# Aortic endograft sizing in trauma patients with hemodynamic instability

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*Objectives:* To investigate changes in aortic diameter in hemodynamically unstable trauma patients and the implications for sizing of thoracic endovascular aortic repair (TEVAR) in patients with traumatic thoracic aortic injury (TTAI).

*Methods:* We retrospectively evaluated all trauma patients that were admitted with hemodynamic instability (mean arterial pressure <95 mm Hg and a pulse ≥100 beats/min) and underwent computed tomography (CT) of the thorax and abdomen both at admission and at another moment (control CT scan), at the Yale New Haven Hospital between 2002 and 2009. The CT examinations were reviewed in a blinded fashion and the aortic diameter was measured at six different levels by a cardiovascular radiologist. Differences in aortic diameter between the initial CTs obtained in the trauma bay and the control CTs were compared using the paired Student *t* test.

*Results:* Forty-three patients were identified, including 32 males. Mean age was 37 ± 16 years, mean injury severity score was 26 ± 15, the mean pulse and blood pressure were 122 beats/min and 103/63 mm Hg, respectively. Overall, the mean aortic diameter was significantly larger at the control CT examinations compared with the initial CT examinations while hemodynamically unstable, at all evaluated levels. Among patients with a pulse ≥130/min, the mean increase in aortic diameter was most consistent at the level of the mid descending thoracic aorta (DTA, +12.6%, *P* = .003) and at the level of the infrarenal aorta (+12.6%, *P* = .004).

*Conclusions:* The aortic diameter decreases dramatically in trauma patients with hemodynamic instability. This decrease in aortic diameter could theoretically lead to inaccurate aortic measurements and undersizing of the endograft in hemodynamically unstable TTAI patients requiring TEVAR. Further research is needed to better predict the actual aortic diameters in individual hemodynamically unstable patients requiring endovascular aortic repair. ( J Vasc Surg 2010;52:39-44.)

Traumatic thoracic aortic injury (TTAI) is the second most common cause of death in trauma patients, accounting for about 8000 deaths per year in the United States.[1,2] Thoracic endovascular aortic repair (TEVAR) has recently offered a less invasive method for the management of TTAI. Large series and meta-analyses have shown that endovascular repair of TTAI significantly reduces the in-hospital mortality and morbidity compared with conventional open surgical repair,[3-7] and that a major shift has occurred in favor of endovascular management of TTAI.[3,8,9] A substantial part of admitted patients with TTAI have multiple injuries with associated hemodynamic instability

or hypovolemic shock, a leading cause of death in trauma patients worldwide.[10]

Hemodynamic forces may result in significant conformation changes of the thoracic aorta.[11] However, currently it remains unclear how the aorta reacts to hemodynamic instability or hypovolemic shock. A decrease in the aortic diameter of 30% has been described in an anecdotal case report of a hypovolemic patient with TTAI.[12] Preoperative computed tomography angiography (CTA) is typically used for endograft sizing in patients requiring TEVAR, and considerably decreased aortic diameters in patients undergoing CTA could result in inaccurate aortic measurements and undersizing of the endograft.

In the present study, potential aortic changes were investigated in trauma patients that were admitted with hemodynamic instability, to better understand the dynamic morphology of the aorta, and to optimize endograft sizing in TTAI patients.

## METHODS

**Study population.** A retrospective trauma registry inquiry (Clinical Data Management, Denver, Colo) identified all trauma patients that were admitted with hemodynamic instability at the emergency department of an ACS-verified and state-designated level I trauma center, between July 1, 2002 and July 1, 2009. The study was approved by the institutional review committee of the Yale University School of Medicine. Admission demographics and recorded vital signs were used to select the patients suffering

from hemodynamic instability following traumatic injuries. Inclusion criteria were: (1) multiple traumatic injuries, (2) a mean arterial pressure ≤95 mm Hg and pulse ≥100 beats/min, and (3) computed tomography (CT) examinations of the thorax and abdomen were performed both at admission and at another moment when the patients was thought to be hemodynamically stable (control CT scan). Exclusion criteria were: (1) patients younger than 18 years of age, (2) incomplete data sets, and (3) a time interval between both CT examinations of more than 12 months.

