PREDICTIVE IMPACT OF INTRA-ABDOMINAL PRESSURE ON SURVIVAL IN OPEN ABDOMEN SURGICAL PATIENTS

Markus Helle

Syventävien opintojen kirjallinen työ

Tampereen yliopisto

Lääketieteen yksikkö

Anestesiologia ja tehohoito

Toukokuu 2012

Tampereen yliopisto
Lääketieteen yksikkö
Anestesiologia ja tehohoito

HELLE MARKUS: PREDICTIVE IMPACT OF INTRA-ABDOMINAL PRESSURE ON SURVIVAL IN OPEN ABDOMEN SURGICAL PATIENTS

Syventävien opintojen kirjallinen työ, 12 s
Ohjaajat: Professori Leena Lindgren ja dosentti Sari Räty

Toukokuu 2012

Avainsanat: Vatsaontelon ylipaineoireyhtymä, vatsaontelon ylipaine, vatsaontelon paine, avoin vatsaontelo

---

Vatsaontelon ylipaineoireyhtymässä kohonnut vatsaontelon paine heikentää elintärkeiden elinten toimintaa. Ensisijainen hoito on konservatiivinen, mutta mikäli vatsaontelon paine ei laske ja tila etenee vatsaontelon ylipaineoireyhtymäksi, vatsanpeitteet avataan ja jätetään auki.

Tutkimuksen tavoitteena oli arvioida, vaikuttaako pelkkä vatsaontelon paine potilaiden ennusteeseen.

Tutkimusaineisto koostui 32 potilaasta, joiden vatsaontelo avattiin ja jätettiin auki syyskuun 2008 ja elokuun 2010 välisenä aikana Tampereen yliopistollisessa sairaalassa. Tutkimuksessa verrattiin toimenpiteestä selvinneitä menehtyneihin.

Tutkimusaineistossa keski-ikä oli 66 vuotta ja kuolleisuus 38 %. Vatsaontelon avausta edeltävä vatsaontelon paine oli keskimäärin yhtä korkea kummassakin ryhmässä. Molemmissa ryhmissä vatsaontelon paine laski ajan myötä, mutta menehtyneillä nopeammin. Menehtyneillä seerumin laktaatti oli korkeampi seurannan alkuvaiheessa ja CRP korkeampi seurannan loppuvaiheessa.

Tutkimuksen päälöydös oli ettei vatsaontelon paine yksinään ennusta potilaan selviytymistä. Varhainen kohonnut laktaatti ja myöhäisvaiheen CRP:n nousu liittyivät huonoon ennusteeseen. Potilaan yleistila ja muut parametrit tulee ottaa huomioon mietittäessä vatsan avaamista.

Sisällys

Introduction

Abdominal compartment syndrome is condition in which elevated intra-abdominal pressure (IAP) affects cardiovascular, respiratory, gastrointestinal and renal functions (1-4). Elevation of IAP has been associated especially to oliguria and acute renal failure (5-8). When intra-abdominal pressure is 12 mmHg or more condition is defined as intra-abdominal hypertension (IAH). With IAP values exceeding 20 mmHg combined to organ dysfunction or failure has condition advanced to abdominal compartment syndrome (ACS) (9). Due to its harmfull pathophysiologic effects, elevated IAP can lead to death (10). Up to one half of all intensive care unit (ICU) patients develop IAH and approximately 5 to 30 per cent of patients develop ACS (11,12). Primary treatment is always conservative but if the IAP does not decrease or condition worsens with signs of ACS, abdomen is surgically decompressed and left open (13).

Different techniques have been designed to protect the open abdomen while the patient is recovering. One of the first techniques was introduced in Bogota, Colombia, and consists of the use of empty plastic intravenous bag sutured to cover the open abdomen (Bogota bag) (14). Besides the Bogota bag, more sophisticated methods, i.e. different mesh and vacuum assisted closure (VAC), are used for this purpose (15-17). Vacuum assisted closure utilizes the negative pressure therapy and has proved to help fascial closure (18-21). If primary fascial closure is not possible, i.e. due to visceral oedema or fascial retraction, a planned ventral hernia is created and reconstructed later on (13,22,23). Because severe morbidity and mortality associated with surgical decompression, the indications should be carefully evaluated (24,25). There are different views when and by which criteria the abdomen has to be opened (25-29).

