a small wound infection in one patient and a massive postoperative hemorrhage from two short gastric vessels in another.

In the three patients who died, autopsy revealed that two had hepatic cirrhosis; one was in coma before death. The third had delirium tremens and aspiration pneumonia at the time of death.

Discussion

The incidence of upper gastro-intestinal bleeding from lacerations near the esophagogastric junction is not known. Reported series of massive upper gastro-intestinal hemorrhages usually include 10 to 20 per cent undiagnosed cases—some probably Mallory-Weiss lesions. Few reports include esophagogastroscopy as a routine diagnostic measure. Palmer [30] in 1961 reported a 3.5 per cent incidence of the Mallory-Weiss syndrome in 650 cases of upper gastro-intestinal bleeding.

The mechanism of the laceration is vomiting with regurgitation of gastric contents against an unrelaxed esophagus and a contracted diaphragm, as originally postulated by Mallory and Weiss.[24] They supported this contention with experimental evidence and considered the combination of distention of the gastric cardia and the corrosive effect of alcohol important etiologic factors. In 1961 Atkinson et al.[2] presented experimental evidence that tears were directly related to pressure in the cardia. They pointed out, as did Fleischner [13] in 1956, the predilection of hiatal hernia for laceration. Atkinson showed that during retching, pressure in the herniated portion of the stomach corresponds to that of the subdiaphragmatic stomach. The intragastric–intrathoracic pressure gradient during retching in these subjects averaged about 100 mm. Hg which would be applied to the wall of the herniated stomach. Degradi et al.[10] reported 12 cases of the Mallory-Weiss syndrome diagnosed endoscopically, all of whom had sliding hiatal hernias. Atkinson *et al.* postulate that even in individuals without hiatal hernias the esophagogastric junction may sometimes rise into the chest in the act of vomiting and there would be subjected to the intragastric-intrathoracic pressure gradient. In 1964 Grimes [16] pointed out the importance of the inferior esophageal constrictor in the mechanism of this lesion.

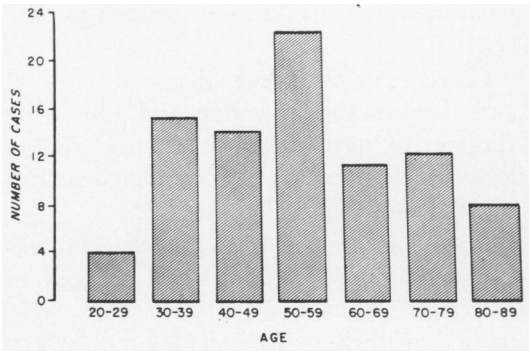

FIG. 2. Age distribution of Mallory-Weiss Syndrome—cases reported in literature.

That vomiting is not necessary to produce the lacerations has been reported.[2, 34] Increased intra-abdominal pressure generated by coughing, straining, asthma, epilepsy, hiccoughing and blunt abdominal trauma have been reported as factors. Kelly [20] reported a case in which the laceration may have been caused by the esophagoscope.

Small and Ellis [33] suggested that the syndrome is similar to spontaneous esophageal perforation. Several authors [15, 23, 33] point out, however, marked anatomic, pathologic and clinical differences between spontaneous perforations of the esophagus and the Mallory-Weiss lacerations.

Initial diagnosis in 121 cases reported has been made at autopsy in 39, at operation in 42 and by esophagogastroscopy in 40. Katz [19] indicated that early endoscopy in upper gastro-intestinal tract bleeding decreased the frequency of the diagnosis of peptic ulcer and increased that of lacerations of the esophagogastric junction. The accuracy of endoscopy is difficult to evaluate because many patients come neither to operation or to autopsy.

Except for Dobbins' [11] case x-ray examinations of the stomach and esophagus have not shown the laceration. X-rays are important, though, in demonstrating associated lesions. Reports of gastric ulcers demonstrated by x-ray in four patients who under-

went operation could be confirmed in only one.

Correct preoperative diagnosis defines and shortens the operation and allows the surgeon to go directly to the site of the lesion. Complications of esophagogastroscopy are rare in skilled hands.[30]

Diagnosis of an unsuspected Mallory-Weiss lacerations was made at autopsy in 34 (54.84%) of 62 patients who were treated medically, 24 (38.71%) of whom recovered and only 4 (6.45%) of whom died after correct diagnosis. Bleeding stopped spontaneously shortly after admission in most of the 24 patients who lived.

The Sengstaken-Blakemore tube has been used without success eight times in reported cases and three times in our series. Etheredge [12] and McPhedran [25] believe that pressure of the balloon is insufficient to control arterial bleeding, but Ferguson in discussing this paper reported successful control of bleeding for two days in one patient.

Of 63 reported operations there were 12 deaths (19.21%). Diagnosis was missed at operation in five of these patients. All patients in whom no source of bleeding can be found should have the esophagogastric junction explored. Bleeding frequently ceases after the patient is anesthetized. In several instances we found lacerations not actively bleeding which bled briskly upon contact with a sponge.

"Blind subtotal gastrectomy," proposed by Cooper et al.,[8] does not attack the lesion [5, 16, 32] and adds risks associated with gastrectomy.

