



UvA-DARE (Digital Academic Repository)

Treatment of ruptured abdominal aortic aneurysms in the Amsterdam area

Reimerink, J.J.

Publication date
2014

[Link to publication](#)

Citation for published version (APA):

Reimerink, J. J. (2014). *Treatment of ruptured abdominal aortic aneurysms in the Amsterdam area*. [Thesis, fully internal, Universiteit van Amsterdam].

General rights

It is not permitted to download or to forward/distribute the text or part of it without the consent of the author(s) and/or copyright holder(s), other than for strictly personal, individual use, unless the work is under an open content license (like Creative Commons).

Disclaimer/Complaints regulations

If you believe that digital publication of certain material infringes any of your rights or (privacy) interests, please let the Library know, stating your reasons. In case of a legitimate complaint, the Library will make the material inaccessible and/or remove it from the website. Please Ask the Library: <https://uba.uva.nl/en/contact>, or a letter to: Library of the University of Amsterdam, Secretariat, Singel 425, 1012 WP Amsterdam, The Netherlands. You will be contacted as soon as possible.

3

CHAPTER 3

CONTROLLED HYPOTENSION IN PATIENTS SUSPECTED OF A RUPTURED ABDOMINAL AORTIC ANEURYSM: FEASIBILITY DURING TRANSPORT BY AMBULANCE SERVICES AND POSSIBLE HARM

Jorik J Reimerink
Liselot L Hoornweg
Anco Vahl
Willem Wisselink
Ron Balm

ABSTRACT

Objective -To evaluate a controlled hypotension protocol for patients suspected of a ruptured aneurysm of the abdominal aorta (rAAA) and to identify possible harm to patients with a final diagnosis other than rAAA.

Design - Retrospective analysis of patients suspected of rAAA and transported by Amsterdam ambulance services between Jan 2006 and Oct 2007.

Methods - Protocol was assessed by reviewing systolic blood pressure (<80mmHg, 80-100mmHg or >100mmHg), administered fluid volume and verbal responsiveness during transport. Patients that could possibly have been harmed by controlled hypotension were identified by final diagnoses.

Results - Fluid administration was according to protocol in 220 of 266 patients analyzed for protocol adherence. The remaining patients received too much (21 patients) or too little fluid (25 patients). Data was missing in 29 patients. A rAAA was diagnosed in 81 (27%) of all 295 patients analyzed for final diagnosis. Controlled hypotension was achieved in 10% of all patients and in 17% of patients with rAAA. Three patients (1%) with diagnosis other than rAAA were possibly at risk by implementing controlled hypotension

Conclusions - Protocol was followed in 83% and protocol violations occurred in 17% of patients. The risk of implementing controlled hypotension for all patients suspected of a rAAA by the ambulance staff was low.

INTRODUCTION

Rupture of an abdominal aortic aneurysm is associated with an overall mortality of 80-90%.^{1,2} Patients with a ruptured aneurysm of the abdominal aorta (rAAA) are typically in hemorrhagic, hypovolemic shock. Hypovolemic shock is treated by administering intravenous fluids. However, in aggressive intravenous fluid administration, the red blood cell count, platelet count and clotting factors and thus coagulation and oxygen delivery will decrease due to dilution.³⁻⁶ Restricting fluid resuscitation and accepting lower systolic blood pressures, until hemorrhage is controlled, is key in these patients. This is called controlled or permissive hypotension. The positive effect of controlled hypotension was demonstrated in animal studies and in patients with blunt and penetrating injuries.³⁻⁶ Controlled hypotension is beneficial for patients with ruptured aneurysms and is thought to improve survival rate.⁷⁻¹¹

The Amsterdam Acute Aneurysm or AJAX Trial is a multi center randomized trial (ISRCTN 66212637), designed to compare conventional open repair with endovascular repair for patients with a proven rAAA.¹² In this trial we also register all patients in the Amsterdam region (1.3 million inhabitants) that are suspected of a rAAA in a prospective database.¹² These patients are transported to one of three trial centers for evaluation and possibly treatment.¹² Because of this regionalisation, transport times may be extended.

