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### MASSIVE HEMATEMESIS\*

ANALYSIS OF 300 CONSECUTIVE CASES CYRIL COSTELLO, M.D.

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One could not justifiably undertake to discuss the topic of massive hematemesis without reference to much in the vast library of reports on this subject but because of its extensiveness a thorough review cannot here be presented. Adequate reports have been made by Gray and Sharpe, I Zinninger, Meulengracht, Heuer, Meyer, Eads, Allen and Benedict, Hinton, healfour, I Jordan and Kiefer, and a host of others. In seeking, however, to learn the best methods for management of this grave condition, one encounters a divergence of opinions among authors as to whether treatment should be medical or surgical and if the latter, how such cases are to be selected. Other problems in therapy include those concerning the advisability of using gastric siphonage, oral feeding, blood transfusions, and sedation.

In order to arrive at a better understanding of the principles of anatomy, physiology, and pathology involved and to correlate these principles with a plan of rational therapy, a study was undertaken at the St. Louis City Hospital.

In this study, 300 patients who had presented the symptoms of massive and severe hematemesis were selected. No patient was included in this study who had not vomited large quantities of gross blood and who did not show evidence of blood loss by shock or by severe anemia or by both. Thus, individuals who may have vomited as much as a cupful of blood, but who did not show evidence of shock or severe anemia, were omitted. A second portion of this project was begun early in 1946 and consisted of treatment of those patients presenting massive hematemesis according to a modified plan. The results in this series of 73 patients have also been determined.

#### CAUSES

In our series of 300 patients (Fig. 1), chronic duodenal ulcer leads the list of causes, accounting for 171 patients or 57%. No diagnosis was made in four patients (1.3%). Acute gastritis was the underlying cause of massive hematemesis in 42 patients (14%) while chronic gastric ulcer accounted for 33 patients (11%). Ruptured esophageal varix occurred in 24 patients (8%) and

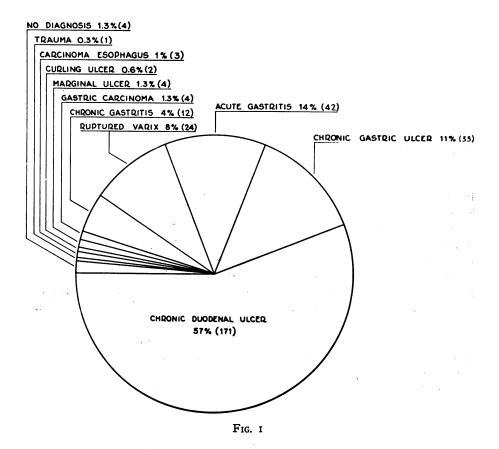
<sup>\*</sup> Submitted for publication, December, 1948.

chronic gastritis in 12 patients (4%). Gastric carcinoma was one of the rarer causes of massive hematemesis accounting for only four such cases (1.3%). Marginal ulcer occurred in four patients (1.3%), Curling ulcer in two patients (5%) and carcinoma of the esophagus in three patients (1%) while trauma of a bullet wound was the cause in one patient (.3%).

### SEX AND AGE INCIDENCE

Two hundred and forty-seven of the patients (82%) were males while only 53 patients (18%) were females (Fig. 2). The incidence of massive hema-

### CAUSES OF MASSIVE HEMATEMESIS IN 300 CASES



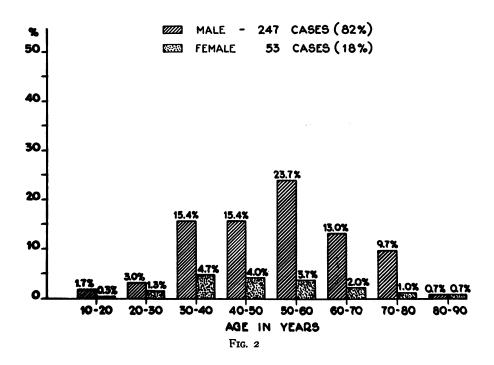
temesis was greater between the fourth and eighth decades in both sexes, the highest incidence occurring in the sixth decade (23% of the males).

