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Present-Day Problems in Nonpenetrating Abdominal Trauma*

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I AM VERY SENSIBLE OF THE HONOR of being invited to deliver the annual Oration on Trauma. My long interest in this field stems from my many years of association with my father, one of the pioneers in this country in the surgery of trauma, and, while more recently my major concentration has been in other realms of surgery, abdominal trauma has never ceased to have a peculiar fascination. Also, as a member of the College's Committee on Trauma for over 25 years, I have passed through this group's vicissitudes in name and in interest, as it progressed from fractures only to take in the entire field of trauma. With the change in general outlook and breadth of perspective of what was formerly the Fracture Committee, it was logical to change the designation of this annual address from the Oration on Fractures to the Oration on Trauma.

Therefore, while my distinguished predecessors have followed the original intent and have confined their discussions largely to the field of fractures, I am venturing to break away from this tradition, to take advantage of the broader potentialities of the entire field of trauma, and to direct my remarks to certain phases of the problem of abdominal injuries.

A recent review of 84 consecutive cases of nonpenetrating abdominal trauma, admitted to our clinic from 1935 to 1953, reveals certain pertinent facts which should serve to highlight the present problems concerning this field of surgery.

There was a total of 145,737 hospital admissions during the period in which these 84 instances of abdominal trauma were encountered. Roughly, this is an incidence of one in 1,750 admissions, an indication that in civilian life nonpenetrating abdominal trauma is not frequently encountered by the individual surgeon; and, unless he has had war experience in the therapy of abdominal trauma, he is not likely to avoid the pitfalls that beset successful treatment of these lesions.

MORTALITY

The mortality and the viscera involved in this series are demonstrated in the following table.

Viscera	Number Cases	Deaths	Mortality Rate
Kidney	25	0	0
Liver	9	3*	33.3%
Pancreas	3	0	0
Spleen	20	6**	30.0%
Stomach, pancreas, duodenum	1	1	100.0%
Intestine			
Duodenum (Retroperitoneal)	2	0	0
Jejunum	8	1	12.5%
Ileum	0	0	0
Colon	2	0	0
Bladder	9	1	11.0%
Diaphragm	2	2	100.0%
External iliac artery rupture	1	1	100.0%
Contusion of abdomen	2	0	0
TOTAL	84	15 or	17.8%

*One not operated upon.
**Three not operated upon.

Fifteen of the 84 cases died, an over-all mortality of 17.8 per cent. Sixty-one (72.6%) involved the solid viscera, 35 of these the spleen and liver, 9 of whom died (25.7%). It is to be observed, therefore, that the greatest mortality and the greatest incidence of nonpenetrating abdominal trauma were found to be in lesions of the solid viscera and, particularly, of the spleen and liver. Rupture of the diaphragm, thoracic lesions in combination with abdominal trauma, and rupture of the greater blood vessels also contribute to mortality.

REPORTED MORTALITY OF LAST 50 YEARS

	Year	Cases	Mortality Rate
Peterson	1900		30.0%
Demal	1933	126	21.9%
Lewis & Trimble	1933	140	20.0%
Welch & Giddings	1950	172	14.0%

While there has been a gradual commendable lowering of mortality from abdominal trauma down the years, mortality is still too high, in view of our present-day potentialities for its prevention. As evidence that our present armamentarium does afford opportunities for a lower mortality, of 23 of our series treated since 1950, only two died (8.7%). Welch and Giddings in reporting on 56 cases treated since 1943, report two deaths (3.6%).

*Oration on Trauma, presented at the Clinical Congress, American College of Surgeons, Chicago, October 7, 1953.

It would seem worthwhile, therefore, not only to point out that a major problem in nonpenetrating abdominal trauma is improved application of ways and means to reduce mortality; but also to indicate factors in mortality which promise fruitful fields for corrective measures.