The aim of our study was to evaluate IAP recordings in open abdomen patients and to compare various IAP-related parameters in the survivors and the non-survivors. We were especially interested whether a single IAP value would predict when to decompress or not.

Patients and Methods

The data consisted of all consecutive open abdomen patients from September 2008 to August 2010. All the patients were mechanically ventilated in the ICU at Tampere University Hospital. The patients were treated according to international guidelines of ICU treatment. Laboratory results for vital functions and IAP were collected from hospital records. Recorded parameters were IAP, serum lactate, pH, C-reactive protein (CRP), plasma creatinine and urea. Some variation within one day was seen in a few laboratory values. These included IAP, lactate and pH. Values closest to 8 a.m., 4 pm. and 12 p.m. were assessed. Other values were recorded once per day.

There were two groups of patients: 1) those who were primarily decompressed during ICU-stay (primary decompression) 2) those whose abdominal wall was left open at the end of emergency laparotomy (secondary decompression). The parameters of the survivors and the non-survivors were compared irrespective of the type of decompression.

The length of ICU-stay, the length of open abdomen treatment and outcome of treatment were assessed. Possible VAC treatment was included in the length of open abdomen treatment and continuous veno-venous hemodiafiltration (CVVHDF) was taken into account in analysis. Follow-up started in the beginning of operation day and lasted for 14 days, during which all of the non-survivors had succumbed. Values were compared between the survivors and the non-survivors. Reference values for studied laboratory values are presented in Table 1.

Statistics

Values are presented as means +/- SD or in case of skewed distribution medians +/- 1st and 3rd quartile ranges. Statistical analysis was performed with Mann-Whitney's U-test for two independent groups since the laboratory values tended to have abnormal distributions. Survival analysis was performed for every baseline laboratory value (first value on operation day) with cox regression analyze. All statistical tests were performed with SPSS 17.0 and *P* values of less than 0.05 were regarded as significant.

Results

The demographics of the 32 patients are presented in Table 2. The mean age of patients was 66 (range 54-78) years. Primary decompression was done in 19 and secondary decompression in 13 patients. Mortality was 38% and after the sixth day of follow-up only one third of the non-survivors were alive.

In both groups, IAP decreased over time and was first time under 12 mmHg on the second day 12 p.m. in the non-survivors and on sixth day 8 am. in the survivors. Initial values of IAP were almost similar in the two groups (Table 2). There was a tendency that IAP was lower in the the non-survivors although the differences were not statistically significant (Figure 1 A).

Serum lactate was significantly higher on the second and third day in the non-survivors (P <0.05) and (Figure 1 B) on the eleventh, twelfth and thirteenth day CRP was also higher in the non-survivors (P <0.05) (Figure 1 D). The values of pH initially and after the fifth day were slightly higher in the survivors (*NS*) (Figure 1 C).

There was no difference in urea values in the study groups. In the non-survivors creatinine peaked to twofold during the latter half of follow-up.

Nineteen of 32 patients (59%) went through CVVHDF treatments during follow-up. Sixtyseven per cent of the non-survivors and 55% of the survivors had at least one CVVHDF. Mortality in CVVHDF patients was 42% and in patients without dialysis 31% (NS). Length of ICU-stay was shorter in patients without dialysis than patients who received CVVHDF treatment [4 days (range 2-7) vs. 8 days (range 5-12), (P<0.05]. The length of open abdomen treatment was similar irrespective of CVVHDF treatment.

Discussion

The main finding of our study was that IAP only did not predict the survival. In decision making whether to decompress or not should also be based on other parameters such as serum lactate and CRP. When comparing laboratory values of the survivors and the non-survivors, the groups were rather similar.

Especially IAP showed no association to outcome and the initial values were remarkably similar. Surprisingly, the non-survivors had an average lower IAP recordings and their IAP decreased faster to normal than in the survivors. This may be due to inadequate and low organ perfusion leading to lower edema in the non-survivors. Elevated IAP may be reduced by CVVHDF treatment, which might have affected our results (24,30). However, our study patients were similarly treated. The initial laboratory values were about similar in the survivors and the non-survivors. Higher lactate values seen in the non-survivors during the second and third day reflect their poor overall circulation. After the sixth day rapidly increasing CRP predicted the death. C-reactive protein is not elevated only in inflammation, but also in many ischemic processes (31).

