The Measurement of Intra-abdominal Pressure as a Criterion for Abdominal Re-exploration

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Acute elevation of intra-abdominal pressure above 30 mmHg caused oliguria in 11 postoperative patients. Operative re-exploration and decompression in seven patients resulted in immediate diuresis. Four patients who were not re-explored developed renal failure and died. If intra-abdominal pressure rises above 25 mmHg in the early postoperative period and is associated with oliguria and normal blood pressure and cardiac index, the patient should undergo re-exploration and decompression of the abdomen.

INCREASED INTRA-ABDOMINAL PRESSURE causes inferior vena caval compression resulting in decreased cardiac filling pressure and low cardiac output.[1-3] Chronic ascites may have minimal circulatory effects.[4] It has been our observation that an acute elevation of intra-abdominal pressure in the early postoperative period causes a marked impairment of renal function independent of blood pressure or cardiac output. We recently reported a laboratory study which confirmed this hypothesis.[5] The purpose of this report is to present a group of patients who became oliguric or anuric due to elevation of intra-abdominal pressure. There was prompt resolution of the compromised renal function when the abdomen was decompressed at re-exploration. In order to determine objectively the level of intra-abdominal pressure necessary to impair renal function, a method of direct measurement has been developed using an indwelling Foley catheter. A correlation between intra-abdominal pressures above 25 mmHg and renal dysfunction has been demonstrated.

Elevated IAP: Early Observations

Four patients were observed prior to our laboratory study and prior to development of a method for the clinical measurement of intra-abdominal pressure. These patients illustrate our basic hypothesis.

Case 1. A 65-year-old obese man was admitted with a leaking abdominal aortic aneurysm (AAA). He underwent prompt operative repair and maintained normal urinary output in the early postoperative period. On the third postoperative day, he developed abdominal distension from recurrent hemorrhage. The bleeding was allowed to "tamponade," and, despite a mean blood pressure of 100 mmHg, cardiac index of 3.0 L/min/M^2, and a pulmonary capillary wedge pressure of 20 mmHg, he progressed to anuria and ultimately died from renal failure and infection.

Case 2. A 28-year-old man with Marfan's syndrome underwent repair of a thoracoabdominal aneurysm. He maintained adequate urinary output (100 ml/hr) until 8 hours after surgery, when he developed abdominal distension and oliguria (urinary output 10 ml/hr). Mean BP was 96 mmHg, PCW was 15 mmHg, CI was 4.0 L/min/M^2. Despite the administration of diuretics and dopamine, urinary output declined. The patient was returned to the operating room where the abdomen was re-explored and decompressed. Immediately, urinary output increased to 200 ml/hr, and hemodynamic measurements remained unchanged (CI 4.1 L/min/M^2, PCW 12 mmHg). He maintained normal renal function and was discharged 2 weeks after surgery.

Case 3. An 81-year-old man presented with a ruptured infrarenal AAA and hypotension, but 8 hours following repair, he developed abdominal distension and his urinary output decreased to 10 ml/hr despite a mean BP of 104 mmHg and PCW of 14 mmHg. There was no renal response to loop diuretics. The patient was returned to the operating room for abdominal re-exploration and decompression. Diuresis was immediate (urinary output 350 ml/hr) with no significant change in his hemodynamic measurements. He made an uneventful recovery.

Case 4. A 63-year-old man underwent repair of an expanding infrarenal AAA. He did well until 12 hours after operation, when oliguria (10 ml/hr urinary output) developed despite the use of dopamine and diuretics. Abdominal distension developed, but the hemodynamic measurements (mean BP 90 mmHg, PCW 15 mmHg) were satisfactory. Abdominal exploration and decompression was performed. Urinary output immediately increased to 120 ml/hr. The patient was discharged 10 days later.

