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Determination of Intra-abdominal Pressure Using a Transurethral Bladder Catheter: Clinical Validation of the Technique

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The determination of intra-abdominal pressures (IAP) may be useful in many clinical situations. The authors recently demonstrated in the canine model a close correlation between actual IAP and the bladder pressure measurements obtained from a transurethral catheter. The purpose of this study was to clinically validate this technique. The authors studied 16 patients, and compared IAP in three positions (supine, with compressions, and semi-erect) utilizing both direct intraperitoneal pressure monitoring and the pressure obtained with a transurethral bladder catheter. Their results demonstrated a linear relationship between the two methods described, with a mean r value of 0.95 in the supine and semi-erect positions, and 0.99 with abdominal compressions ($P < 0.0001$). The authors conclude that transurethral monitoring of bladder pressure offers a safe, simple, and highly accurate method for evaluating IAP at the bedside. Studies evaluating the indication for its use in the operating room and intensive care settings appear warranted. (Key words: intra-abdominal pressure; monitoring techniques.)

INCREASED INTRA-ABDOMINAL PRESSURE (IAP) may be associated with a variety of clinical situations that may adversely affect cardiac, renal, respiratory, and metabolic functions.[1–5] Despite this, the diagnosis of increased IAP is infrequently made probably because the ability to measure IAP at the bedside is not available. Several authors have used the pressure measurements obtained from an indwelling transurethral bladder catheter to estimate IAP.[5,6] The reliability of this method, however, has not been demonstrated. Using a canine model, we validated the accuracy of this technique over a wide range of abdominal pressures.[7] In contrast to the dog, the human bladder is an extraperitoneal organ, which raised questions concerning the clinical applicability of this technique. The purpose of this study, therefore, was to determine whether urinary bladder pressure accurately reflects IAP in humans.

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Materials and Methods

Postoperative patients with bladder catheters were entered into this study if they had one of the following: 1) closed system abdominal drain ("Jackson Pratt Type") placed intraoperatively, or 2) required paracentesis. Patients were excluded if they had a history of bladder disease or an open abdominal incision. The study was approved by our Investigational Review Board. All patients or responsible family members signed written consent prior to the study.

Using sterile technique, an average of 250 ml of normal saline was infused through the urinary catheter to gently fill the catheter tubing into the bladder and eliminate air in the drainage catheter. With the bladder catheter to gravity drainage to avoid increasing bladder pressure, the catheter was then clamped distal to the sampling membrane. A 20-gauge needle was inserted through the catheter sampling membrane and the bladder catheter pressures were transduced using 84-inch arterial pressure tubing (Sorenson Research, Salt Lake City, UT), and Gould disposable transducers (model TXXR, Gould Inc., Oxnard, CA). Pressure measurements from the abdominal drains (or paracentesis catheter) were obtained using the same monitoring techniques. The closed system drains required instillation of saline to clear air from the catheters. Transducers for the bladder catheter and abdominal drain were zeroed at the level of the pubis. Pressures were displayed and recorded (fig. 1). After a 2-min equilibration period, both the intra-abdominal and urinary bladder pressures were recorded in the supine position, supine with gentle manual abdominal compression, and semi-erect (45° angle). The pressures varied freely with respirations; therefore, all data were recorded as mean pressures at end-expiration (fig. 2).

The data were analyzed for linear regression and correlation using the SAS REG procedure of the SAS personal computer statistical package (SAS Institute Inc., Cary, NC).

Results

Sixteen postoperative patients were studied, 12 females and four males (mean age 74 ± 3 yr). Fourteen patients