To optimize conditions for patients with a rAAA during transport, a protocol for controlled hypotension was designed in which a target systolic blood pressure of 80-100 mmHg was chosen for all patients suspected of rAAA.¹² However, not all patients suspected of rAAA in the ambulance, are diagnosed with rAAA. Patients with a final diagnosis other than rAAA are also treated according to the controlled hypotension protocol before a diagnosis is made. In these patients, delaying fluid resuscitation might have been harmful.^{13,14}

The aim of this study is to evaluate our existing protocol of controlled hypotension for patients suspected of rAAA in the ambulance and to determine if, based on the final in-hospital diagnosis, patients with final diagnoses other than rAAA were potentially harmed by controlled hypotension.

METHODS

Patient selection

Patients were selected from the cohort of patients with suspected rAAA from the Amsterdam Acute Aneurysm trial. We retrospectively reviewed all patients suspected of rAAA being transported by the Amsterdam ambulance services between January 1st 2006 until October 10th 2007. Based on the suspicion of rAAA, all these patients should have been exposed to the controlled hypotension protocol. Patients referred from other hospitals, patients that were not presented by regional ambulance service and patients with a suspicion other than rAAA before presentation, were not exposed to our controlled hypotension protocol and were excluded from this analysis.

Parameters retrieved from the ambulance ride registration forms were: systolic blood pressure, administered volume of fluid, verbal unresponsiveness and necessity for intubation or cardiopulmonary resuscitation (CPR). The interval between arrival at the patient and arrival at the hospital was the time in which controlled hypotension could be applied and was also reviewed. Patients finally diagnosed as rAAA were analyzed as a subgroup.

Protocol adherence

According to our protocol, the target systolic blood pressure of patients suspected of rAAA is 80-100 mmHg. All patients are allowed standard intravenous drip. A fluid challenge is allowed if the blood pressure is below 80mmHg or if a patient is verbally unresponsive. Fluid administration should be restricted when a blood pressure of 80-100mmHg is achieved. In patients with a blood pressure >100mmHg, no active blood pressure lowering is performed but fluid administration should be restricted.

Patients were subdivided according to systolic blood pressure at presentation; <80 mmHg, 80-100mmHg and \geq 100mmHg. We then assessed if systolic blood pressure remained stable, increased or decreased by comparing first recorded blood pressure with last recorded blood pressure. If only one blood pressure was known, patients were considered stable at that particular blood pressure. We reviewed the total volume of administered intravenous fluid and assessed if this was according to protocol. The volume was reported by the number of 500ml bags of fluid used. Volume administration up to 500ml was allowed in all patients and seen as part of the standard intravenous drip. Fluid administration of more than 500ml was considered a fluid challenge.

We assessed if protocol was followed and if patients in whom the blood pressure remained <80mmHg or decreased to less than 80 mmHg had received a proper fluid challenge. We also assessed if patients with a final blood pressure of >100mmHg were indeed restricted in fluid administration. A fluid challenge was always allowed if the final blood pressure of patients was between 80-100mmHg. All verbally unresponsive patients were also allowed a fluid challenge disregarding their blood pressure. We considered a patient to be controlled hypotensive if the final blood pressure was between 80-100mmHg and the patient was verbally responsive.

Diagnoses and possible harm

To identify patients that could possibly be harmed by controlled hypotension, final diagnoses of all patients exposed to our controlled hypotension protocol were identified from emergency room files and correspondence. Patients with diseases other than rAAA known to induce shock and possibly requiring treatment by fluid resuscitation, were reviewed for presence of hypotension and fluid restriction. These patients could have been harmed by controlled hypotension. The possible harm was expressed as mortality of this sub group of patients. Since the Amsterdam aneurysm trial is an ongoing trial, data on mortality of patients diagnosed rAAA can not be released.

RESULTS

The patient cohort for the Amsterdam acute aneurysm study registered 295 patients with a suspected rAAA that were transported by regional ambulance services between January 1st 2006 until October 10th 2007. Patient characteristics show a mean age of 71.9 years (SD 11.5) with a male predominance of 74.3%. Of 29 patients data were missing because blood pressure readings were not recorded during transport (13) or because ambulance ride registration forms could not be retrieved (16). The remaining 266 patients were reviewed for protocol adherence during transport. Final diagnoses were analysed in all 295 patients. The mean time between the ambulance arriving at the patient and arriving at the hospital was 31.5 minutes (CI: 30.1 – 32.9 min).