In the light of our findings it is more and more clear that IAP alone is not indicative to opening the abdomen and do not benefit the patient. The overall condition and other parameters should be taken into account when making the decision to open or not. This view is also supported by Eddy *et al* (26), De Waele *et al* (28) and Leppaniemi (25). In certain situations opening the abdomen and leaving it open is the only option (27) and in many patients the open abdomen can be closed within the same hospital stay (15,32). Avoiding excessive volume loading and intra-abdominal edema may decrease the need for decompression (33,34). Possible complications of this treatment are another issue. Once the abdomen has been opened every effort should be made to achieve primary fascial closure. In patients who cannot

undergo primary fascial closure, a large ventral hernia is followed and needs reconstruction later on (15,35). These patients require longer ICU and hospital stays, face a risk for fistula formation and other complications and require more reinterventions let alone the notable consumption of financial resources (22,35,36). However, even in these patients longterm outcome seems to be good once the abdomen has eventually been closed.(37,38).

Study limitations

Our study was retrospective which thus makes the recordings and follow-up possibly less comprehensive. Also the sample size was small. We do not believe that greater number of patients, however, would have lead to different results. Also from ethical point of view, a prospective study in high IAP patients is impossible to perform.

Conclusions

The main finding of our study was that IAP only did not predict the survival. Early lactatemia and late high CRP were signs of poor outcome. Decompression is costly and needs to be weighed carefully.

References

(1) Diebel LN, Dulchavsky SA, Brown WJ. Splanchnic ischemia and bacterial translocation in the abdominal compartment syndrome. Journal of Trauma-Injury Infection & Critical Care 1997 Nov;43(5):852-855.

(2) Barnes GE, Laine GA, Giam PY, Smith EE, Granger HJ. Cardiovascular responses to elevation of intra-abdominal hydrostatic pressure. Am J Physiol 1985 Feb;248(2 Pt 2):R208-13.

(3) Malbrain ML. Abdominal pressure in the critically ill: measurement and clinical relevance. Intensive Care Med 1999 Dec;25(12):1453-1458.

(4) Bloomfield GL, Ridings PC, Blocher CR, Marmarou A, Sugerman HJ. Effects of increased intra-abdominal pressure upon intracranial and cerebral perfusion pressure before and after volume expansion. Journal of Trauma-Injury Infection & Critical Care 1996 discussion 941-3; Jun;40(6):936-941.

(5) Sugrue M, Jones F, Deane SA, Bishop G, Bauman A, Hillman K. Intra-abdominal hypertension is an independent cause of postoperative renal impairment. Archives of Surgery 1999 Oct;134(10):1082-1085.

(6) Mullens W, Abrahams Z, Skouri HN, Francis GS, Taylor DO, Starling RC, et al. Elevated intra-abdominal pressure in acute decompensated heart failure: a potential contributor to worsening renal function?. J Am Coll Cardiol 2008 Jan 22;51(3):300-306.

(7) Dalfino L, Tullo L, Donadio I, Malcangi V, Brienza N. Intra-abdominal hypertension and acute renal failure in critically ill patients. Intensive Care Med 2008 Apr;34(4):707-713.

(8) Mohmand H, Goldfarb S. Renal dysfunction associated with intra-abdominal hypertension and the abdominal compartment syndrome. Journal of the American Society of Nephrology 2011 Apr;22(4):615-621.

(9) Malbrain ML, Cheatham ML, Kirkpatrick A, Sugrue M, Parr M, De Waele J, et al. Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. I. Definitions. Intensive Care Med 2006 Nov;32(11):1722-1732.

(10) Malbrain ML, Chiumello D, Pelosi P, Bihari D, Innes R, Ranieri VM, et al. Incidence and prognosis of intraabdominal hypertension in a mixed population of critically ill patients: a multiple-center epidemiological study. Crit Care Med 2005 Feb;33(2):315-322.

(11) Malbrain ML, Chiumello D, Pelosi P, Wilmer A, Brienza N, Malcangi V, et al. Prevalence of intra-abdominal hypertension in critically ill patients: a multicentre epidemiological study. Intensive Care Med 2004 May;30(5):822-829.

(12) Vidal MG, Ruiz Weisser J, Gonzalez F, Toro MA, Loudet C, Balasini C, et al. Incidence and clinical effects of intra-abdominal hypertension in critically ill patients. Crit Care Med 2008 Jun;36(6):1823-1831.

(13) Cheatham ML, Malbrain ML, Kirkpatrick A, Sugrue M, Parr M, De Waele J, et al. Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. II. Recommendations. Intensive Care Med 2007 Jun;33(6):951-962.

