Intraabdominal Hypertension

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Uncorrected intraabdominal hypertension can lead to multisystem organ failure. Most often the cardiac, respiratory, and renal systems are involved. This author describes the physiologic factors that increase intraabdominal pressure. Treatment approaches (both medical and surgical) are discussed.

The perioperative care of the critically injured patient has improved significantly over the last 10 years. Many patients who previously would have succumbed to their injuries are now being saved. Advancements in invasive monitoring, enteral nutrition, and management of infectious diseases are partially responsible for these changes. As trauma patients survive longer in the intensive care unit (ICU), we are faced with new challenges in critical care management. Despite our current best efforts, some patients still die after prolonged hospitalization in the ICU; the major causes of death are sepsis and multisystem organ failure. As we progress into the 1990s, we must address these problems in an increasingly older population. Multisystem organ failure is more likely to develop in this population, whose physiologic reserve is already limited.

Intraabdominal hypertension, which may lead to multisystem organ failure, has been investigated for many years; however, its recognition and treatment as a distinct clinical syndrome have been described only recently.[1-4] Intraabdominal hypertension is an abnormal increase in intraabdominal pressure (IAP) of sufficient magnitude to initiate some physiologic dysfunction. Most commonly, the cardiac, respiratory, and renal systems are involved.

Etiology

Numerous etiologic factors have been described for the development of intraabdominal hypertension.[2,5-9] Hemoperitoneum is the most common initial presentation in traumatized patients in whom intraabdominal hypertension develops. Most patients have either a significant intraabdominal injury or celiotomy. Typically, the patient received a solid organ or major vessel injury that required emergency surgery. Large volumes of crystalloids and blood products were necessary for resuscitation. At the completion of surgery, the patient may be hypothermic and coagulopathic. Adequate rewarming and infusion of blood components usually correct the coagulopathy, and no further problems develop. However, some patients do not respond to treatment and continue to have diffuse capillary bleeding after abdominal closure. These patients are at risk for the development of intraabdominal hypertension. After surgery, these patients require continuous fluid and blood product administration. If the blood does not clot, it will continue to accumulate in the peritoneal cavity, causing intraabdominal hypertension. Uncorrected intraabdominal hypertension contributes to multisystem organ failure.

Large retroperitoneal hematomas and consequent edema are contributing sources of intraabdominal hypertension. Principal causes of retroperitoneal bleeding are significant pelvic ring disruptions and major parenchymal lesions of the kidneys. Pelvic ring disruptions may be life threatening depending on the magnitude of blood loss. The retroperitoneum may hold several liters of undetected blood, which acts synergistically with other sources to increase intraabdominal hypertension. Renal parenchymal injuries, in addition to generating retroperitoneal hematomas, may make the kidney more susceptible to the effects of intraabdominal hypertension.[10]

A third cause of acute intraabdominal hypertension is pneumoperitoneum. ICU patients in whom respiratory failure and secondary restrictive lung disease have developed frequently have high airway pressures. These patients are often subjected to high levels of positive end-expiratory pressure (PEEP) to maintain adequate oxygenation. Barotrauma is a well-recognized complication of high airway pressures and possibly high levels of PEEP. Sepsis will increase the patient's susceptibility to barotrauma even further by increasing extravascular lung water, decreasing lung compliance, and increasing airway pressures. Sepsis may disrupt the lung parenchyma, allowing barotrauma to occur at lower pressures. Barotrauma commonly manifests as a pneumothorax or pneumomediastinum; a less common presentation is pneumoperitoneum (Macklin phenomenon).[11] Air that has dissected along the bronchial airways and vessels to the mediastinum may eventually erupt into the peritoneal cavity, resulting in a pneumoperitoneum.