Systolic blood pressure (n=266)

Initially, as measured by the ambulance staff, 41 patients (15%) had a systolic blood pressure of <80mmHg, 36 patients (14%) had a systolic blood pressure of 80-100mmHg and 189 patients (71%) had a systolic blood pressure of \geq 100mmHg. (*Table 1*)

Of those patients presenting with a blood pressure <80mmHg, the pressure remained <80 mmHg in 24 patients and increased to >80mmHg in 17 patients (80-100mmHg in 7 patients and to >100mmHg in 10 patients). Of those 24 patients with persisting low blood pressure (<80mmHg) 12 were verbally unresponsive of which 10 needed CPR and 8 were intubated. Six of these 24 patients died before or during transport. CPR was applied in 4 out of the 17 patients in whom the blood pressure rose to >80mmHg afterwards.

Thirty-six patients presented with an initial blood pressure of 80-100mmHg. Blood pressure decreased to <80mmHg in 3 patients, remained stable at 80-100mmHg in 16 patients and increased to >100mmHg in 17 patients. One of the patients in whom the blood pressure had dropped was intubated precautionary because of a brief period of unresponsiveness. Two other patients out of these 36 patients were verbally unresponsive for a longer period of time.

One-hundred-and-eighty-nine patients presented with an initial blood pressure of >100mmHg. Blood pressure decreased to <80mmHg in 8 patients, to 80-100mmHg in 6 patients and remained >100mmHg in 175 patients. Four of the patients in whom blood pressure dropped became unresponsive during transport and CPR and intubation were necessary in three of these four patients.

Thirty-five patients had a final systolic blood pressure <80mmHg, 29 patients had a final blood pressure of 80-100mmHg and 202 patients had a final blood pressure >100mmHg. Of all 29 patients with a final blood pressure between the target values of 80-100mmHg, 27 patients were verbally responsive, therefore they could be considered "controlled hypotensive". This represents 10% of all patients analysed for protocol adherence.

Protocol adherence (n=266)

One-hundred-and-thirteen patients did not receive any intravenous fluids, 113 patients received <500ml, 28 patients received a fluid challenge between 500-1000ml and 12 patients received a fluid challenge of >1000ml.

Based on the blood pressure alone, fluid administration was according to protocol in 218 patients (82%), 23 patients (9%) received a fluid challenge despite sufficient blood pressures and 25 patients (9%) did not receive a fluid challenge despite their final blood pressure of less than 80 mmHg.