FACTORS IN MORTALITY

1. ACTUAL CAUSES OF DEATH

The actual causes of death in our series may be summarized as follows:

1. *Hemorrhage*.....7
   Secondary to rupture of:
   - (a) Spleen..... 4
   - (b) Liver, spleen, lung..... 1
   - (c) Bladder..... 1
   - (d) Iliac artery..... 1

2. *Peritonitis*.....3
   Secondary to rupture of:
   - (a) Stomach..... 1
   - (b) Jejunum..... 1
   - (c) Liver..... 1

3. Concomitant thoracic lung injury....4

4. Concomitant cerebral trauma.....1

It will be observed that hemorrhage and peritonitis were responsible for two-thirds of the mortality and represent in large part preventable deaths. However, concomitant cranial and thoracic injuries constituted a definite lethal element in those casualties with multiple trauma.

2. MULTIPLE INJURIES

Forty-three (56.2%) of the 84 cases had multiple injuries, 14 (32.5%) of these died. All but one of the deaths in this series occurred in accident victims with multiple injuries. These consisted of a combination of (1) head and neck, (2) chest, and/or (3) extremity lesions with (4) abdominal trauma.

These combinations of concomitant trauma introduce at least two elements contributing to mortality. First, the extra-abdominal injury itself, such as an open comminuted fracture of skull with severe cerebral trauma, severe crush of chest, et cetera, may be the cause of death. Second, the extra-abdominal injury may mask or delay the symptoms and signs of abdominal visceral rupture and the indications for operation.

To avoid mortality in multiple injuries, therefore, certain requisites are essential. They are (1) sound judgment capable of establishing priority or definitive treatment for the injuries incurred; and (2) constant vigilance to insure that an abdominal emergency demanding immediate operation is recognized.

3. FAILURE TO RECOGNIZE AN ABDOMINAL EMERGENCY

Thirty-nine of our 84 cases were not operated upon; of these 9 died. The nonoperated cases consisted of—

Contusion of kidney	23
Contusion of abdominal wall	2
Liver laceration or contusion	5
Multiple injuries (all died)	6
Splenic rupture (all died)	3

All injuries of the kidney were mild contusions or lacerations. None showed significant changes in the intravenous urogram. Hematuria was the chief symptom, which cleared up spontaneously. None required operation, and none died.

The cases designated as liver laceration or contusion were patients with upper abdominal trauma, jaundice, mild anemia, nausea and vomiting, in whom a lesion of the liver seemed most likely. They recovered spontaneously.

The cases of multiple injuries and splenic rupture all died, a total of nine. The six cases of multiple injury were *in extremis*, dying within three hours of admission. However, one of these that came to autopsy and the 3 cases of splenic rupture are of sufficient interest to merit a brief summary.

Case I. This patient had a very extensive crushing injury to the right chest and fracture of ten ribs with marked dyspnea. He was admitted *in extremis* and died within three hours after arrival at the hospital. The full significance of injury to the liver was masked by the excessive trauma to the chest. Autopsy revealed not only extensive laceration of the liver, but also rupture of the spleen and laceration of the lung, as well as multiple bilateral fractures of ribs.

Case II. A man who had a pneumonectomy for bronchiogenic carcinoma two years before injury was brought to the hospital, three hours after a motor accident, in shock, semicomatose and dyspneic. Several ribs in his left chest and his tibia were fractured. He failed to respond to multiple transfusions and died within ten hours of admission. The patient was quite emaciated and metastatic carcinoma, in addition to the obvious fractures and chest injury, was suspected, but at

autopsy a ruptured spleen with no evidence of recurrent carcinoma was found.

Case III. A delayed unrecognized rupture of spleen proved lethal in a man who had multiple fractures of ribs, laceration of a lung, and fracture of a forearm. Fatal hemorrhage occurred on the fifth posttrauma day. The extensive thoracic injury completely masked evidence indicating significant abdominal trauma.

It is evident that severe thoracic injury may well obscure signs or symptoms indicative of intra-abdominal hemorrhage or rupture of an abdominal viscus. However, it would seem equally certain that with extensive lesions of the chest, particularly if unilateral, upper abdominal trauma in addition must be suspected, and indications for abdominal exploration persistently sought. Better therapy and routine consideration of early operation in combined thoraco-abdominal trauma including rupture of the diaphragm should be fruitful in lowering mortality from abdominal injuries.