Many technical aids to exploration and mobilization of the esophagogastric junction have been described.[3, 12, 16, 37, 38] We have found mobilization of the esophagus of value in high esophageal lacerations. We have not opened the esophagus.[38] We have used Levine tubes for traction [3] to expose the lower esophagus and a sterile sigmoidoscope for exploration of the area.[37] Grimes [16] pointed out that the stomach wall in this area is easily evaginated for inspection. Differential packing of the stomach [16] aids in localization of bleeding source as does pulling the inflated balloon of a Foley catheter against the esophagogastric junction.

There is nothing in the literature regarding subsequent bleeding. Two of our 17 surviving patients bled subsequently and neither required hospitalization or treatment.

Summary

The paucity of cases of Mallory-Weiss syndrome reported in the medical literature since the original report in 1929, gives the impression that the lesion is rare. Routine early endoscopic examination of the esophagus and stomach in patients with bleeding from the upper gastro-intestinal tract results in a marked increase in the frequency with which the characteristic esophagogastric laceration is detected and in increased awareness of the possibility of its existence in all instances of upper gastrointestinal bleeding. Correct diagnosis leads to proper treatment, whether operative or unoperative. The occasional incorrect endoscopic diagnosis need not delay proper treatment.

Twenty patients with the Mallory-Weiss syndrome were admitted to Brooke General Hospital during a 3½-year period. The syndrome is defined and its possible mechanisms discussed. On the basis of clinical characteristics of patients in this series, a program of management has been outlined. Principles of management of patients with this syndrome include a trial of conservative supportive treatment followed by surgical intervention if conservative management fails. High gastrotomy, adequate exposure of the esophago-gastric region, and oversewing the lacerations are among the technical procedures which contribute to

recovery. The general futility of using the Sengstaken-Blakemore tube and of blind gastric resection is discussed.

Seventeen of 20 patients survived. The three who died were all alcoholics, two were known cirrhotics, one being in liver failure. The third died of delirium tremens and aspiration pneumonia. All eight patients who underwent operation survived.

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Book Review

Cryotherapy of Ocular Diseases. John Bellows, M.D., Philadelphia, J. B. Lippincott Co., 1966. 150 pp. $10.00.

THIS publication, Cryotherapy of Ocular Diseases, is a splendid addition to the library of ophthalmic surgery. In this era of great change, when better and safer methods in the interest of prevention of blindness are being sought, Dr. Bellows discusses in a clear and concise way with excellent illustrations, the application of low temperature technics, with particular references to cataract extraction and repair of retinal separation. He takes the reader carefully through the physical aspects of ice, free and bound water, the different types of freezing, slow, rapid and ultra-rapid and the effects of survival of frozen organic tissue—as well as the principles of cryobiology, cryogens and cryogenic instrumentation (it is of passing interest to note that Dr. A. V. Roslavtsev, Director of the Helmholtz Eye Institute in Moscow, USSR, gave me the Krwawicz probe as a gift, while there on the first medical exchange program in ophthalmology in 1963—it was his feeling the cryolt method may very well prove to be the best and safest way to remove a cataract. This is in agreement with the vast experience of Dr. Bellows).

The basic principle of lens extraction, particularly intumescent and partially subluxated lenses, are compared with the conventional methods, setting forth the greatly decreased percentage of failures, the wider margin of safety and the reduced element of tension on the part of the operating surgeon. With care during application of the low temperature instrument used, the operative and postoperative complications are greatly reduced over the conventional methods.

Cryotherapy in the repair of retinal separation is likewise explained with clarity and apparently replaces the conventional methods of diathermy and the laser ray, effectively creating an adhesive chorioretinitis. Other aspects of anterior segment pathology are discussed and successfully treated. Today, surgeons need not fear shrinkage of the vitreous which often occurs with diathermy or photo-coagulation as well as accidental penetration and avoidance of hypotany and hemorrhage, the complications resulting in surgical failures. Re-operation, when necessary is safe and simplified especially with thin and atrophic scleras. It is well to remember, however; success in repair of separated retinas depends on the cause, the location, the extent of the damaged retina and the duration of the separation, regardless of the method utilized.

The author discusses the effective application of cyclo-cryotherapy in the treatment of uncontrolled glaucoma patients at low temperatures of minus 60 to minus 80 degrees centigrade, over the region of the ciliary body. There is little pain or discomfort to the patient. When such an extensive area of the ciliary body is treated with diathermy, a severe form of uveitis often results, leading to phthesis bulbi.

In light of this newer means of effective therapy, we must not lose sight of the fact, that many experienced surgeons who still use the older methods have most satisfactory results. The pioneering efforts in the application of cryosurgery by such men as Bietti, Deutschmann, Krwawicz, Temple Fay and Henry Wycis of Philadelphia are beautifully exemplified by Dr. Bellows.

We are particularly indebted to Dr. Bellows for his splendid book reflecting his vast experience and research which we are quite certain will be freely used by Ophthalmic surgeons throughout the world.

With its simple but dignified binding and fine quality paper, the book is an excellent production.—MILTON J. FREIWALD, M.D.