(14) Mattox KL. Introduction, background, and future projections of damage control surgery. Surg Clin North Am 1997 Aug;77(4):753-759.

(15) Barker DE, Kaufman HJ, Smith LA, Ciraulo DL, Richart CL, Burns RP. Vacuum pack technique of temporary abdominal closure: a 7-year experience with 112 patients. Journal of Trauma-Injury Infection & Critical Care 2000 discussion 206-7; Feb;48(2):201-206.

(16) Kirshtein B, Roy-Shapira A, Lantsberg L, Mizrahi S. Use of the "Bogota bag" for temporary abdominal closure in patients with secondary peritonitis. Am Surg 2007 Mar;73(3):249-252.

(17) Aydin C, Aytekin FO, Yenisey C, Kabay B, Erdem E, Kocbil G, et al. The effect of different temporary abdominal closure techniques on fascial wound healing and postoperative adhesions in experimental secondary peritonitis. Langenbecks Archives of Surgery 2008 Jan;393(1):67-73.

(18) Garner GB, Ware DN, Cocanour CS, Duke JH, McKinley BA, Kozar RA, et al. Vacuum-assisted wound closure provides early fascial reapproximation in trauma patients with open abdomens. Am J Surg 2001 Dec;182(6):630-638.

(19) Miller PR, Meredith JW, Johnson JC, Chang MC. Prospective evaluation of vacuum-assisted fascial closure after open abdomen: planned ventral hernia rate is substantially reduced. Ann Surg 2004 discussion 614-6; May;239(5):608-614.

(20) Bee TK, Croce MA, Magnotti LJ, Zarzaur BL, Maish GO,3rd, Minard G, et al. Temporary abdominal closure techniques: a prospective randomized trial comparing polyglactin 910 mesh and vacuum-assisted closure. Journal of Trauma-Injury Infection & Critical Care 2008 discussion 342-4; Aug;65(2):337-342.

(21) Batacchi S, Matano S, Nella A, Zagli G, Bonizzoli M, Pasquini A, et al. Vacuum-assisted closure device enhances recovery of critically ill patients following emergency surgical procedures. Crit Care 2009;13(6):R194.

(22) Fabian TC, Croce MA, Pritchard FE, Minard G, Hickerson WL, Howell RL, et al. Planned ventral hernia. Staged management for acute abdominal wall defects. Ann Surg 1994 discussion 651-3; Jun;219(6):643-650.

(23) DiCocco JM, Magnotti LJ, Emmett KP, Zarzaur BL, Croce MA, Sharpe JP, et al. Long-term follow-up of abdominal wall reconstruction after planned ventral hernia: a 15-year experience. J Am Coll Surg 2010 695-8; May;210(5):686-695.

(24) Cheatham ML. Nonoperative management of intraabdominal hypertension and abdominal compartment syndrome. World J Surg 2009 Jun;33(6):1116-1122.

(25) Leppaniemi A. Surgical management of abdominal compartment syndrome; indications and techniques. Scandinavian Journal of Trauma, Resuscitation & Emergency Medicine 2009;17:17.

(26) Eddy V, Nunn C, Morris JA,Jr. Abdominal compartment syndrome. The Nashville experience. Surg Clin North Am 1997 Aug;77(4):801-812.

(27) Schein M. Surgical management of intra-abdominal infection: is there any evidence? Langenbecks Archives of Surgery 2002 Apr;387(1):1-7.

(28) De Waele JJ, Hoste EA, Malbrain ML. Decompressive laparotomy for abdominal compartment syndrome--a critical analysis. Crit Care 2006;10(2):R51.

(29) Leppaniemi AK. Laparostomy: why and when? Crit Care 2010;14(2):216.

(30) Bonfim RF, Goulart AG, Fu C, Torquato JA. Effect of hemodialysis on intra-abdominal pressure. Clinics (Sao Paulo, Brazil) 2007 Apr;62(2):145-150.

(31) Lobo SM, Lobo FR, Bota DP, Lopes-Ferreira F, Soliman HM, Melot C, et al. C-reactive protein levels correlate with mortality and organ failure in critically ill patients. Chest 2003 Jun;123(6):2043-2049.

(32) Boele van Hensbroek P, Wind J, Dijkgraaf MG, Busch OR, Carel Goslings J. Temporary closure of the open abdomen: a systematic review on delayed primary fascial closure in patients with an open abdomen. World J Surg 2009 Feb;33(2):199-207.