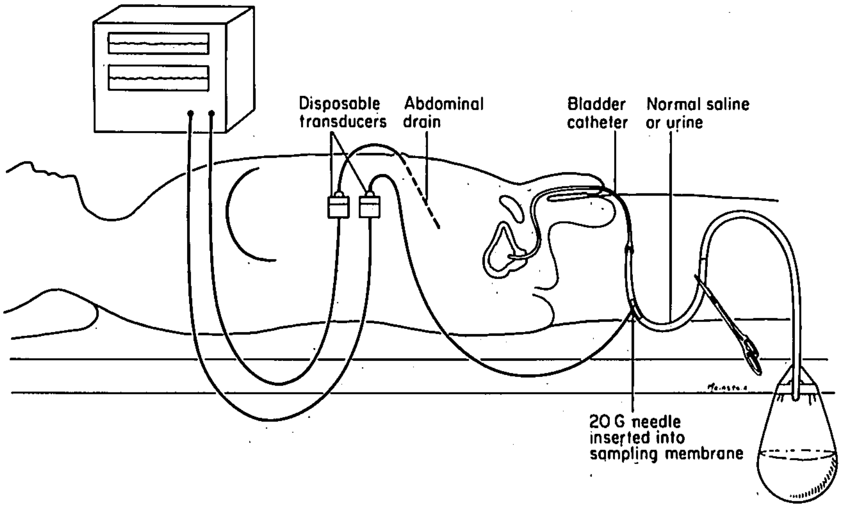

FIG. 1. Schematic illustration of a patient in the supine position showing the closed system drain and transurethral bladder pressure monitoring technique.

had a closed-system drain placed intraoperatively, and two patients underwent diagnostic paracentesis while in the intensive care unit (table 1). Ventilation was controlled in three of the patients. Forty-six separate measurements were taken in the supine and semi-erect positions, and supine with abdominal compressions. Patients 3 and 4 did not have semi-erect pressure measurements because of hemodynamic instability. Patient 6 was restless at the time of pressure determination with abdominal compression, and a satisfactory recording was not obtained (table 2).

The correlation between the two methods of IAP evaluation was $r = 0.91$ ($P < 0.0001$) independent of patient position. With compression, the correlation was $r = 0.99$ ($P < 0.0001$) (fig. 3).

Discussion

The results of this study demonstrate that bladder pressure and intra-abdominal pressure are nearly identical in humans. Over the range of intra-abdominal pressures measured, the transurethral catheter provides an accurate measurement of IAP, and provides validation for a new, simple bedside monitoring tool.

Normal intra-abdominal pressure is 0 to subatmospheric.[8,9] Elevations in IAP will occur when the distensible component of the peritoneal cavity (peritoneum, abdominal muscles, and diaphragm) become less compliant. This can be secondary to increased intra-abdominal volume (*i.e.*, ascites, abdominal tumors, and pneumoperitoneum), or from extra-abdominal pressure (*i.e.*, military anti-shock trowsers).[10,11] At such a time, direct compression of all abdominal components occur, venous return from the lower extremities is impaired, and intra-abdominal pressure is transmitted *via* the diaphragm to the thoracic cavity.[3,12] Perfusion pressures (mean arterial pressure—mean venous pressure) to intra-abdominal organs, the abdominal musculature, peritoneum, and the diaphragm are reduced to the degree of the IAP elevation.

Most of the currently available data regarding the pathophysiology associated with elevated IAP come from animal models, and these can be summarized. When the IAP rises above 25 mmHg, there is evidence of significant changes in hemodynamic, respiratory, renal, and metabolic functions.[8,13] Cardiac output, left ventricular contractility, and stroke volume are reduced; and systemic

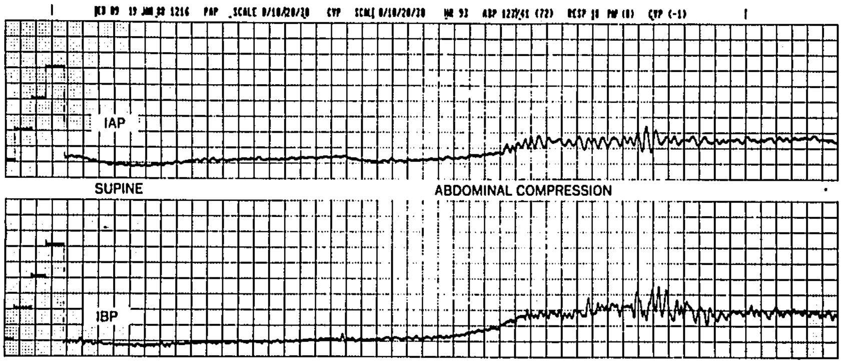

FIG. 2. Example of simultaneous monitor strip comparing both methods (IAP = intra-abdominal pressure using a closed system drain; IBP = intra-bladder pressure using a transurethral catheter) in a patient supine and with abdominal compressions. The strips were run at 25 mm/sec speed, and the pressures as shown by standardization bars at left of each strip are 0, 10, 20, and 30 mmHg.