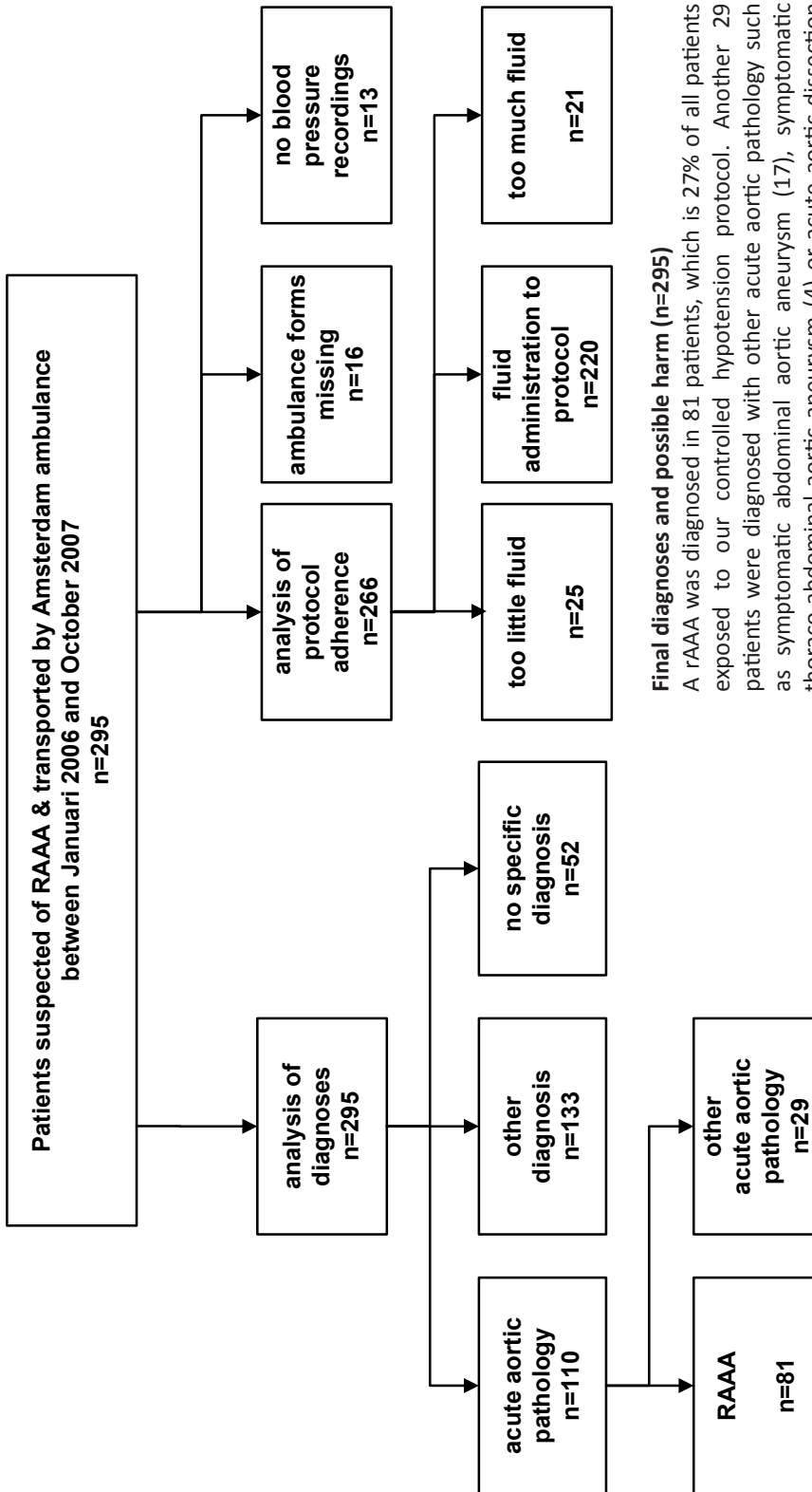
When taking the clinical condition of the patients into consideration, it appears that out of 23 patients who received a fluid challenge despite adequate blood pressure, two were verbally unresponsive thereby justifying the fluid challenge. So based on both clinical status and blood pressure, 220 patients (83%) were treated according to protocol and 46 patients (17%) were not. Additionally, 12 out of 25 patients that did not receive a fluid challenge despite a low systolic blood pressure were responsive and hemodynamically stable with a blood pressure just below 80mmHg. In another 6 patients with a final systolic blood pressure <80mmHg, attempts to obtain peripheral intravenous access were unsuccessful so no fluid challenge could be administered. (*table 1*).

Table 1; Systolic blood pressures and fluid administration

initial SBP	final SBP	<500ml	>500ml	verbally unresponsive	CPR	intubated
<80mmHg n=41	<80mmHg n=24	19(-)	5	12	10	8
	80-100mmHg n=7	5	2	1	1	-
	>100mmHg n=10	6	3(+) 1(+u)	4	3	3
80-100mmHg n=36	<80mmHg n=3	2(-)	1	-	-	1
	80-100mmHg n=16	12	4	1	-	-
	>100mmHg n=17	12	4(+) 1(+u)	1	-	-
>100mmHg n=189	<80mmHg n=8	4(-)	4	4	3	3
	80-100mmHg n=6	5	1	-	-	-
	>100mmHg n=175	161	14(+)	-	-	-
total	266	226	40	23	17	15

All patients (266) suspected of rAAA during transport by regional ambulance and analyzed for protocol adherence are displayed. Patients are divided horizontally according to systolic blood pressure (SBP) at presentation and final SBP.

Vertically, a division is made between patients with a total volume administered of <500ml and >500ml. Protocol violation by administering a fluid challenge when not indicated are marked as (+). Protocol violations made by restricting fluid with low blood pressures are marked as (-). Patients where fluid challenge was justified because of verbal unresponsiveness, despite sufficient blood pressure, are marked (+u) Absolute numbers of verbally unresponsive patients, patients where CPR or intubation was necessary are displayed in the final 3 vertical columns.