(33) Balogh Z, McKinley BA, Cocanour CS, Kozar RA, Valdivia A, Sailors RM, et al. Supranormal trauma resuscitation causes more cases of abdominal compartment syndrome. Archives of Surgery 2003 discussion 642-3; Jun;138(6):637-642.

(34) Madigan MC, Kemp CD, Johnson JC, Cotton BA. Secondary abdominal compartment syndrome after severe extremity injury: are early, aggressive fluid resuscitation strategies to blame?. Journal of Trauma-Injury Infection & Critical Care 2008 Feb;64(2):280-285.

(35) Miller PR, Thompson JT, Faler BJ, Meredith JW, Chang MC. Late fascial closure in lieu of ventral hernia: the next step in open abdomen management. Journal of Trauma-Injury Infection & Critical Care 2002 Nov;53(5):843-849.

(36) Cheatham ML, Safcsak K, Sugrue M. Long-term implications of intra-abdominal hypertension and abdominal compartment syndrome: physical, mental, and financial. Am Surg 2011 Jul;77(Suppl 1):S78-82.

(37) Cheatham ML, Safcsak K, Llerena LE, Morrow CE,Jr, Block EF. Long-term physical, mental, and functional consequences of abdominal decompression. Journal of Trauma-Injury Infection & Critical Care 2004 discussion 241-2; Feb;56(2):237-241.

(38) Cheatham ML, Safcsak K. Longterm impact of abdominal decompression: a prospective comparative analysis. J Am Coll Surg 2008 Oct;207(4):573-579.

Tables

Table 1. Reference values of laboratory measurements.

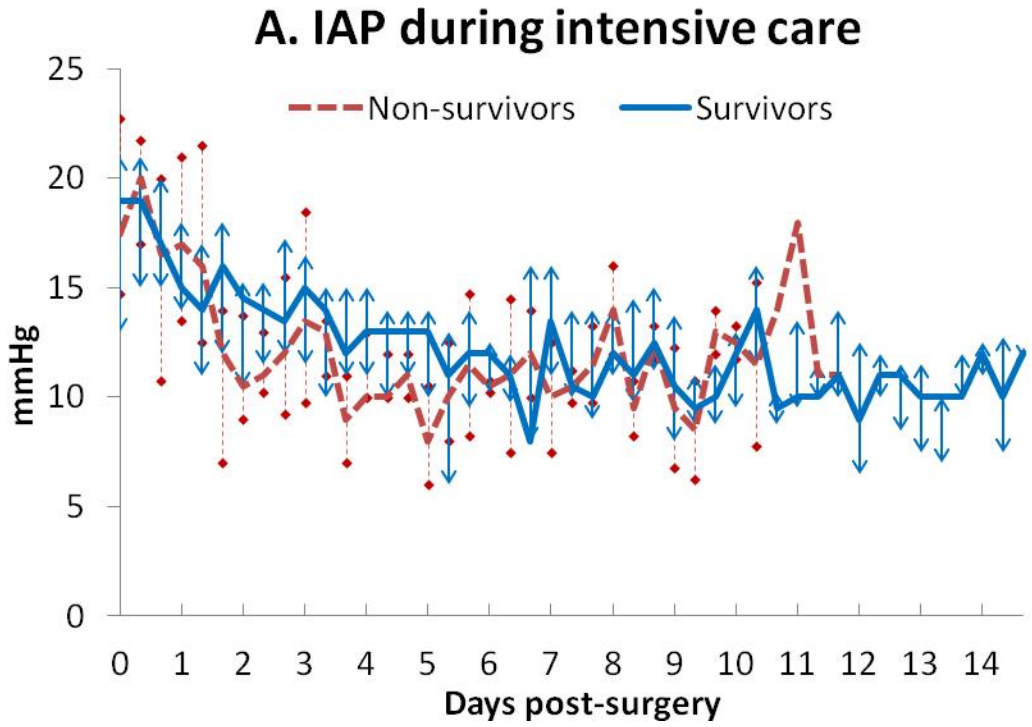
Laboratory measurements	Reference values
C-reactive protein (CRP)	<10mg/L
Intra-abdominal pressure (IAP)	<12 mmHg
Creatinine	60-100 µmol/L (M) 50-90 µmol/L (F)
Lactate	0.5-1.6 mmol/L
Urea	3.5-8.1 mmol/L (M) 3.1-7.9 mmol/L (F)
pH (arterial)	7.35-7.44