Other causes of intraabdominal hypertension arise more insidiously. Ascites is a well-recognized cause of intraabdominal hypertension in patients with cirrhosis, other forms

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of liver disease, and certain malignancies and in those undergoing peritoneal dialysis.[8] Factors accounting for the formation of ascites in the trauma patient include large volumes of resuscitation fluid, negative nitrogen balance, positive pressure ventilation, and sepsis. Large amounts of resuscitation fluids eventually sequester in the interstitial space of the abdominal wall and as free fluid in the peritoneal cavity. Severe multisystem trauma leads to a large negative nitrogen balance and a subsequent decrease in serum proteins. This decrease in colloid oncotic pressure causes edema and ascites. Increased positive pressure ventilation increases mesenteric venous pressure and capillary pressure and eventually generates ascites. With sepsis, increased capillary membrane permeability permits fluid to leak into the peritoneal cavity. In contrast to hemoperitoneum and pneumoperitoneum, which develop over the course of hours, the intraabdominal hypertension associated with ascites develops over several days. The same factors that lead to ascites also cause changes in interstitial fluid and abdominal wall compliance.

Interstitial visceral edema is influenced by infusion of large volumes of fluid, poor nutritional states, and sepsis. This interstitial edema leads to an increased volume of the abdominal viscera. IAP increases as the intraabdominal organs swell and expand. This effect is magnified by a corresponding decrease in abdominal wall compliance.

Normally, abdominal wall compliance allows a significant amount of ascites or blood to accumulate within the peritoneal cavity without increasing IAP. Decreased plasma proteins and increased capillary membrane permeability lead to the extravascular migration of fluid and cause edema in the interstitial spaces of the abdominal wall. Abdominal wall compliance decreases as interstitial fluid increases. With decreased compliance, small accumulations of blood or fluid within the abdominal cavity may significantly increase the IAP.

Patients often have a combination of these factors that lead to intraabdominal hypertension. After surgery, a patient frequently will have some residual fluid in the abdomen, which increases with subsequent "third-spacing" of resuscitation fluids. This problem is compounded if the patient has a small retroperitoneal hematoma as a result of a pelvic ring disruption, which enlarges over the next several hours. Over the next few days, an intestinal ileus develops, interstitial fluid accumulates, and increasing amounts of positive pressure ventilation are required.[1,3] Singularly, none of these factors creates intraabdominal hypertension; however, a combination of factors may give rise to significant risk for its development.

Pathophysiology

Cardiac

The physiologic consequences of increased IAP have been studied for several years. Much of the basic science foundation was derived from animal studies, usually on dogs.[7,9,12-15] The literature on human subjects is generated from studies of chronic causes of intraabdominal hypertension such as ascites, ovarian masses, and abdominal tumors.[8] Studies on acute causes have been published only recently, and most are based on diagnostic laparoscopy. Minimal data are currently available on the physiologic effects of intraabdominal hypertension in posttraumatic injuries (see box below).[1]

Dramatic changes in cardiac output can occur with intraabdominal hypertension according to the formula: Cardiac output = Heart rate × Stroke volume. Most patients have a mild tachycardia that does not change statistically with further increases in abdominal pressure. Stroke volume, which depends on the basic components of preload, afterload, and contractility, does undergo significant changes with intraabdominal hypertension. The major effects are on preload and afterload, with some indirect influence on contractility. Preload refers to the volume of blood within the left ventricle at the end of dias-

Physiologic manifestations of intraabdominal hypertension

Cardiovascular	↓	Cardiac output	
	↑	Central venous pressure	
	↑	Systemic vascular resistance	
	↑	Pulmonary artery pressure	
	↑	Pulmonary artery occlusion pressure	
Pulmonary	↑	Dead space	↓ Compliance
	↑	$\dot{Q}s/\dot{Q}t$	↑ Peak airway pressure
	↓	Pao_2/Fio_2	↑ Mean airway pressure
Renal		Oliguria	
	↓	Creatinine clearance	
	↓	Urine/plasma osmolality	
	↑	Serum creatinine	

↓ Decreased ↑ Increased

tole (LVEDV). Technically, LVEDV largely determines the degree of stretching of myocardial muscle fibers before their contraction (Starling's law).[16] Clinically, the LVEDV is estimated by measuring the pulmonary artery occlusion pressure (PAOP) in a patient with normal left ventricular compliance. Left ventricular compliance decreases in patients with intrinsic heart disease, high pleural pressures, and large transdiaphragmatic pressures.[15]