Final diagnoses and possible harm (n=295)

A rAAA was diagnosed in 81 patients, which is 27% of all patients exposed to our controlled hypotension protocol. Another 29 patients were diagnosed with other acute aortic pathology such as symptomatic abdominal aortic aneurysm (17), symptomatic thoraco-abdominal aortic aneurysm (4) or acute aortic dissection (4). Other diagnoses varied and were grouped according to organ

Final diagnoses and possible harm (n=295)

A rAAA was diagnosed in 81 patients, which is 27% of all patients exposed to our controlled hypotension protocol. Another 29 patients were diagnosed with other acute aortic pathology such as symptomatic abdominal aortic aneurysm (17), symptomatic thoraco-abdominal aortic aneurysm (4) or acute aortic dissection (4). Other diagnoses varied and were grouped according to organ system. (Table 2). Fifteen patients died before a diagnosis could be established and no post-mortem examination was performed. In 5 of these 15 patients an rAAA was excluded by ultrasonography. Of 37 patients no specific diagnosis could be retrieved from the ambulance or hospital registration systems.

In 22 patients we registered a final diagnosis, other than acute aortic pathology, that could induce hemodynamic shock. Common final diagnoses among these patients were acute pancreatitis (7), gastrointestinal perforation (4) and acute cholecystitis (4). Of these 22 patients, 4 were hypotensive at some stage during transport. Three of these 4 patients were also restricted in fluid administration. One of these last three patients, diagnosed with severe acute pancreatitis, died within 24 hours after admission. Two other patients out of 22 died within 24 hours. Both these patients were hemodynamically stable with blood pressures >100mmHg during transport. It is possible that some of the 15 patients that died before a diagnosis was made were also suffering from a disease other than rAAA known to induce hemodynamic shock, and were thus possibly harmed by controlled hypotension protocol. The majority of these patients (10/15) was in deep hemodynamic shock upon arrival of the ambulance and 5 of them died after unsuccessful CPR before transport. Intravenous access was not obtained in all 5 of these patients.

Table 2 Diagnoses in order of frequency

diagnosis	n	percentage
rAAA	81	27.5%
acute aortic pathology	symptomatic AAA 17	5.7%
	other 12	4.1%
gastrointestinal	51	17.3%
non-specific pain	23	7.8%
neuro/systemic	21	7.1%
hepatobiliary	18	6.1%
renal/urogenital	13	4.4%
cardiac	7	2.4%
died before diagnosis	15	5.1%
no specific diagnosis	37	12.5%
total	295	100%

Final diagnoses of patients suspected of rAAA and transported by regional ambulance services. Diagnoses are grouped according to organ system. Neurological and systemic diagnoses are grouped together

rAAA (n=81)

In total; 81 patients were diagnosed with a rAAA and exposed to our controlled hypotension protocol. Seventeen patients had a final systolic blood pressure <80mmHg, in 13 patients blood pressure was in the target range of 80-100mmHg. In 11 patients the pressure rose to >100mmHg during transport and 30 patients had a stable blood pressure >100mmHg. No blood pressure readings were recorded in 4 patients, ambulance registration forms were not retrieved in 6 patients with proven rAAA. Seven of 81 patients diagnosed with rAAA needed CPR during transport of which one of these 7 was stabilized quickly; the other 6 patients were intubated. In total 8 patients were verbally unresponsive during transport.

Protocol adherence could be analyzed in 71 patients of whom blood pressure recordings were available. A protocol violation by withholding a fluid challenge despite a blood pressure <80mmHg was made in 11 patients. Seven patients were administered a fluid challenge despite adequate blood pressures. In total 18 protocol violations (25%) were made in patients diagnosed rAAA, Protocol was followed in the remaining 53 patients (75%).

Of the 13 patients with a final blood pressure of 80-100, 12 patients (17%) were verbally responsive and stable, they were considered controlled hypotensive.