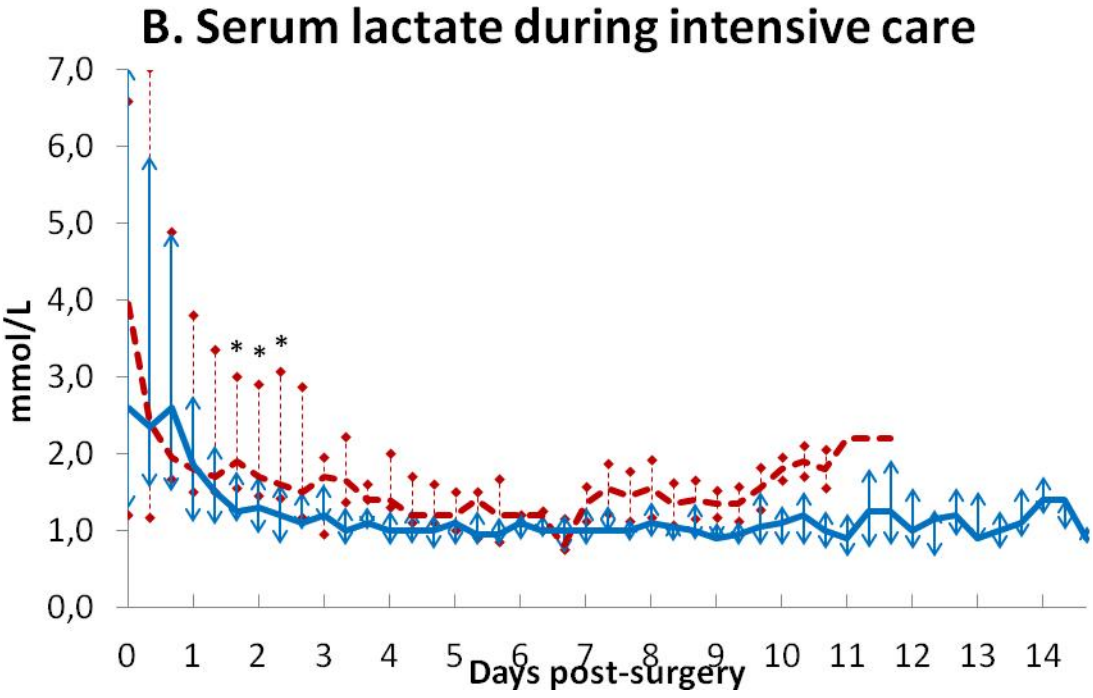
Table2. Demographics. Laboratory values are medians of first measured values on operation day.

Demographic characteristics	Survivors (n=20)	Non-survivors (n=12)	*P* value
Age (years)	65 (±13)	64 (±12)	*NS*
Sex (male)	15 (75%)	9 (75%)	*NS*
Primary decompression	12 (63%)	7 (62%)	*NS*
Secondary decompression	8 (37%)	5 (38%)	*NS*
IAP (mmHg)	19 (13-21)	18 (15-23)	0.71
Crea (µmol/l)	133 (101-205)	128 (108-177)	0.86
pH	7.29 (7.26-7.39)	7.27 (7.2-7.3)	0.28
CRP (mg/l)	91 (23-215)	144 (48-203)	0.55
Lactate (mmol/l)	2.6 (1.3-7.1)	4.0 (1.2-6.6)	0.88
Urea (mmol/l)	11.1 (9.6-13.6)	11.7 (8.1-16)	0.53
Open abdomen treatment length (days)	19 (14-30)	5 (3-11)	0.00
ICU length of stay (days)	7(4-11)	5 (3-8)	0.31
Hospital length of stay (days)	40 (30-50)	10 (6-15)	0.00
Diagnosis			total
Trauma	0	1	1
Pancreatitis	1	1	2
Tumour	3	1	4
Peritonitis	1	2	3
RAAA / other bleeding	6	2	8
Sepsis	4	1	5
GI-ischemy/-occlusion	2	2	4
Other*	3	2	5

Primary decompression, decompressed during ICU-stay; secondary decompression, abdominal wall left open at the end of emergency laparotomy. *Other diagnosis: cholecystitis, incarcerated hernia, ascites, lactate acidosis, liver cirrhosis. Tumour was often combined with bleeding in abdominal cavity but classified as tumour.