TABLE 1. Patient Characteristics and Method of Measuring Intragastric Pressure (IAP)

Patient Number	Age	Male/Female	Surgery	IAP Method
1	89	F	Gastrectomy	Closed system
2	61	M	Cholecystectomy	Closed system
3	96	F	Laparotomy	Paracentesis
4	78	F	Duodenal ulcer oversew	Closed system
5	83	F	Esophagectomy	Closed system
6	82	F	Esophagectomy	Closed system
7	86	F	Abdominal abscess drainage	Paracentesis
8	74	M	Cholecystectomy	Closed system
9	78	F	Cholecystectomy	Closed system
10	76	F	Cholecystectomy	Closed system
11	73	F	Esophagogastrectomy	Closed system
12	66	M	Gastrojejunostomy	Closed system
13	74	F	Ovarian carcinoma debulking	Closed system
14	75	F	Colectomy, splenectomy	Closed system
15	70	F	Gastrectomy	Closed system
16	80	M	Gastrectomy	Closed system

vascular resistance, central venous pressure, and pulmonary artery wedge pressure increase.[2,3] Mean arterial pressure and heart rate are usually unaffected.[3,7] Systemic blood flow is redistributed so that a higher percentage cardiac output goes to the upper extremities, and a lower percentage to the abdominal organs and lower extremities.[2] These hemodynamic changes are heightened in the hypovolemic animal.[5,14]

Animal data have revealed significant decreases in arterial oxygenation and pulmonary blood flow with increased IAP.[4] Of particular importance is that the addition of positive end-expiratory pressure markedly increases the detrimental effect of IAP on cardiac and pulmonary function.[1,4,15] Experimental models of increased IAP have also documented a reduction in renal perfusion pressure, changes in renal blood flow, and decreases in glomerular filtration rates.[8] Oliguria/anuria have been experimentally produced with elevation of abdominal pressure.[8,16]

Elevations of IAP above 30 mmHg have been commonly reported in a variety of clinical conditions often encountered in seriously ill patients.[17–19] Despite this, few clinical studies involving elevated IAP exist due to the perceived need for invasive peritoneal monitoring. From case reports and studies performed in patients receiving peritoneal dialysis or using military anti-shock trousers, it appears that increased IAP has similar physiologic effects as those described in the experimental models.[12,18,20] A recent study in a select group of surgical patients documented anuria when the IAP was greater than 30 mmHg and a reversal with lowering of the abdominal pressure.[16] Cerbona *et al.* investigated the hemodynamic and renal consequences of performing paracentesis in a group of 15 cirrhotics with tense ascites.[21] A reduction of the mean IAP from 33 ± 15 to 17.1 ± 8.5 cm H_2O resulted in a significant decrease in blood urea nitrogen and systemic resistance, and an increase in creatinine clearance, cardiac index, left ventricular stroke work, and arterial-venous oxygenation difference.

As demonstrated in the present study, monitoring IAP in seriously ill patients is basically noninvasive, with no associated risks, since the majority of these patients have existing bladder catheters. The procedure is technically simple, and there were no complications in the patients studied. Attention should be paid to respiratory variations, and the need to partially fill the empty bladder, to insure a fluid column in the catheter. Although no patient had extremely high IAP (>30 mmHg), our previous study in dogs demonstrated that the bladder catheter technique

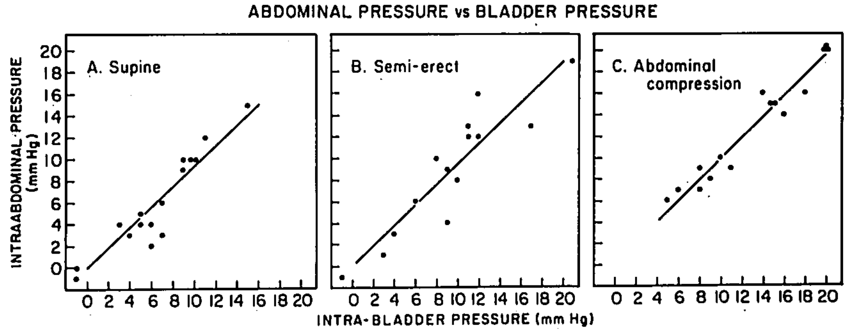

FIG. 3. Linear correlation between intra-abdominal pressure (IAP) and intra-bladder pressure (IBP) measurements, supine (*A*), semi-erect (*B*), and abdominal compressions (*C*).

accurately represented IAP to intra-abdominal pressures as high as 70 mmHg.[7]

The role of IAP monitoring has not been clearly defined. In cases of pressures greater than 20 mmHg associated with anuria, intervention to reduce the IAP appears warranted. The use of positive end-expiratory pressure in patients with concomitant increased IAP and low output states or respiratory insufficiency needs to be closely evaluated. Due to the accuracy, simplicity, and low cost of the described technique, prospective randomized studies evaluating the effects of IAP in critically ill patients should be performed. It is conceivable that recognition and possible manipulation of this parameter may be useful in a wide range of clinical situations.