DISCUSSION

The majority (71%) of patients exposed to controlled hypotension protocol during transport and analyzed for protocol adherence, was presented with a systolic blood pressure of ≥ 100 mmHg. In our protocol we do not provide for active medical blood pressure reduction. The result is that controlled hypotension according to our protocol is not a realistic goal in most patients suspected of rAAA in the Amsterdam region. Based on the final blood pressure, only 10% of all patients (27/266) and 17% of patients diagnosed rAAA (12/71) can be categorized as truly controlled hypotensive according to our criteria.

The high percentage of normotensive patients in the entire group is probably explained by the fact that in all 295 patients only 81 (27%) suffered from a rAAA and a combined 110 (37%) from acute aortic pathology. Although the diagnostic accuracy of 27% for true rAAA is low, it does represent the actual situation of patients transported by Amsterdam ambulance where the clinical diagnosis possible rAAA is made. It is likely that the ongoing multicentre aneurysm trial in our region has increased awareness of the ambulance staff for possible rAAA. This could have decreased diagnostic accuracy.

Overall, controlled hypotension protocol was followed in 83% of all patients and in 75% of patients diagnosed with rAAA. Of course, protocol violations should always be avoided. More protocol violations were observed by withholding a fluid challenge to patients than by administering too much fluid. However 12 of these 25 patients remained verbally responsive and stable. They might have had sufficient blood pressures although their blood pressures were below the target range we specified. In another six patients no intravenous access was obtained due to extensive hemodynamic shock and therefore these protocol violations were difficult to prevent.

A total of 113 patients did not receive any intravenous fluids. For patients with a suspected rAAA, an intravenous drip as a precaution is advisable. We did not review presence of intravenous access itself, only the volume of fluid administered. Most of the patients that were not administered any fluid were hemodynamically stable with a blood pressure of >100mmHg. It is possible these patients were administered so little fluid this was not registered on the ambulance ride registration forms.

The ideal systolic blood pressure for patients with rAAA is not known and probably depends on co-morbidity and individual history.⁸ If a patient remains conscious and has no signs of ischemia, blood pressure is believed to be sufficient.⁸ In other studies lower target values for systolic blood pressure are described.^{7,9,15} For practical reasons we chose 80-100 mmHg as target value for ambulance personnel. We believe that this is a target range that can be achieved in most patients without jeopardising those patients with other diagnoses than rAAA that might be harmed by low blood pressures. With our protocol we intended to endorse a less aggressive fluid resuscitation policy in normotensive patients. With only 12 patients administered >1000ml we seem to have achieved that goal.

Several other studies describe active lowering of blood pressure to reach the target range by medication such as nitroglycerine or esmolol.^{9,15} However, because of the high percentage of patients diagnosed with a different disease in our series, we question the applicability and safety of this policy for all patients suspected of rAAA in the ambulance.

Because of the small number of patients at risk, the possible harm by controlled hypotension for patients with other final diagnoses than rAAA was difficult to objectify. Of those patients with a final diagnosis other than rAAA that could possibly induce shock, only a small number (4) was actually hypotensive and even a smaller number (3) was also restricted in fluid administration. One of these patients died within a day after admission. It cannot be ruled out that aggressive resuscitation in this patient could have had beneficial effects. Fifteen patients died before a diagnosis was made. It is possible some of these patients suffered from a disease that would have benefitted from aggressive resuscitation. We can only assume the real harm done by implementing controlled hypotension in all patients suspected of rAAA is very low. A longer duration of transport might increase the possible harm.

In order to even lower the possible harm, a higher accuracy of suspicion could be achieved by implementing evaluation with ultrasound in an early stage. Several studies report the usefulness of an on-board ultrasound.^{16,17} This could lead to a higher accuracy and thus more adequate treatment including medical treatment of hypertension in the prehospital environment.

Because this is a retrospective study, the precise relation of infused fluid and blood pressure is difficult to analyse. Also, the retrospective interpretation of hemodynamic instability and necessity for fluid administration from an ambulance form is difficult. In 37 patients no specific diagnosis was made or recorded. It is not likely any of these patients suffered from serious illness, otherwise more data would be recorded. It is however possible that some of 10 patients that died before diagnosis without ruling out an AAA indeed had a rAAA.