These cases led us to the hypothesis that a rapid elevation of intra-abdominal pressure could cause acute renal insufficiency. Normal hemodynamic measurements and the administration of dopamine and loop diuretics did not reverse the oliguria. Abdominal decompression caused immediate improvement in renal function even if no definite bleeding site was found. In the one patient who was not re-explored, renal failure persisted until death. We then performed a laboratory study to test the hypothesis.

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Laboratory Findings

Experiments in our laboratory demonstrated that dogs subjected to an intra-abdominal pressure of 20 mmHg suffered a 75% reduction in glomerular filtration rate and renal blood flow. This impairment in renal function was not corrected by the augmentation of cardiac output. The pathophysiology was a severe rise in renal vascular resistance, most likely related to direct renal parenchymal compression.[6] In the laboratory we developed a technique of easily measuring intra-abdominal pressure. We were able to test and use this technique clinically.

Technique of Measurement of IAP

The wall of the urinary bladder behaves as a passive diaphragm when the bladder volume is between 50 and 100 ml. Pressure measurements recorded simultaneously through a transurethral urinary bladder catheter and a peritoneal dialysis catheter were equal during several infusions of peritoneal dialysis solution. This unity is confirmed for pressures ranging from 5 to 50 mmHg and verifies that intra-abdominal pressure can be accurately measured through an indwelling urinary bladder catheter. This technique is now employed routinely in our surgical intensive care unit, and it appears that intra-abdominal pressure averages 3–15 mmHg after abdominal surgery (Table 1).

Method

1. Sterile saline (50–100 cc) is injected in the empty bladder through the indwelling Foley catheter.
2. The sterile tubing of the urinary drainage bag is cross-clamped just distal to the culture aspiration port.
3. The end of the drainage bag tubing is connected to the indwelling Foley catheter.
4. The clamp is released just enough to allow the tubing proximal to the clamp to flow with fluid from the bladder, then reapplied.
5. A 16-gauge needle is then used to Y-connect a cup monometer or pressure transducer through the culture aspiration port of the tubing to the drainage bag.
6. The top of the symphysis pubis bone is used as the zero point with the patient supine.

Correlation of Intra-abdominal Pressure Measurement with Renal Insufficiency

Intra-abdominal pressure measurements were made in 10 patients during the past 18 months and correlated with their clinical course. Three patients who had undergone abdominal aortic procedures developed signs of intra-abdominal blood loss with normal blood pressure, cardiac output, and adequate urinary output (>1 ml/kg/hr). Intra-abdominal pressure measurements ranged from 10 to 15 mmHg in these patients. They were observed and transfused as necessary. All recovered without abdominal exploration, and none developed renal insufficiency.

TABLE 1. *Random Intra-abdominal Pressures Measured During the First 24 Hours Following Elective Surgical Procedures*

Procedure	Age (years)	IAP (mmHg)*
Aorto-bifemoral bypass	56	6
Gastrectomy	46	7
Abdominal aortic aneurysm resection	73	13
Femoral-popliteal bypass	75	3
Aorto-bifemoral bypass	53	13
Pulmonary lobectomy	71	6
Pulmonary decortication	64	3
Low anterior colon resection	71	10
Abdominal aortic aneurysm resection	75	9
Abdominal-perineal resection	61	4

* Reference point for zero is the symphysis pubis with the patient supine.

Seven patients had marked elevation of IAP (>25 mmHg) and all manifested renal insufficiency (Table 2). The four patients who underwent abdominal re-exploration and evacuation of blood had a prompt diuresis, although two later succumbed to sepsis. The timed course of the relationship of rising intra-abdominal pressure and decreasing urinary output was documented for one patient in detail and compared to other patients in the study group. This is detailed in figure 1.

Three patients with marked elevation of IAP (ranging from 40 to 77 mmHg) were not re-explored, and all developed acute renal failure and died in the early postoperative period.