Return of blood to the right side of the heart is the major determinant of preload, and venous return to the heart is regulated by the mean systemic pressure and the venous resistance. Mean systemic venous pressure is the pressure within the capacitance vessels—small veins, venules, and capillaries. Flow through the venous system is driven by the difference between the mean systemic pressure and the right atrial pressure and is regulated by venous resistance. Venous resistance to the flow of blood occurs in the larger veins and vena cava. Kashtan et al.[7] studied the effects of intraabdominal hypertension on venous return to the heart in dogs. They found that venous resistance and mean systemic venous pressure increased with increasing IAP. The respective rise in these pressures determined if venous return to the heart was augmented or reduced. The patient's volume status is the critical variable. Patients with an inadequate preload have a rapid decline in venous return with increasing abdominal pressure. Those with an adequate preload initially have augmented venous return with increasing abdominal pressure. Regardless of the patient's volume status, intraabdominal hypertension eventually decreases venous return. Kashtan et al. explained that, in all cases, increasing IAP led to increased mean systemic venous pressure. Increased pressure occurred with compression of splanchnic vessels. The rise in venous resistance in intraabdominal hypertension was caused by compression of the vena cava. In the hypovolemic patient, a relatively small increase in IAP may sufficiently compress the vena cava and decrease venous return. In contrast, a hypervolemic patient will initially have an increased venous return. The inferior vena cava (IVC) will resist compression as long as the IVC pressure exceeds the intraabdominal pressure. Until compression of the IVC takes place, increasing IAP will increase mean systemic venous pressure without a marked rise in venous resistance. This will result in increased venous return to the heart and a subsequent increase in cardiac output.

Afterload is the resistance to ventricular ejection. Clinically, systemic vascular resistance (SVR) is the main determinant of afterload. A relationship exists between the SVR and cardiac output as follows: SVR = [(Mean arterial pressure − central venous pressure)/Cardiac output] × 80. In adequately volume-loaded patients who have normal cardiac contractility, increased SVR generally reflects increases in arteriolar resistance. Under these circumstances, pharmacologically decreasing SVR may increase the cardiac output. Studies on intraabdominal hypertension demonstrate that increasing IAP corresponds with increased SVR.[12,17] This increase in SVR is probably the result of increased arteriolar resistance. Without a significant change in blood pressure, increasing systemic vascular resistance will decrease cardiac output.

Increases in SVR are not uniform across all vascular beds within the abdomen.[12,17] Barnes et al.[12] measured visceral blood flow and vascular resistance in dogs with intraabdominal hypertension. Increasing the IAP from 0 to 40 mm Hg raised resistance in the celiac, superior mesenteric, femoral, and renal arteries by 26%, 70%, 103%, and 136%, respectively. Three factors responsible for the increased SVR in intraabdominal hypertension are vascular compression, myogenic reflexes, and vasoactive hormones.

The splanchnic vasculature possesses a myogenic reflex whereby increases in venous pressure, by direct compression of the smaller veins, lead to a reflex arterial vasoconstriction.[12] Vasopressin and angiotensin may be released in intraabdominal hypertension.[18] These vasoactive substances are potent vasoconstrictors. A decrease in transcardiac pressure, caused by increased pleural pressure, may lead to reflexive arterial vasoconstriction.[12]

Contractility refers to the force and velocity of ventricular contraction. Metabolic changes that occur as a result of intraabdominal hypertension can alter the contractile state of the myocardium. Acidosis and hypoxemia impair contractility and the heart's ability to respond to catecholamines. Lactic acidosis develops when blood is shunted away from the abdomen, decreasing tissue perfusion and increasing anaerobic metabolism. Tissue perfusion is further impaired by an absolute decrease in cardiac output caused by increased arteriolar resistance. These patients often have sustained a primary lung injury, which can impair pulmonary compensation for metabolic acidosis. Intraabdominal hypertension itself can cause arterial hypoxia and hypercarbia and further impair the pulmonary compensatory process. In this manner, intraabdominal hypertension may indirectly decrease the contractile state of the myocardium.

Pulmonary

Pediatric surgeons have known of the respiratory complications of intraabdominal hypertension for many years.[19,20] Attempts to primarily close gastroschisis have resulted in respiratory failure. The effects of intraabdominal hypertension on the pulmonary system are caused by an increase in pleural pressure and a decrease in thoracic volume. About 20% of the IAP is transmitted into the thorax through the dia-

phragm.[12] Beyond IAPs of 20 mm Hg, peak airway pressures rise markedly.[9,12] The relationship is not linear but can still be used to estimate the degree of intraabdominal hypertension.