### MORTALITY IN PATIENTS TREATED BY CUSTOMARY MEASURES

The overall mortality rate in 300 cases was 25%. Consistent with observations and reports of others studying this subject, mortality rate was found to

increase in those who had reached or passed the fifth decade of life. Thus it was seen that among males, while in the fourth decade, only 10% died, 20% died in the fifth decade, 28% in the sixth, and 38% in the seventh (Fig. 3). Certain pathologic lesions bore a higher mortality incidence than others (Fig. 4). Thus of four patients who bled massively from carcinoma of the stomach, all died. The two patients with Curling ulcers, both died, and three patients of the four who presented marginal ulcers died. Of the 24 patients with ruptured esophageal varices, 71% died and in those patients who bled from carcinoma

## SEX AND AGE INCIDENCE OF MASSIVE HEMATEMESIS IN 300 CASES



of the esophagus, 66% died. Of 33 chronic gastric ulcer patients, deaths ensued in 48%, while in those with chronic duodenal ulcer only 13% died. In patients who presented acute gastritis (42) only 12% died, and of those who presented chronic gastritis as the basis of massive hemorrhage (12) 9% died.

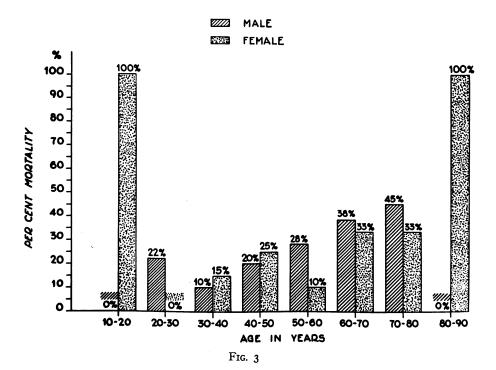
### **PATHOLOGY**

Surgical or autopsy examination afforded opportunity for pathological examination in about 85% of those patients who died. Of particular interest and significance was the status of the eroded blood vessel. As is well known, the commonest vessels involved in such major upper intestinal hemorrhages are the branches of the right and left gastric and the pancreatico-duodenal

arteries and the esophageal veins. The size of vessels involved varied from large and prominent gastric arteries to those involved in multiple superficial gastric ulcers which could not positively be identified. In one instance erosion of a chronic gastric ulcer had extended into liver sinusoids.

Sections of arteries and veins involved were not obtained in all instances. However, in those who died with erosion of a large, easily demonstrable artery (usually in the base of a chronic ulcer) microscopic studies were usually performed. In nearly all of these sections there was seen and described a partial

### MORTALITY BY AGE AND SEX IN 300 CASES OF MASSIVE HEMATEMESIS



or complete block of the eroded wall by antemortem thrombus, and in these the stomach and duodenum contained little or no fresh blood. This demonstration of thrombus formation in even large eroded gastric arteries is of paramount importance in arriving at a rational plan of therapy. It is evident that many of these patients did not die, as has been commonly assumed, from acute exsanguination through a wide open artery. This misconception has led to recommendations that patients with arteriosclerosis and chronic ulcers should be subjected more promptly to operative closure of the bleeding point. It would rather appear on the basis of pathologic studies that just the contrary is true, for thrombus has often already formed prior to the time of death and such a

death, therefore, must be attributed not to continued acute hemorrhage but rather to the complications of diminished blood volume (shock, cardiac failure, anemia, pneumonia, etc.) produced after the hemorrhage had ceased. This fact is confirmed by the clinical course of patients who died. They did not die in a matter of minutes or hours (with one exception) as would be expected from a persistent fulminant hemorrhage, but died from two days to three weeks after the onset of bleeding. Of the 75 patients who died, 71 of them did not receive even a moderate fraction of blood which would have been required to replace the amount lost.

On this evidence then one must logically reason that therapy should primarily be directed not toward surgical closure of the injured vessel which natural processes often will close spontaneously, but toward support of the depleted blood volume.

### PHYSIOLOGY

The body response to massive blood loss is well known. In an attempt to supply vital centers with the remaining blood volume there is a generalized vasoconstriction, an increased rate of heart action, and absorption of tissue fluids into the general circulation. If inadequate blood volume continues, anoxia with its irreversible tissue damage ensues. In view of the universal acceptance of this well-established fact, it is surprising to find recommendations still made that in such a situation blood should not be used or if used, should be used sparingly. The theory that restoring blood pressure risks "blowing out" a forming thrombus has no scientific substantiation while on the other hand, no one has yet been able to improve on shock-prevention as the best shock-therapy. To wait for a fall of blood pressure to 80 or 100 mm. of mercury before administering a "small transfusion" as is often recommended is a direct contradiction of sound physiologic principles.