Discussion

There are many reports that increased intra-abdominal pressure affects renal function by reducing cardiac output.[6,7] We recently reported a laboratory study demonstrating that increased IAP impairs renal function by increasing renal vascular resistance.[5] Neither ureteral compression nor decreased cardiac output was the cause of the renal impairment. In the canine model, intra-abdominal pressures of 20 mmHg caused a marked reduction in renal blood flow despite normal or high cardiac outputs.

There have been few clinical reports on the effects of increased IAP. One recent clinical study reported four patients with acute postoperative renal insufficiency whose renal function improved after abdominal decompression.[8] No measurements of intra-abdominal pressure were obtained, and therefore, no specific criteria for re-exploration were cited.

None of our patients with renal insufficiency and elevated IAP responded to the usual methods of producing diuresis, including the use of dopaminergic agents, diuretics, or volume replacement and maintenance of ad-

TABLE 2. *Correlation of Intra-abdominal Pressure (IAP) and Renal Insufficiency*

Pt.	Procedure	Age	C.I.	PCW	Mean BP	Urine Output	IAP	Result
Patients Re-explored								
1	Repair aortic stab wound	64	3.3	19	120	0/hr	45	late death from sepsis
	(after re-exploration and decompression)		4.2	20	130	150/hr	23	
2	Gastrectomy	55	3.5	25	105	5/hr	34	
	(after re-exploration and decompression)		3.3	20	100	200/hr	15	excellent
3	Ruptured AAA	72	3.0	18	100	5/hr	40	
	(after re-exploration and decompression)		3.2	18	100	150/hr	10	excellent
4	Resection pelvic tumor	29	—	22	100	5/hr	30	late death from sepsis
	(after re-exploration and decompression)		—	15	90	100/hr	10	
Patients not Re-explored								
5	Hemobilia repair	55	2.0	24	100	5/hr	77	death
6	Portacaval shunt	68	3.0	24	100	0/hr	60	death
7	Splenorenal shunt	42	—	20	100	5/hr	40	death

equate blood pressure, cardiac filling pressure, and cardiac output. All patients who underwent abdominal re-exploration and decompression had a prompt diuresis. The four patients treated without re-exploration remained in renal failure and all died. It is of interest that only three of the seven patients re-explored were found to have a specific site of bleeding. The other four patients underwent evacuation of the clotted blood, but no bleeding site was identified.

The direct measurement of IAP through an indwelling transurethral bladder catheter has become a simple and reliable diagnostic technique for us. Physical examination is often not helpful, in that abdominal pressures below 20 mmHg usually result in a tense abdomen. IAPs below 20 mmHg in a postoperative patient in the absence of rapid blood loss or renal insufficiency are an indication for continued observation. An intra-abdominal pressure above 25 mmHg in a postoperative patient with an adequate blood volume and a low urinary output is an indication for abdominal re-exploration and decompression.

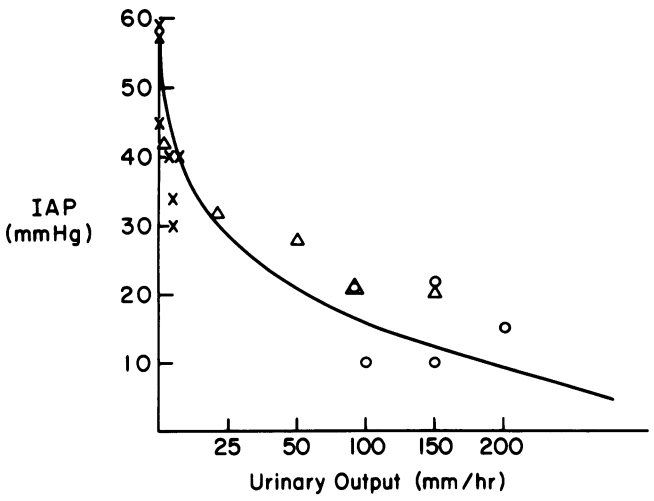

FIG. 1. The correlation of intra-abdominal pressure (IAP) and urinary output. Δ = patient #1; × = without re-exploration; and O = after re-exploration.

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