The loss of lung volume causes a collapse of compliant alveoli and a decrease in functional residual capacity. This change in alveolar ventilation results in hypercarbia and arterial hypoxemia. As intrathoracic pressure increases, direct compression of the pulmonary vasculature occurs. Compression of the pulmonary vessels increases pulmonary vascular resistance and exacerbates a ventilation-perfusion mismatch.

Renal

Most of the data on the renal consequences of intraabdominal hypertension are derived from dog models. Controlled intraabdominal hypertension is created either by surgically implanting inflatable bladders or by instilling fluid into the peritoneal cavity. Harman et al.[6] showed that as IAP increased from 0 to 20 mm Hg, the glomerular filtration rate decreased by 25%. When IAP was increased to 40 mm Hg, the dogs developed anuria. The fivefold increase in renal vascular resistance as IAP is increased from 0 to 20 mm Hg increases to 15-fold when the IAP rises to 40 mm Hg. Maintenance of adequate preload and cardiac output twice the baseline were unable to prevent this decrease in glomerular filtration rate. Caldwell and Ricotta[21] presented conflicting data on the relationship of cardiac output to renal failure. In their studies on mongrel dogs, they measured cortical and medullary renal blood flow and hemodynamic parameters during increases in abdominal pressure. Oliguria developed with increasing IAP. Their results showed a direct correlation between renal blood flow and cardiac output. Measurement of renal venous pressure during these studies indicated a 1 to 2 mm Hg greater pressure in the renal veins than in the inferior vena cava. Increases in renal venous pressure led to an immediate specific reflex of renal artery vasoconstriction.[13] LeRoith et al.[18] noted that increased IAP leads to a corresponding increase in serum antidiuretic hormone levels in dogs. Inferior vena cava pressures correlated almost 100% with IAP. Vaughan et al.[22] postulated that direct compression of the ureters had minimal influence on the development of renal failure because the renal collecting system is able to generate up to 90 mm Hg pressure. Dogs subjected to intraabdominal hypertension with ureteral stents had the same changes in renal function as unstented dogs.

The exact mechanism for the renal dysfunction caused by intraabdominal hypertension is not known. Renal blood flow decreases as cardiac output decreases, particularly in the patient with hypovolemic trauma; therefore, decreases in cardiac output are likely to be significant in the mechanism of oliguria. Oliguria with intraabdominal hypertension may also occur when the cardiac output is normal or supranormal. Caldwell[17] suggested an alternate hypothesis for oliguria with intraabdominal hypertension. The filtration gradient (FG) is the mechanical force across the glomerulus that results in urine production.[23] This measure is the difference between glomerular filtration pressure and proximal tubular pressure.[19] Compression of the renal pelvis naturally occurs in the traumatized patient with intraabdominal hypertension. The proximal renal tubular pressure will approximate IAP in this patient. An estimation of glomerular filtration pressure is the difference between the mean arterial pressure (MAP) and the IAP. The FG across the glomerulus is estimated as: FG = MAP − 2(IAP). For example, if a patient's IAP reaches 40 mm Hg and the MAP is 80 mm Hg, the resulting force across the glomerulus is zero. In this model one can easily see why most patients become anuric at IAPs of 40 mm Hg. Surveillance of MAP, IAP, and urine output allows estimation of FG. This calculation is useful in monitoring the results of medical management and determining the need for surgical decompression.

Diagnosis

The normal IAP in humans is approximately zero. Following celiotomy, average IAPs are 3 to 15 mm Hg.[21] The most accurate technique to determine IAP is to measure it directly with an indwelling peritoneal catheter.[20] A safer method is to monitor IVC pressures through a long femoral venous catheter. IVC pressures correlate almost 100% with pressures measured by direct peritoneal catheters. A technique that involves minimal invasiveness yet correlates highly with IAP is measurement of intravesicular pressure. This technique is based on the property that the adult urinary bladder behaves as a passive diaphragm with bladder volumes between 50 and 100 ml.[21] Intravesicular pressure measurements correlate with IAPs of 5 to 50 mm Hg. Kron[2] has described the method used at the Maryland Institute for Emergency Medical Services Systems Shock Trauma Center. Fifty to 100 ml of sterile saline solution is injected into the empty bladder through an indwelling Foley catheter. The sterile tubing of the urinary drainage bag is cross clamped just distal to the culture aspiration port. The end of the drainage bag tubing is connected to the indwelling Foley catheter. The clamp is released enough to allow the tubing proximal to the clamp to fill with fluid from the bladder and then is reapplied. A 16-gauge needle is used to Y-connect a central venous pressure (CVP) manometer or pressure transducer through the culture aspiration port of the tubing to the drainage bag. The top of the symphysis pubis bone is used as the zero point with the patient in the supine position.