Case IV. A man aged 58 was admitted to the medical service and was never seen by a surgeon. Four months before admission, while lifting a wheel barrow with a heavy load of gravel, he had a sudden severe pain in the left loin and back. Although it was relieved by rest, pain had recurred practically every day since, radiating at first to the left thigh, but more recently to the left shoulder. For three months he also had had a cough, shortness of breath on exertion, weight loss of 25 pounds, anorexia and from three to four loose stools a day. He showed marked emaciation, an enlarged heart with mitral systolic murmur, a tender mass in upper left abdomen from 10 to 12 centimeters in diameter, and a right inguinal hernia. Blood pressure read 218/140, temperature 101°, pulse 120. He was admitted as a cardiorenal problem, though malignancy of the stomach, colon or kidney was suspected in addition.

Laboratory data are as follows: hemoglobin, 7.5 grams per cent; red blood cells, 3,200,000 per cu. mm.; white blood cells, 11,800 per cu. mm.; sedimentation rate, 30 mm/hr. Electrocardiogram showed myocardial disease. X-ray showed elevation of diaphragm on the left, with atelectasis of lower lobe of left lung. X-ray examination of colon and intravenous urogram were negative.

Nine days after admission, a sudden desire to defecate was followed by rapid collapse and death. Autopsy showed coronary sclerosis, myocardial hypertrophy and a completely unsuspected rupture of an otherwise normal spleen, with large old and fresh intraperitoneal blood clots and a very large old left perirenal hematoma.

This extraordinary case of delayed rupture of spleen or rupture with intermittent hemorrhage over a period of four months following injury, indicates the widespread implications of abdominal trauma, and the difficulty in recognizing delayed hemorrhage from trauma when the interval from time of injury to the frank evidence of hemorrhage is long, or prolonged. Olander and Reiman[9] have recently described a similar case in which the interval between injury, and operation and recognition of concealed and delayed hemorrhage from old rupture of the spleen, was two and one half years. Certainly in obscure abdominal tumors with any history of trauma, hematoma from a ruptured viscus must be kept in mind as a potential diagnosis. In sudden collapse in these patients, acute hemorrhage must be considered as a possible etiological factor.

4. MULTIPLE INTRA-ABDOMINAL LESIONS

Multiple intra-abdominal lesions in nonpenetrating trauma are not common. Two cases were found in our series and both died. One was a rupture of liver with laceration of spleen and lung. The other was a man with a large rupture of the stomach and extensive contamination of the peritoneum by food. After suture of the rent in the stomach, he died of fulminating peritonitis in 48 hours. Autopsy revealed, in addition, a retroperitoneal rupture of the duodenum and laceration of the pancreas in its middle third.

However, it is likely that of those cases admitted *in extremis*, who died in a few hours, any or all might have had multiple intra-abdominal trauma.

5. LATE RECOGNITION OF A RUPTURED VISCUS

The patient, as well as the surgeon, may be at fault in not recognizing the potentialities and seriousness of an injury and in refraining from consulting a physician.

In our present series the only patient with intestinal rupture who died was a man of 70, who was struck in the abdomen by a piece of plywood being cut by a buzz saw. Twenty-four hours after injury he entered the hospital in shock, having ignored early mild symptoms of pain, and vomiting. He had not consulted a doctor previously. At operation, after multiple transfusions were begun, two

ruptures in a badly contused proximal jejunum were found, with extensive peritonitis. Resection of the involved jejunum failed to suffice; the patient died of peritonitis in 48 hours.

6. DELAY IN ONSET OF HEMORRHAGE

Delay in onset of hemorrhage or in symptoms indicative of hemorrhage following trauma may be due to two causes. (1) One is subcapsular hemorrhage in a solid viscus following a subcapsular laceration or contusion. The hemorrhage may at first be intracapsular; hours, even days, weeks, or months after injury, severe hemorrhage ensues as a result of late rupture of the capsule. (2) The other cause is temporary arrest of hemorrhage by clot or adhesion. The initial hemorrhage may result in the formation of a clot that may temporarily arrest the hemorrhage or an adhesion to nearby viscera may cause cessation of active bleeding for an interval.