**Data collection.** All CT examinations were performed on either a General Electric (Milwaukee, Wisc) 4-slice or 64-slice volume CT system. The CT studies were nongated and were reviewed in a blinded fashion by a cardiovascular radiologist with 5 years of experience. Axial CT images were used to measure the aortic diameter at six different levels including the ascending aorta at the level of the pulmonary artery, the mid aortic arch, the proximal descending thoracic aorta (DTA) at the level of the termination of the aortic arch, the mid DTA, the distal DTA 1 cm above the celiac axis, and the infrarenal abdominal aorta. If the aorta was angulated, multiplanar reformations were used to measure the proper aortic diameter perpendicular to the long axis of the aorta. Subsequently, the maximum and minimum diameters of the inferior vena cava (IVC) were measured at the level of the mid infrarenal IVC halfway between the most superior renal vein and the convergence of the common iliac veins.

**Statistical analysis.** The diameter measurements of the aorta and IVC were compared in all patients between the CTs at admission and the control CTs. The paired Student $t$ test was used to investigate potential diameter differences in the overall cohort and among patients with a pulse above 130 beats/min. SPSS software version 15.0 was used for all statistical analyses, a $P$ value <.05 was considered statistically significant.

## RESULTS

**Admission characteristics.** Forty-three patients were selected for evaluation. The median time interval between the measurement of the vital signs and the CT examination was 30 minutes (interquartile range 26 minutes; range, 0 to 97 minutes), and the median time interval between the CT examination at admission to the emergency department while hemodynamically unstable, and the control CT examination was 11 days (interquartile range, 28 days; range, 1.5 days to 351 days). The mean age was 37.2 ± 16 years (range, 18 to 90 years), and 74% were male (Table I). Mean pulse was 121.6 ± 18 beats/min (range, 100 to 170), and 27.9% (n = 12) had a pulse ≥130/min. The mean arterial pressure (MAP) of the cohort was 77.3 mm Hg (range, 54.3 to 94 mm Hg), the mean respiratory rate was 21.9 (range, 12 to 35), and 16.3% (n = 7) were already intubated and mechanically ventilated when the first vital signs were recorded at the emergency department.

**Changes in aortic diameter.** Overall, the mean aortic diameter was significantly larger at the control CT examinations compared with the CT examinations at admission

**Table I.** Admission characteristics

|  | *N or mean* | *(% or ± SD)* |
|---|---|---|
| Age | 37.2 | (±16) |
| Male gender | 32 | (74) |
| Trauma |  |  |
|   Motor vehicle crash | 28 | (65) |
|   Gunshot/stab wounds | 7 | (16) |
|   Fall | 8 | (19) |
| Injury severity score | 25.7 | (±16) |
| Vital signs |  |  |
|   Pulse | 121.6 | (±18) |
|   Systolic pressure | 103.4 | (±16) |
|   Diastolic pressure | 63.3 | (±12) |
|   MAP | 77.3 | (±12) |
|   Respiratory rate | 21.9 | (±6.1) |
|   Hematocrit | 35.5 | (±7.5) |

*MAP,* Mean arterial pressure; *SD,* standard deviation.

while hemodynamically unstable, at all evaluated levels (Table II). The overall mean increase in aortic diameter varied from 3.3% ($P$ = .012) at the level of the ascending aorta, to 11.2% ($P$ < .001) at the level of the infrarenal aorta (Table II). The increase in aortic diameter varied considerably among individual patients as well, and in some cases, however, no increase in aortic diameter, or even a smaller aortic diameter was observed on the control CT examination (Table II). The correlation between the MAP of the trauma patients, while hemodynamically unstable, and the increase in aortic diameter at the level of the mid DTA on the control CT is depicted in Fig 1.