Figures


A. IAP during intensive care
Non-survivors
Survivors
mmHg
25
20
15
10
5
0
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14
Days post-surgery


B. Serum lactate during intensive care
mmol/L
7,0
6,0
5,0
4,0
3,0
2,0
1,0
0,0
* * *
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14
Days post-surgery

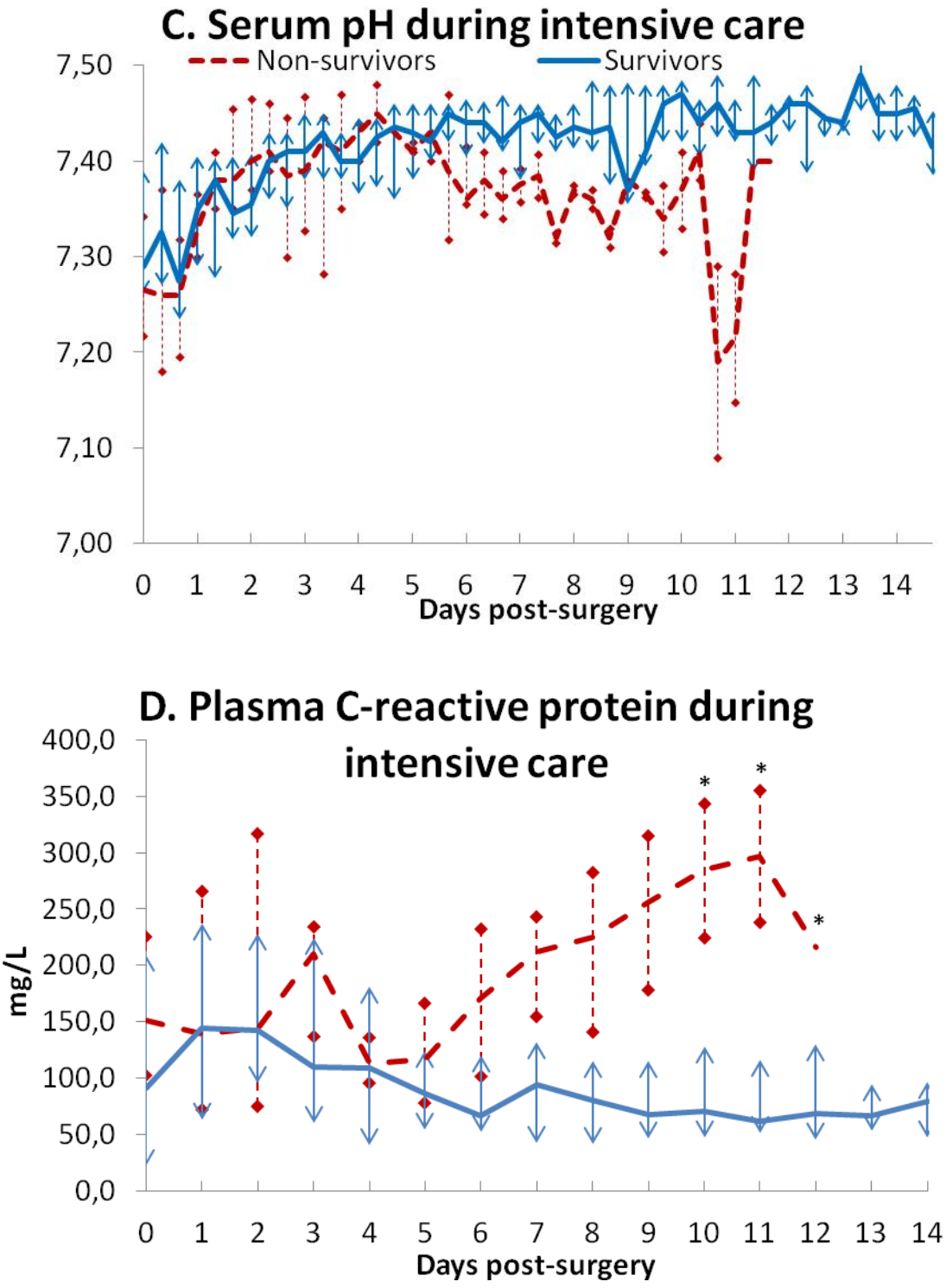


Figure 1. Laboratory values over time with IQR. *$P < 0.05$ between the groups