7. DELAY IN SIGNS, PERFORATION OF HOLLOW VISCUS

Delay in the signs and symptoms characteristic of a laceration or rupture of a hollow viscus may result from (1) shock initially may mask signs of a perforation or rupture, but when appropriate treatment has overcome shock the clinical picture will be clear; (2) spasm or spastic contraction of intestine following injury may prevent, for an interval, leaking of intestinal contents; and delay signs of peritoneal irritation and peritonitis; (3) temporary closing over of a laceration of the intestine by the omentum or by adhesion to adjacent viscera may also delay leaking of intestinal fluids, and thereby delay signs and evidence of peritonitis; and (4) concomitant head and chest injuries may mask or delay symptoms characteristic of rupture as a result of unconsciousness, peripheral paralysis or extensive lung and rib injuries.

Summary

Mortality in subcutaneous abdominal trauma, therefore, rests largely upon multiple injuries both intra- and extra-abdominal, their severity and the high incidence of their masking symptoms of abdominal emergency; extensive lesions of the solid viscera; failure to recognize, or late recognition of, intra-abdominal hemorrhage or an abdominal lesion requiring operation; and delay in onset of hemorrhage or signs of perforation of a hollow viscus.

Obviously, exceptional vigilance to detect indications for immediate abdominal exploration is particularly demanded in chest injuries with rib fractures, cases in shock, in cases in which intra-abdominal hemorrhage seems likely, and in cases of cerebral injury.

MEASURES TO COMBAT HIGH MORTALITY

1. GENERAL MANAGEMENT OF OBSCURE CASE

The general management of the obscure case is of vast importance. Constant observation, 24 hours a day, with frequent check and recheck of all cases in which at the first examination indication for operation is not clear, is imperative. Simple lip service to this pronouncement does not suffice. The supervision and re-examination should be done by the resident or a staff surgeon. It must be the responsibility of the surgeon in charge or his delegated substitute. Onset of significant signs and symptoms indicating operation may be delayed; yet eventual rapid change to a clinical picture readily recognized as demanding operation may occur.

Repeated blood counts, blood volume, hemoglobin and hematocrit determinations may be helpful. Repeated x-ray observations may be enlightening, but nothing can supplant, in *lifesaving significance, the repeated clinical examination of the patient,* until decision for operation has been reached or convalescence is complete.

2. IMPROVED TREATMENT OF SHOCK, AND OPERATION WHILE IN SHOCK

The primary cause of shock is loss of blood, which may be due either to the trauma itself or to hemorrhage. It is important to know when hemorrhage is the etiological factor. Aids to the diagnosis of hemorrhage are the estimation of blood volume, hematocrit, blood counts and hemoglobin determination. Early in severe hemorrhage, blood counts, hemoglobin and hematocrit determination may not give a true indication of severity because of the lag in alteration and change in concentration of the circulating blood. Repeated blood counts and hemoglobin determinations, however, will indicate the significant trend.

Abdominal paracentesis or needle aspiration with a 20 gauge needle and a 20 cc. syringe may yield invaluable information in determining the presence of abdominal hemorrhage in a doubtful case.

Fabian Bachrach

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Treatment of shock by the immediate use of plasma expanders, such as dextran, polyvinylpyrrolidone and gelatin, until blood can be matched and multiple simultaneous transfusions begun, has proved a tremendous asset.

Finally, when shock is due to hemorrhage, operation while patient is in shock but after corrective treatment has been begun will save many lives. This procedure has been well demonstrated in war injuries.

3. EXPLORATORY OPERATION

If reduction of mortality is to be pursued to its irreducible minimum, exploratory laparotomy must be included in our armamentarium. A clear-cut picture of an emergency or indication for operation may not be invariably present. An accurate diagnosis may not be possible. If a downward trend is demonstrated, or a worsening of the patient's condition is obvious, for which rupture or hemorrhage can account, exploratory operation should be promptly undertaken. A negative exploration is far better than a missed diagnosis of a lesion remediable by operation.

4. ANCILLARY AIDS

There are at hand in present-day therapy certain ancillary aids that can play a definite part in the reduction of mortality. These are antibiotics; pre- and postoperative gastric suction; appropriate therapy for the metabolic needs of the patient; better and varied techniques of anesthesia; and definitive measures for the control of hemorrhage—local application of oxycel and gelfoam, suture of rupture or rents in large blood vessels, and vitamin K given parenterally (particularly with liver trauma).