The mean increase in aortic diameter at the different levels appeared to be larger among patients admitted with a pulse ≥130 (Table III, Fig 2). In this subgroup, the mean increase in aortic diameter was most consistent at the level of the mid DTA (+12.6%, $P$ = .003) and at the level of the infrarenal aorta (+12.6%, $P$ = .004). At the remaining levels, the mean increase in aortic diameter failed to reach statistical significance (Table III), most likely due to the small sample size (n = 12).

**Conformational changes of the IVC.** Overall, the maximum and minimum IVC diameters were 8.5% ($P$ = .002) and 36.2% ($P$ < .001) larger on the control CT examination compared with the CT at admission while hemodynamically unstable. Since the increase in minimum IVC diameter was substantially larger than the increase in the maximum IVC diameter, it appears that the shape of the IVC changed as well, from an elliptic shape to a more round shape on the control CTs. The change in IVC measurements varied considerably among individual patients, and in some cases, no increase or even a decrease in the minimum and/or maximum diameter of the IVC was observed on the control CT examination (Table II).

The mean increase in IVC diameters appeared to be larger among patients admitted with a pulse ≥130 (Table III). In this group, the maximum and minimum IVC diameters increased with 15.0% ($P$ = .028) and 40.0% ($P$ = .016), respectively.

**Table II.** Mean diameter changes of the aorta and IVC (N = 43)

| Level | HD unstable (mm) | Control (mm) | Diff (mm) | Increase (%) | Range (%) | P value |
|---|---|---|---|---|---|---|
| Aorta | | | | | | |
| Ascending | 27.4 | 28.3 | 0.9 | 3.3 | −10.8-19.8 | .012 |
| Arch | 22.2 | 23.6 | 1.4 | 6.3 | −7.5-26.1 | .011 |
| Proximal DTA | 21.9 | 23.1 | 1.2 | 5.5 | −6.7-38.8 | .010 |
| Mid DTA | 19.2 | 21.1 | 1.8 | 9.4 | −8.0-40.0 | <.001 |
| Distal DTA | 18.1 | 19.4 | 1.3 | 7.2 | −7.9-54.3 | <.001 |
| Infrarenal | 14.3 | 15.9 | 1.6 | 11.2 | −5.9-44.9 | <.001 |
| Inferior vena cava | | | | | | |
| Max diameter | 19.9 | 21.6 | 1.7 | 8.5 | −8.3-49.1 | .002 |
| Min diameter | 11.6 | 15.8 | 4.2 | 36.2 | −12.8-203.1 | <.001 |

*Diff,* Difference in mm; *DTA,* descending thoracic aorta; *HD unstable,* hemodynamically unstable; *IVC,* inferior vena cava.
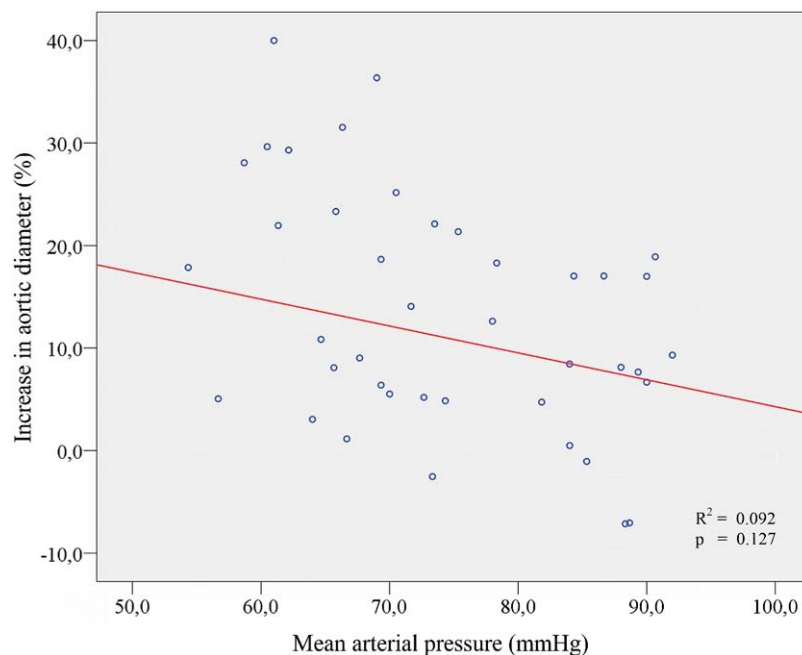