In conclusion; controlled hypotension protocol was followed in 83% and protocol violations occurred in 17% of all patients transported with possible rAAA. True controlled hypotension was achieved in only 10% (27/266) of all patients and in 17% (12/71) of patients diagnosed with rAAA. The diagnostic accuracy of the ambulance staff for rAAA was 27% (81/295). Half of the patients with rAAA were hemodynamically stable with final blood pressures >100mmHg. Based on final diagnoses, the risk of implementing controlled hypotension for all patients suspected of a rAAA by the ambulance staff was low.

REFERENCE LIST

- 1 Hoornweg LL, Storm-Versloot MN, Ubbink DT, Koelemay MJ, Legemate DA, Balm R. Meta analysis on mortality of ruptured abdominal aortic aneurysms. *Eur J Vasc Endovasc Surg* 2008; 35:558-70.
- 2 Kantonen I, Lepantaló M, Brommels M, Luther M, Salenius JP, Ylonen K. Mortality in ruptured abdominal aortic aneurysms. The Finnvasc Study Group. *Eur J Vasc Endovasc Surg* 1999; 17:208-12.
- 3 Bickell WH, Wall MJ, Jr, Pepe PE, Martin RR, Ginger VF, Allen MK, et al. Immediate versus delayed fluid resuscitation for hypotensive patients with penetrating torso injuries. *N Engl J Med* 1994; 331:1105-9.
- 4 Bickell WH, Stern S. Fluid replacement for hypotensive injury victims: how, when and what risks? *Curr Opin Anaesthesiol* 1998; 11:177-80.
- 5 Mapstone J, Roberts I, Evans P. Fluid resuscitation strategies: a systematic review of animal trials. *J Trauma* 2003; 55:571-89.
- 6 Revell M, Greaves I, Porter K. Endpoints for fluid resuscitation in hemorrhagic shock. *J Trauma* 2003; 54:S63-S67.
- 7 Crawford ES. Ruptured abdominal aortic aneurysm. *J Vasc Surg* 1991; 13:348-50.
- 8 Roberts K, Revell M, Youssef H, Bradbury AW, Adam DJ. Hypotensive resuscitation in patients with ruptured abdominal aortic aneurysm. *Eur J Vasc Endovasc Surg* 2006; 31:339-44.
- 9 van der Vliet J, van Aalst DL, Schultze Kool LJ, Wever JJ, Blankensteijn JD. Hypotensive hemostatis (permissive hypotension) for ruptured abdominal aortic aneurysm: are we really in control? *Vascular* 2007; 15:197-200.
- 10 Veith FJ, Ohki T. Endovascular approaches to ruptured infrarenal aorto-iliac aneurysms. *J Cardiovasc Surg (Torino)* 2002; 43:369-78.
- 11 Veith FJ, Gargiulo NJ. Endovascular aortic repair should be the gold standard for ruptured AAAs, and all vascular surgeons should be prepared to perform them. *Perspect Vasc Surg Endovasc Ther* 2007; 19:275-82.
- 12 Amsterdam Acute Aneurysm trial Collaborators. Amsterdam Acute Aneurysm trial: background, design, and methods. *Vascular* 2006; 14:130-5.
- 13 Mann HJ, Nolan PE, Jr. Update on the management of cardiogenic shock. *Curr Opin Crit Care* 2006; 12:431-6.
- 14 Vincent JL, Gerlach H. Fluid resuscitation in severe sepsis and septic shock: an evidence-based review. *Crit Care Med* 2004; 32:S451-S454.
- 15 Mayer D, Pfammatter T, Rancic Z, Hechelhammer L, Wilhelm M, Veith FJ, et al. 10 years of emergency endovascular aneurysm repair for ruptured abdominal aortoiliac aneurysms: lessons learned. *Ann Surg* 2009; 249:510-5.
- 16 Lapostolle F, Petrovic T, Lenoir G, Catineau J, Galinski M, Metzger J, et al. Usefulness of handheld ultrasound devices in out-of-hospital diagnosis performed by emergency physicians. *Am J Emerg Med* 2006; 24:237-42.
- 17 Mazur SM, Sharley P. The use of point-of-care ultrasound by a critical care retrieval team to diagnose acute abdominal aortic aneurysm in the field. *Emerg Med Australas* 2007; 19:71-5.