5. BETTER THERAPY FOR TRAUMA OF SOLID VISCERA

As injury to the solid viscera is so frequently encountered, and attended by the highest mortality, a summary of recent improvements in diagnosis and therapy of these lesions is pertinent.

A. *Liver*

In rupture of the liver, an illustrative case will serve to highlight the major problems.

A man of 64 years was brought to the hospital following a motor accident, within an hour of injury. He was irrational, violent and in shock. Pulse was imperceptible, blood pressure 50 systolic, cardiac rate 104, respirations 40. He had marked pallor.

His obvious injuries were a four-centimeter long laceration of scalp (occiput); fracture of right humerus (lower third); fractures of right tibia and fibula (upper third); and multiple small lacerations of face. In addition, his abdomen was full, the upper half tender and rigid. Rupture of a viscus was suspected.

Double simultaneous intravenous infusion of plasma (1,000 cc.) was immediately instituted, followed by 1,500 cc. of blood transfusion. The fractures were put up in traction, the laceration debrided and sutured. Red blood count was 3,930,000, hemoglobin, 12 grams per cent, hematocrit 40 mm/hr. Two hours after admission the abdomen was still rigid, almost board-like, but blood pressure was then 80/50.

Under ether intratracheal anesthesia, the multiple blood transfusions being continued, abdominal exploration revealed a huge oblique laceration of the inferior surface of right lobe of the liver, from the right lateral edge to the base and 2,000 cc. of blood and blood clots in the peritoneal cavity. Clots and blood were rapidly evacuated, laceration

of liver packed with oxycel (three strips) and held together by manual pressure for 5 to 10 minutes. Bleeding was well controlled. Area was drained. A total of 1,500 cc. of blood was given during the operation. Three hours after the operation, pulse was 110, blood pressure 120/80. Recovery was uneventful.

The significant features of this case were no subjective symptoms, patient being irrational, semicomatose and in shock; marked upper abdominal rigidity with fullness; multiple injuries, head, extremities and abdomen; and signs of severe intra-abdominal hemorrhage, rupture of both spleen and liver suspected, but no absolute diagnosis possible. Also immediate treatment for shock, including plasma and multiple simultaneous transfusions, with slight improvement in pulse and blood pressure following treatment; emergency splinting of fractures, debridement and suture of lacerations during treatment of shock; operation in shock, but with simultaneous multiple transfusions continued; intratracheal ether anesthesia; control of hemorrhage from huge laceration of liver by application of oxycel to the lacerated surfaces and manual pressure; drainage of subhepatic space, but no packing; and finally, excellent recovery.

Accurate diagnosis in this case could not be made, due to the lack of both subjective symptoms and definite localizing signs. Rupture of a viscus with severe hemorrhage seemed evident and immediate operation indicated. Pain in the right shoulder area and in the upper right abdomen with localized tenderness, and rigidity accompanied by nausea and vomiting are frequently present in liver laceration. Jaundice or marked pallor and diminished peristalsis may be observed. Bradycardia has been reported.

Aids to diagnosis will be abdominal paracentesis demonstrating blood in the peritoneal cavity; falling red blood count as indicated by repeated tests; and density of upper right abdomen demonstrated by x-ray. Another diagnostic aid is presence of fracture of lower ribs, right thorax. Fracture of one or more of the lower ribs so frequently accompanies liver trauma that rupture of liver should always be suspected when a rib fracture of lower right thorax is present. The use of intravenous diodrast or thorotrast may be of aid. Dye will diffuse into upper right abdomen one to two hours after injection.

Plasma was used in this case, as the injury occurred before the advent of plasma expanders; but plasma substitutes, particularly dextran, are greatly to be preferred as the primary intravenous therapy for shock until matched blood is available. Dextran should be used in ample quantity and by multiple infusions when necessary. But blood in sufficient quantities, in shock due to severe hemorrhage, is the great requisite. Multiple transfusions as soon as possible should be begun, and as soon as improvement in blood pressure is noted, preferably when at least an 80 mm. systolic pressure has been attained, operation may be undertaken, with continuation or stepping up of transfusions. Intratracheal ether has much to recommend it as the preferable anesthesia. A thoraco-abdominal incision will greatly aid the approach to lacerations involving the hepatic dome or superior surface.