In pediatric patients, intragastric

pressures are thought to correlate well with IAPs. Measurements are made in a similar fashion as the intravascular pressures. A single-lumen nasogastric tube is inserted into the stomach. After the instillation of saline solution into the tube and stomach, the nasogastric tube is connected to either a CVP manometer or a pressure transducer. The correlation between stomach pressures and IAP is not as good in adults as in children. Several factors may account for this. The stomach behaves less like a closed system than the bladder. Fluid instilled into the adult stomach may leak out of the pylorus or even up the esophagus. In the presence of hiatal hernias, intragastric pressure will be influenced by intrathoracic pressure. Other techniques such as measuring various arterial, superior vena cava, right atrial, airway, rectal, and rectus sheath pressures have failed to show significant clinical correlation.[13]

Treatment

A major difficulty in treating intraabdominal hypertension is its recognition. Most cases of intraabdominal hypertension probably go undetected because the diagnosis is not entertained. Failure to recognize this entity can ultimately result in multisystem organ failure. Once the diagnosis is considered, measuring intravesicular pressures is a fairly simple technique. Because intraabdominal hypertension may develop over the course of several hours or days, repeated measurements of IAP are necessary to diagnose it.

Techniques available to help prevent intraabdominal hypertension include early and aggressive use of warmed blood products in the severely injured patient to abort coagulopathy, angiographic embolization for rapidly expanding retroperitoneal hematomas caused by pelvic ring disruptions, and tube decompression to relieve pneumoperitoneum. Ascites formation can be reduced by colloids and the judicious use of diuretics. Some have recommended drainage of ascites in cases where intraabdominal hypertension is known to result from sterile ascites.[8] Achievement of positive nitrogen balance will prevent unnecessary protein erosion. Intravenous lines are a common source of occult bacteremia in ICU patients; septic foci should be sought and treated vigorously (see Rosenthal and Joshi, page 323, and Smith and Joshi, page 422).

Adequate resuscitation is the first step in the management of any critically ill patient. Estimating the adequacy of fluid resuscitation may be difficult. Increasing IAP can elevate CVP, and falsely elevated CVP readings may not reflect cardiac preload volume accurately. Underlying pulmonary complications combined with increasing pleural pressures increase pulmonary artery pressures, which must be considered in the interpretation of pulmonary catheter measurements. Absolute pulmonary artery and pulmonary artery occlusion pressures may not reflect the patient's true volume status, especially if airway pressure is elevated significantly. Trends in hemodynamic pressures in response to volume challenges are probably more important than the absolute number. Urinary output is important: decreased urinary flow may be the result of excessive IAP or inadequate resuscitation. Oliguria will develop in all patients, regardless of their cardiovascular status, when the IAP becomes large enough. A patient with an adequate urinary output is less likely to be underresuscitated.

We evaluate these difficult patients by measuring the cardiac output using the indocyanine green dye technique.[24] Following the injection of a known bolus of green dye into the central venous system, the appearance of the dye in the peripheral arterial system is recorded. The dye's appearance and the shape of the curve are used to calculate the cardiac output and dispersive volume of the lungs, in addition to other variables. The central dispersive volume of the lungs calculated by this method has been shown experimentally to be within 10% of the direct measurement of pulmonary blood volume.[24] The advantage of this method is that CVP and PAOP are pressure measurements of preload status, whereas the green-dye method yields a volume measurement of the preload status. When CVP and PAOP are elevated significantly, we have found the green-dye method to provide a superior estimation of the patient's preload volume.