References

1. Richardson JD, Trinkle JK: Hemodynamic and respiratory alterations with increased intra-abdominal pressure. J Surg Res 20: 401–404, 1976
2. Barnes GE, Laine GA, Giam PY, Smith EE, Granger HJ: Cardiovascular responses to elevation of intra-abdominal hydrostatic pressure. Am J Physiol R208–R213, 1985
3. Robotham JL, Wise RA, Bromberger-Barnea B: Effects of changes in abdominal pressure on left ventricular performance and regional blood flow. Crit Care Med 13:803–809, 1985
4. Burchard KW, Ciombor DM, McLeod MK, Slothman GJ, Gann DS: Positive end expiratory pressure with increased intra-abdominal pressure. Surg Obstet 161:313–318, 1985
5. Kashtan J, Green JF, Parsons EQ, Holcroft JW: Hemodynamic effects of increased abdominal pressure. J Surg Res 30:249–255, 1981
6. Kron IL, Harman PK, Nolan SP: The measurement of intra-abdominal pressure as a criterion for abdominal re-exploration. Ann Surg pp 28–30, 1984
7. Iberti TJ, Kelly KM, Gentili DR, Hirsch S, Benjamin E: A simple technique to accurately determine intra-abdominal pressure. Crit Care Med 15:1140–1142, 1987
8. Harman PK, Kron IL, McLachlan HD, Freedlender AE, Nolan SP: Elevated intra-abdominal pressure and renal function. Ann Surg 196:594–597, 1982
9. Emerson H: Intra-abdominal pressures. Arch Int Med 7:754–784, 1911
10. Schurig R, Gahl GM, Becker H, Schiller R, Kessel M, Paeprer H: Hemodynamic studies in long-term peritoneal dialysis patients. Trans Am Soc Artif Intern Organs 3:215–218, 1979
11. Shenasky JH, Gillenwater JY: The renal hemodynamic and functional effects of external counterpressure. Surg Gynecol Obstet 134:253–258, 1972
12. Guazzi M, Polese A, Magrini F, Fiorentini C, Olivari MT: Negative influences of ascites on cardiac function of cirrhotic patients. Am J Med 59:165–170, 1975
13. LeRoith D, Bark H, Nyska M, Glick SM: The effect of abdominal pressure on plasma antidiuretic hormone levels in the dog. J Surg Res 32:65–69, 1982
14. Toomasian JM, Glavinovich G, Johnson MN, Gazzaniga AB: Hemodynamic changes following pneumoperitoneum and graded hemorrhage in the dog. Surg Forum 29:32, 1978
15. Buchler B, Magder S, Katsardis H, Jammes Y, Roussos C: Effects of pleural pressure and abdominal pressure on diaphragmatic blood flow. J Appl Physiol 58:691–697, 1985
16. Richards WO, Scovill W, Baekyo S, Reed W: Acute renal failure associated with increased intra-abdominal pressure. Ann Surg 197:183–187, 1982
17. Knauer CM, Lowe HM: Hemodynamics in cirrhotic patient during paracentesis. N Engl J Med 276:491–496, 1967
18. Gaffney FA, Thal ER, Taylor WF, Bastian BC, Weigelt JA, Atkins JM, Blomqvist CG: Hemodynamic effects of medical anti-shock trousers (MAST). J Trauma 21:931–937, 1981
19. Nadrowski LF: Pathophysiology and current treatment of intestinal obstruction. Rev Surg 31:381–407, 1974
20. Smith JH, Merrell RC, Raffin TA: Reversal of postoperativeanuria by decompressive celiotomy. Arch Intern Med 145:533–534, 1985
21. Cerabona T, Savino J, Agarwal N: Urinary bladder measurements of intra-abdominal pressure (IAP) in ascitic cirrhotics predictive of hemodynamic and renal function (abstract). Crit Care Med 16:431, 1988