To control hemorrhage still active at time of operation, pressure of thumb over the portal vein and hepatic artery against a finger or fingers in the foramen of Winslow may be helpful. The laceration of the liver may then be inspected, cleansed of clots, debrided when necessary, large vessels ligated, and sheets of oxycel or gelfoam spread over the surface; and the bleeding finally controlled by manual pressure holding the laceration together, or by suture. Actual packing of the liver will be rarely indicated, but the area should be drained to avoid intraperitoneal leakage of bile and bile peritonitis.

B. *Spleen*

An analysis of mortality in rupture of the spleen in our series is of interest.

	Number	*Splenectomy*	*Deaths*
Splenic rupture with concomitant injuries	13	10	6*
Splenic rupture alone	12	12	0
TOTAL	25	22	6

*No operation for three, splenectomy for three.

In two of the three cases that died following splenectomy, cause of death was the extra-abdominal lesion. The chance of survival following splenectomy in the uncomplicated case would seem to be excellent. There were 6 cases of delayed hemorrhage, two were not operated upon and died, the remaining four survived splenectomy.

Mortality from splenic rupture is found, therefore, particularly in cases with concomitant trauma —which either masks the signs of splenic injury or is the actual cause of death—and in cases of unrecognized and delayed hemorrhage.

Pain, tenderness, and rigidity in the upper left abdomen with pallor, and shock, even though moderate, are the symptoms and signs suggestive of splenic rupture. Occasionally, there is pain in the left shoulder or scapular region and frequently associated trauma of the left lower chest. Fracture of ribs in the left lower thorax should always arouse suspicion of splenic injury and, when accompanied by diffuse pain, dyspnea and abdominal muscle guarding may so overlay signs of splenic rupture that detection of any intra-abdominal lesion may be exceedingly difficult.

Aids to the diagnosis of a splenic lesion in the presence of associated injuries and in obscure cases are, therefore, of special value and importance and are (1) a flat x-ray film of the abdomen which may reveal density in upper left abdomen, elevation of left diaphragm, and displacement of stomach to right; (2) radiograph one to two hours after intravenous injection of thorotrast or diodrast, which will show extravasated dye in the upper left abdomen[2]; and (3) abdominal paracentesis which may demonstrate blood or bloody fluid in the peritoneal cavity.

Clinically, traumatic ruptures of the spleen may be divided into three groups: multiple trauma including rupture of the spleen; rupture of spleen—the only major injury with a recognizable symptom complex; and delayed rupture of the spleen or rupture with delayed or intermittent symptoms.

Delay in signs and symptoms of splenic rupture occurs as a result of a subcapsular hemorrhage in which rupture of the capsule and free intraperitoneal bleeding does not immediately follow the actual injury; or because of intermittent bleeding from the original laceration or rupture.

While the interval of delay from the time of injury to signs of splenic injury and intraperitoneal hemorrhage is usually a matter of a few days, there are on record cases of extraordinarily long delay in onset of signs of acute hemorrhage; but symptoms referable to the left upper abdomen, such as pain, intermittent in character, or a tumor mass are often present. In these cases suspicion of a splenic lesion should be aroused, particularly in the presence of intermittent pain and a history of antecedent trauma.

Successful treatment of splenic rupture to avoid mortality, therefore, depends upon prompt blood replacement by multiple transfusions with prompt splenectomy; the severity of the concomitant lesions; and early diagnosis of the obscure case.

C. *Pancreas*

Lesions of the pancreas, the result of subcutaneous trauma, may be either a simple contusion with hematoma, laceration of the gland of varying degrees or complete rupture. Extensive injury to the pancreas is not frequently encountered.

Isolated trauma of the pancreas is more commonly a minor lesion which, however, may be followed by the development of a hematoma or pseudocyst. Drainage of the pancreas may result in a pancreatic fistula when the main pancreatic duct or one of its larger branches has been lacerated. Mortality from rupture of the pancreas in association with other injuries is high.