Volume status in the patient with intraabdominal hypertension is critical. Small losses in volume can have disastrous consequences on venous return. Flow through the vena cava becomes dependent on its patency, and venous volume is the major force resisting collapse of the inferior vena cava. Fluids, generally colloids, should be used to maintain intravascular volume during resuscitation, including periods of critical illness.

Medical management

Not all patients with intraabdominal hypertension require surgical decompression; some can be managed medically. Patients to be considered for nonsurgical therapy must be observed closely. Their abdominal pressures must be less than 40 mm Hg. Physiologic derangements should be minor and relatively stable. Volume resuscitation should be adequate and reassessed frequently. In addition to volume resuscitation, other techniques to increase the cardiac output are helpful in the management of these patients.[1-4]

To improve cardiac output, inotropic support should be used. A combination of agents is commonly administered. Low-dose (2 to 3 μg/kg/min) dopamine is often started not only for its inotropic effects but also for its ability to increase splanchnic and renal blood flow. The effect is mediated by dopaminergic receptors in the renal vasculature. Dobutamine can then

be added if low-dose dopamine is unsatisfactory in raising the cardiac output. Dobutamine will tend to lower the systemic vascular resistance while increasing the cardiac output.

Lowering the systemic vascular resistance is a third method for increasing the cardiac output. Sodium nitroprusside or other short-acting intravenous vasodilators may be used. Vasodilators will reduce visceral SVR caused by arteriolar vasoconstriction but not that caused by direct venous compression. Therefore, extraabdominal vasculature may vasodilate to a greater extent than the abdominal visceral arterioles. This vasodilation could result in shunting blood away from the intraabdominal viscera, which could increase acidosis and worsen tissue hypoxia. For this reason, vasodilator therapy should be avoided in patients without adequate volume loading.

Cardiac output, volume status, and oxygen consumption should be determined frequently after each intervention. Medical management may continue as long as the patient remains stable or is improving. Any sign of deterioration should be treated by surgical decompression and release of the intraabdominal hypertension.

Surgical management

Surgical decompression is required in those patients with initial IAPs greater than 40 mm Hg. Decompression is also helpful in patients with IAPs between 25 and 40 mm Hg not responding to medical therapy. The goal of surgery is to decrease the IAP by increasing abdominal capacitance. Before reoperation, a reasonable attempt should be made to correct any preexisting coagulopathy. Volume loading and cardiac status should be optimized with inotropes and possibly vasodilators. Increased patient acuity may not permit transport to the operating room. In these instances, opening the abdominal incision at the bedside has resulted in dramatic improvements in the patient's condition.

Table I. *Changes with surgical decompression of intraabdominal hypertension*

	Preoperative	Postoperative
Respiratory		
Pao_2/Fio_2	84	376
Peak airway pressure	42 cm H_2O	26.0 cm H_2O
Mean airway pressure	23 cm H_2O	15.6 cm H_2O
PEEP	16 cm H_2O	8 cm H_2O
Renal		
Urine output	10-20 ml/hr	300 ml/hr
Creatinine clearance	42 mg/dl	93 mg/dl
Cardiovascular		
Heart rate	118 beats/min	128 beats/min
Cardiac output	8.5 L/min	11.5 L/min
Stroke volume	70 ml	90 ml
SVR index	1510 dyne · sec · cm ^{5} · m^2	864 dyne · sec · cm ^{5} · m^2
$\dot{V}o_2$ index	115 ml/min/m^2	153 ml/min/m^2

Fio_2, Fraction of inspired oxygen; $\dot{V}o_2$, oxygen consumption per unit time.

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The following example demonstrates a recent case of intraabdominal hypertension following blunt trauma. A 69-year-old woman was involved in a motor vehicle accident. The initial assessment and resuscitation were uneventful. The patient was stable in the admitting area, and the only identified injuries were several right-sided rib fractures and superficial abrasions. A computed tomographic scan of the abdomen revealed a moderate right retroperitoneal hematoma. Angiography demonstrated an avulsed right lumbar artery that was subsequently embolized. The right kidney had a normal angiographic picture. The patient was observed in the ICU.

The patient required 6 units of blood over the next 24 hours to maintain a hemoglobin level of about 10 gm/dl. She required increasing respiratory support. The chest x-ray film was clear but showed bilateral elevated diaphragms. Over 5 L of fluid and inotropic support were required to maximize oxygen consumption. Urinary output was maintained about 1 ml/kg/hr.