### BLOOD REPLACEMENT PLAN

If these pathologic facts and physiologic principles are correct, the optimal choice of treatment in massive hematemesis should be essentially replacement of as much blood as has been lost as promptly as possible until thrombus formation has sealed the eroded vessel or vessels. In testing these principles during the past two years, 73 patients were treated by adequate and prompt blood replacement. The distribution of these 73 patients by sex and age corresponds closely to that of the previous group studied. There were for example 51 of these 73 patients who had reached or passed the fifth decade of life, and there were eight of these who had reached or passed the eighth decade of life. Operative interference during active bleeding was not undertaken in any of these cases. Feedings were usually administered, and the type of feeding varied between the Meulengracht diet, the Sippy diet, liquid diet, and protein-dextrimaltose ("pre-digested") liquids. As should be anticipated, the difference in mortality rate was striking (Fig. 5). Only 4% of this group of 73 patients died as compared to 25% of the 300 treated by various methods.

In further analyzing this difference in mortality, it is clear that the single most important factor in minimizing death has been that of adequate blood replacement. It is highly doubtful if this policy could be routinely used in an institution which does not have a blood bank facility. The total quantity of blood required by many of these patients is enormous as judged by transfusion standards of a decade ago. Often patients who had lost large quantities of blood and who were still bleeding when admitted to the hospital received as much as three and four liters of blood during the first 24-hour hospital period. The frequent use of intravenous saline solution should be condemned as it does nothing toward re-establishing blood volume, and encourages pulmonary edema. The fluid of most value to be used while blood is being cross-matched

### MORTALITY (25%) 75 DEATHS IN 300 CASES

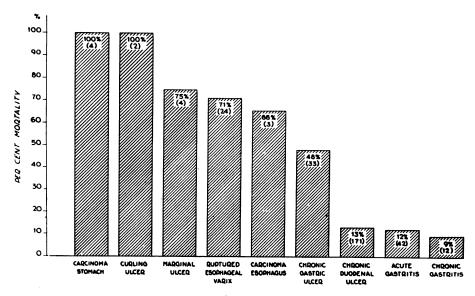


Fig. 4

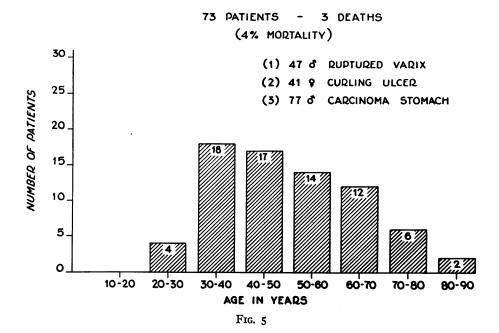
is plasma. Another essential for enforcing adequate blood replacement is the readily available facility for rapid determination of blood requirements. The need for blood cannot properly be determined by a patient's general appearance, pulse, or blood pressure. The older methods of blood study such as blood count and hematocrit are either undependable or too time consuming to be of value in these cases. The most desirable method for accurately following blood volume changes is the copper sulfate falling drop method. The device for this simple procedure has been set up on the hospital divisions and in the matter of a few minutes with only a few drops of blood, the initiated houseofficer is able to discover the specific gravities of blood and plasma, the hemoglobin, and the hematocrit.

#### MASSIVE HEMATEMESIS

In addition to blood volume replenishment, several other factors have been given special attention.

- 1) Gastric siphonage
- 2) Diet and antacid therapy
- 3) Sedation
- 4) Surgery
- 5) Early differential diagnosis.
- I) Gastric Siphonage. The insertion of a Levine tube via the esophagus into the stomach was seen to produce fatal hemorrhage in two patients. It was instrumental in provoking increased hemorrhage in several others. Ice water for lavaging was used in the two cases of fatality, and one of these

# 1946 - 1947 (ADEQUATE BLOOD REPLACEMENT)



patients expired while the process was being carried out. The danger of traumatizing a varix or ulcer should interdict use of the tube unless there is definite gastric distention and nausea.

2) Diet and Antacid Therapy. The question as to whether feeding should or should not be employed has long been debated. Meulengracht has reported favorably on feeding pureed diets although a large proportion of his ulcer patients presented only minor bleeding. In the light of current knowledge that most of these patients have a deficit of protein and vitamin which impedes

tissue repair, it becomes imperative that they receive large amounts of readily assimilable protein, carbohydrate, and vitamin. The most satisfactory method for administering these essentials we have found has been oral ingestion of 200 cc. every two hours of the following preparation:

Predigested protein powder (polypeptide)	150 Gm.
Dextri-maltose	300 Gm.
Vitamin C (cevitamic acid)	ı Gm.
Liquid multiple vitamin	I cc.*
Water q.s.	2400 cc.

This preparation is well tolerated with rare exception and serves not only the purpose of supplying greatly needed nutriment but also produces symptomatic relief of ulcer pain. This is apparently accomplished through the protein antacid effect which is much more effective in degree and in duration than the more commonly employed antacid powders. This same preparation has been highly satisfactory in relieving severe ulcer pain of impending perforations. The polypeptide component is used rather than amino acids since it is palatable.