Extensive injury is accompanied by shock, vomiting, severe epigastric pain and abdominal rigidity. In mild trauma to the pancreas early symptoms are vague, but intermittent attacks of epigastric pain with vomiting or often mild persistent pain with exacerbations of marked severity, frequently develop.

Actual diagnosis of a pancreatic rupture will be aided by determination of the blood amylase or lipase, which will be elevated in trauma to the pancreas.

In minor trauma, expectant or conservative therapy will suffice until the development of a hematoma or pseudocyst indicates surgery. Extensive rupture demands immediate laparotomy, with concomitant treatment for shock. Control of hemorrhage and drainage of the traumatized pancreas should prove adequate. When the main pancreatic duct is not involved, suture of the pancreas in selected cases might be worth while. If retroperitoneal hematoma in the region of the pancreas is found with other intraperitoneal trauma, careful exploration of the hematoma for evidence of pancreatic damage would seem justified, and drainage instituted.

Early recognition of a pancreatic laceration and early operation offer the most toward reduction of mortality, but the high mortality from a pancreatic lesion in association with other intraperitoneal trauma still awaits satisfactory solution. Careful search for pancreatic involvement in every laparotomy for abdominal trauma, appropriate operative therapy and better postoperative care may turn the tide.

Summary

Measures available to combat high mortality in nonpenetrating abdominal trauma therefore include improved treatment of shock; operation while in shock; exploratory operation in selected cases; certain ancillary aids, such as the antibiotics, better anesthesia, definitive means for the control of hemorrhage; and better therapy in trauma of the solid viscera.

CONCLUSION

In conclusion, the most pressing present-day problem in nonpenetrating abdominal trauma is reduction of high mortality. An attempt has been made to indicate the apparent causes of this mortality, to outline measures that promise to aid in its very great reduction, and to emphasize particularly that our present-day armamentarium is adequate for accomplishing this end. It but remains for the better and more effective application of these recently acquired techniques. Statistics indicate that a satisfactory trend in the proper direction has already begun. That this trend should continue to its legitimate conclusion is an impelling challenge to surgery and surgeons of today

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The College in National Affairs

MELVIN A. CASBERG, M.D., F.A.C.S., has resigned as assistant secretary of defense, effective January 1, 1954. He is succeeded by Frank B. Berry, M.D., F.A.C.S.

In 1951 Dr. Casberg left his position as dean of the medical school of St. Louis University to succeed Richard L. Meiling, M.D., F.A.C.S., as chairman of the Armed Forces Medical Policy Council. This latter agency was discontinued in 1953 when Dr. Casberg was appointed an assistant secretary of defense. He is now resuming the practice of surgery in California.

Dr. Berry has been clinical professor of surgery at Columbia University since 1946. His initial appointment, until Congress acts, is as assistant to the secretary of defense.

The following Fellows have accepted appointments to the Medical Task Force of the Commission on Organization of the Executive Branch of the Government (the new Hoover Commission): Edward D. Churchill, Michael E. DeBakey, Evarts A. Graham, and Paul R. Hawley.

Three Films Receive Awards

AWARDS to three outstanding motion pictures were made at the 1953 Convocation in Chicago.

From the Surgical Film Exhibition, long an important feature of each Clinical Congress, the film entitled *Patent Ductus Arteriosus, Clinical Considerations* received the certificate of merit. In two parts, one being physiology and diagnosis, the other surgical treatment, this film was made by George H. Humphreys II, M.D., F.A.C.S., New York.

Malrotation of the Bowel, Diagnosis and Surgical Treatment is the film from the Motion Picture Symposium on Unusual Lesions in Abdominal Surgery to which an award went. It is the work of Willis J. Potts, M.D., F.A.C.S., Chicago.

To R. Kennedy Gilchrist, M.D., F.A.C.S., Chicago, went the third award, for his film entitled *Surgical Treatment of Diverticulitis of the Sigmoid.* This was made for the Ciné Clinic.

A special committee appointed for the time of the Congress chose the above films.