In the second 24 hours, the patient required additional supportive measures including afterload reduction. She showed no signs of ongoing blood loss. Intravesicular pressures on the morning of the second day were 30 to 32 cm H_2O. Later that day, IAP had risen to 38 cm H_2O. Urinary output suddenly decreased to less than 20 ml/hr. The patient was taken to surgery for operative decompression of the abdomen.

Surgery resulted in dramatic improvements in all physiologic parameters (Table I). The excess clot was removed and a right nephrectomy was performed. (The ureteropelvic junction was completely disrupted and could not be repaired because of the patient's condition.) Retroperitoneal hemostasis was secured. The skin was closed and the fascia left open without a measured increase in IAP. Except for a course of postoperative pneumonia, the patient recovered without difficulty.

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Following decompression, improvements usually occur in all organ systems.[1-4] Cardiac function returns to normal or supranormal values. Systemic vascular resistance decreases significantly. In a hypovolemic patient, a rapid fall in arteriolar resistance and increase in venous capacitance may cause a precipitous fall in the blood pressure. Patients may require small doses of

vasoconstrictors to maintain vascular tone in addition to volume therapy. Venous return to the heart also returns to normal values. Patients with markedly elevated IAPs may develop a transient acidosis at the time of reperfusion.

In a review of our trauma patients who have intraabdominal hypertension, pulmonary mechanics show an equally prompt response to intraabdominal decompression. Peak and mean airway pressures fall by 19% and 23%, respectively. Pao_2 increases by 137% and the $Paco_2$ decreases by 24%. The ratio of Pao_2 to fraction of inspired oxygen (Fio_2) improves over 200%. Pulmonary shunt decreases 32%. Static lung compliance is increased by 82%.

Urinary flow will usually increase while the patient is still on the operating table. Oliguric renal failure often converts to high-output renal failure. Serum creatinine level gradually falls as creatine clearance improves. Survivors are expected to restore near normal renal function.

During celiotomy, all old blood clots should be evacuated and the peritoneal cavity lavaged. Intraabdominal septic foci should be sought. Any necrotic tissue must be removed. Good surgical hemostasis should be attempted before closure. Drains may be used if coagulation defects persist. When hemoperitoneum leads to intraabdominal hypertension, evacuating the clots may provide enough space to close the abdomen satisfactorily. During closure of the abdomen, pulmonary artery, peak airway, and intravesicular pressures must be monitored. A return of elevated values indicates that with abdominal closure intraabdominal hypertension will recur. In the majority of cases in which this occurs, fascial closure is not possible.

Several options are available when the fascia cannot be closed. Generally we have preferred to place a fascial bridge to prevent an abdominal wall hernia (Fig. 1). Commonly a polytetrafluoroethylene patch is used. The graft is sewn to the fascia with monofilament sutures. Tissue edema has usually prevented closure of the skin over the patch. Without skin covering, fluid losses through the wound can be considerable. We have chosen to cover the wound with a sterile occlusive plastic drape. Drains are placed between the polytetrafluoroethylene patch and the drape to assess fluid losses. These wounds must be monitored carefully for any signs of infection. Frequent