- a) Sedation. The most generally adopted plan for sedation in the past has been that of administering morphine or barbiturate as needed. In practice this usually degenerates to a matter of a sedative being finally given after the patient has repeatedly complained to the nurse of restlessness, fear, or pain. The numerous psychogenic impulses attendant upon massive hemorrhage and nausea have appeared often to provoke further active bleeding and it has, therefore, been our policy to relieve these patients totally of their adverse stimuli. This has been accomplished by administering a single initial dose of morphine followed by hypodermic injections of two grains of sodium phenobarbital every two hours as needed to keep the patient drowsy. We have considered the patient properly sedated when it is necessary physically to arouse him in order to obtain replies to questions. The danger of oversedation must, of course, be carefully avoided.
- 4) Surgery. The major disadvantages to surgical procedure during active bleeding are: a) Patients are in poor condition from blood loss and while bleeding is active they may be expected to become worse.
- b) Nutritional deficiencies of a chronic nature must be anticipated in patients whose ulcers are active enough to have produced erosions of major vessels. Operations undertaken before these deficiencies have been corrected must result poorly in many cases.
- c) The nature and location of the ulcerative lesion cannot safely or accurately be determined early in the course of hemorrhage and routine operative approach with its risk cannot be serviceable or successful enough in treating all causes of massive hemorrhage to justify its use. As pointed out by Stone<sup>14</sup> such an operation may still fail to discover or relieve the trouble.
  - d) Finally, the ultimate test of value, that of clinical results in the non-

<sup>\*</sup> Any liquid vitamin which contains the daily requirements should suffice. Upjohn's "Zymadrops" has become our material of choice.

surgical treatment of massive hemorrhage speaks unequivocally against operative interference. Prior to our initiation of this non-operative plan of therapy, ten patients had been operated upon late but while still bleeding and death ensued in 90%.

An interesting study in this regard is being conducted by Stewart<sup>18</sup> et al. They are combining massive blood replacement and exploratory laparotomy within the first 24 hours of hospitalization. Their results in such treatment of 19 patients have been accompanied by four deaths representing a mortality of 21% which may improve as the series of cases enlarges but which we doubt will ever compare favorably to active and complete non-surgical management.

A word should be said about that small group of patients who continue bleeding for seven to ten days or who begin bleeding again after apparent control. While the urge to intervene is great, the wisdom of persisting in the plan as set forth has been demonstrated conclusively in this series in that all patients finally ceased bleeding and were improved to the point that surgery could be undertaken without great risk. On the other hand, the inadvisability of operating on this type of patient is forcibly demonstrated by our extremely poor results of nine deaths in those ten patients so treated prior to our adopting the non-surgical plan.

5) Early Differential Diagnosis. The patient with active or recent major intestinal hemorrhage should not be subjected to enthusiastic diagnostic routines. Hemorrhage should be treated only symptomatically until controlled and then the matter of diagnosis may be pursued without the great danger of reprecipitating or exaggerating hemorrhage. In our own series of patients we have seen major hemorrhages re-started by barium studies and gastric analyses. The information so obtained rarely if ever alters the prescribed course of therapy early in the condition and so it becomes a matter of accomplishing an earlier diagnosis solely for the purpose of the record at the expense of increased mortality. It requires seven to ten days for moderately firm fibroblastic tissue reaction to form in an organizing thrombus and this picture should be borne in mind by the gastroscopist and fluoroscopist lest irreparable harm be done.

### SUMMARY

Some of the results from a study of 300 patients with severe, massive hematemesis have been presented. The results of 73 patients treated by a massive blood replacement plan have also been presented. Mortality by this latter plan has been reduced to 4%. There was no death from chronic peptic ulcer, the commonest cause of massive hematemesis.

The jollowing plan is, therefore, recommended for treatment of massive hematemesis during the actively bleeding stage:

- 1) Careful determination of blood needs by the copper sulfate falling drop method repeated frequently.
- 2) Complete restoration of blood as promptly as possible.

- Oral administration of predigested protein-carbohydrate-vitamin mixture.
- 4) Adequate, continuous sedation.

The following procedures have been found to increase mortality during the actively bleeding stage:

- 1) Surgery
- 2) Indwelling stomach tube with constant suction
- 3) Gastric lavage
- 4) Active gastric diagnostic studies.

The problems in management of these patients after the active hemorrhage stage has passed have not been the province of discussion at this time, but instead, emphasis has been placed on the principles and factors of importance in the management during the stage of active hemorrhage. Following the control of hemorrhage and the restoration of nutrient and blood essentials, the advisability of elective surgical intervention may more easily be determined.

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