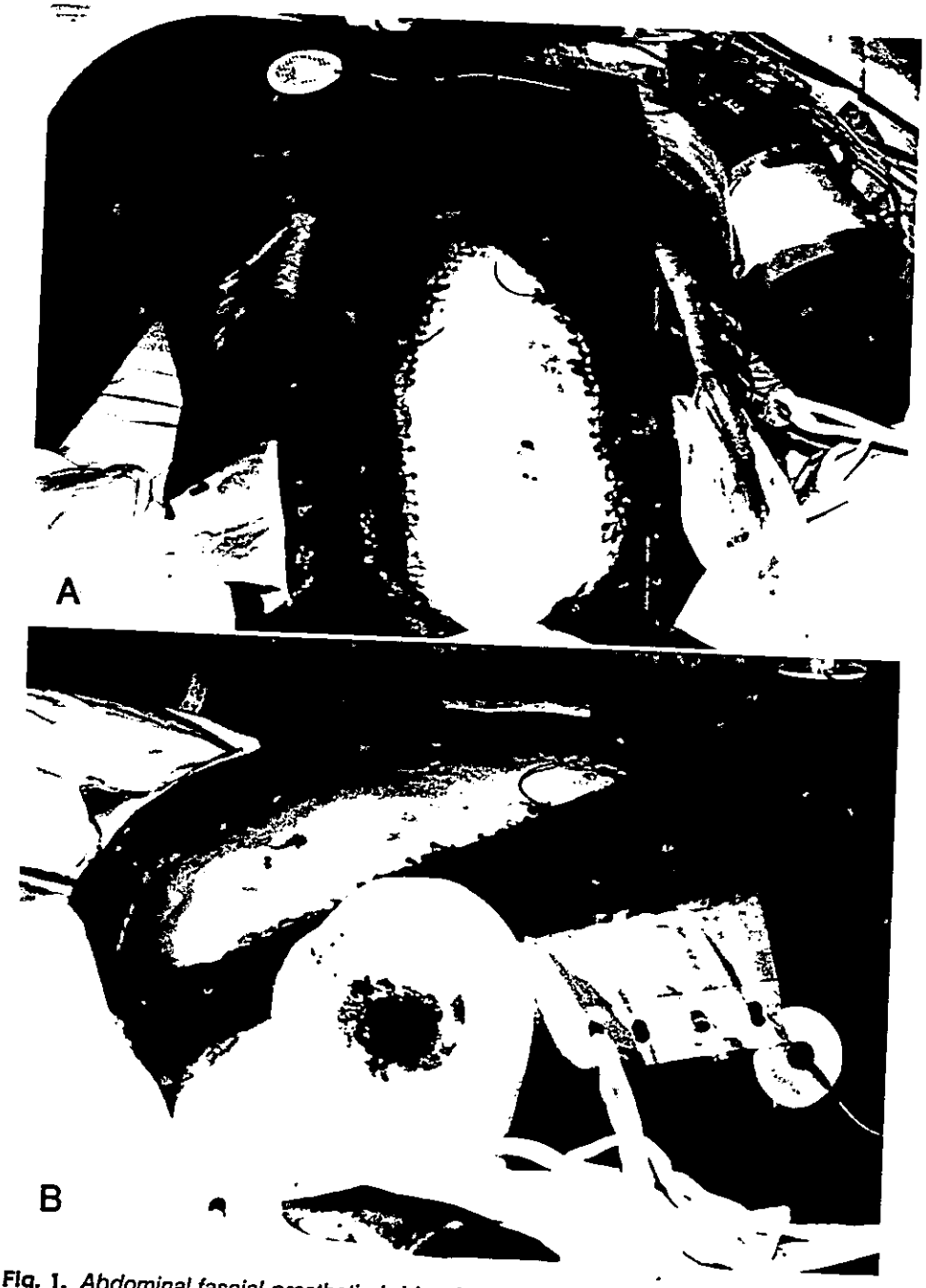

Fig. 1. *Abdominal fascial prosthetic bridge for intraabdominal hypertension. A. Polytetrafluoroethylene patch is secured to fascia with a running monofilament suture. B. Colostomy exits at lateral to wound.*

changes of the drape under aseptic conditions are required several times a week. Surveillance wound cultures are usually taken at that time.

A few patients who required decompression at the bedside were managed by leaving the fascia open without a bridge. In these cases, the abdominal viscera were well adhered into the peritoneum. The wounds were dressed with sterile saline gauze and then covered with a plastic drape. Eventually these patients must be brought back to the operating room for repair of their abdominal wall hernia.

Summary

Intraabdominal hypertension is a potentially correctable cause of multisystem organ failure in the severely injured patient. As in all areas of trauma surgery, a high index of suspicion is necessary to make the diagnosis. When sought, intraabdominal hypertension can be diagnosed early and treated successfully. Selected patients can be managed by aggressive nonoperative therapy that includes preload and afterload manipulation and inotropic support. Surgical therapy is effective in decompressing intraabdominal hypertension. The major difficulty after operative decompression is abdominal wall closure. Patients who survive their acute illness may require subsequent fascial reconstruction, but this "cost" is worth the effort to mitigate against multisystem organ failure.

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