**Fig 1.** MAP of trauma patients while hemodynamically unstable and increase in aortic diameter at the level of the mid DTA on the control CT. *CT,* Computed tomography; *DTA,* descending thoracic aorta; *MAP,* mean arterial pressure.

## DISCUSSION

Endovascular repair has shown to reduce the in-hospital mortality and morbidity of TTAI compared with conventional open surgical repair,[3-7] and a major shift has recently occurred from open surgery toward endovascular management of TTAI.[3,8,9] However, endovascular management of TTAI is still plagued by considerable endoleak rates, typically ranging between 2.8% to 14.3%.[3-6,13-15] The majority of these endoleaks are proximal type 1 endoleaks, which often require reintervention or even explantation of the endograft.[4,14,15]

Several factors may attribute to the relatively high occurrence of endoleak in this patient group. Blunt TTAI is typically located at the aortic isthmus distal to the left subclavian artery.[4,16-18] Endografts may conform poorly to the inner curvature of the aortic arch, which may result in

increased risks of endograft-related complications such as endoleak. The emergency nature may also contribute to high endoleak rates. For emergency TEVAR, physicians can only use those endografts that are available as stock. In addition, urgent situations may not allow optimal endograft sizing and deployment, which may increase risks of endoleak.

The results of this study suggest another factor that may lead to inadequate endograft sizing, namely the influence of hemodynamic instability on the aortic diameter. A considerable percentage of patients with TTAI are admitted with hemodynamic instability and/or in hypovolemic shock as a result of associated injuries. We observed that the aortic diameter was significantly smaller, from the ascending thoracic aorta to the infrarenal aorta, on the CT examination of hemodynamically unstable trauma patients, com-

**Table III.** Mean diameter changes of the aorta and IVC in patients with a pulse $\geq$130/min (N = 12)

| Level | HD unstable (mm) | Control (mm) | Diff (mm) | Increase (%) | Range (%) | P value |
|---|---|---|---|---|---|---|
| Aorta | | | | | | |
|   Ascending | 26.2 | 27.3 | 1.1 | 4.2 | −1.3-17.6 | .184 |
|   Arch | 21.1 | 22.8 | 1.7 | 8.1 | −7.5-16.3 | .382 |
|   Proximal DTA | 20.3 | 22.9 | 2.6 | 12.8 | −3.2-38.8 | .068 |
|   Mid DTA | 18.3 | 20.6 | 2.3 | 12.6 | 6.7-36.4 | .003 |
|   Distal DTA | 18.2 | 19.4 | 1.2 | 6.6 | −7.3-25.7 | .133 |
|   Infrarenal | 14.3 | 16.1 | 1.8 | 12.6 | 3.0-33.9 | .004 |
| Inferior vena cava | | | | | | |
|   Max diameter | 19.3 | 22.2 | 2.9 | 15.0 | −6.4-48.7 | .028 |
|   Min diameter | 11.5 | 16.1 | 4.6 | 40.0 | −12.8-170.0 | .016 |

*Diff,* Difference in mm; *DTA,* descending thoracic aorta; *HD unstable,* hemodynamically unstable; *IVC,* inferior vena cava.
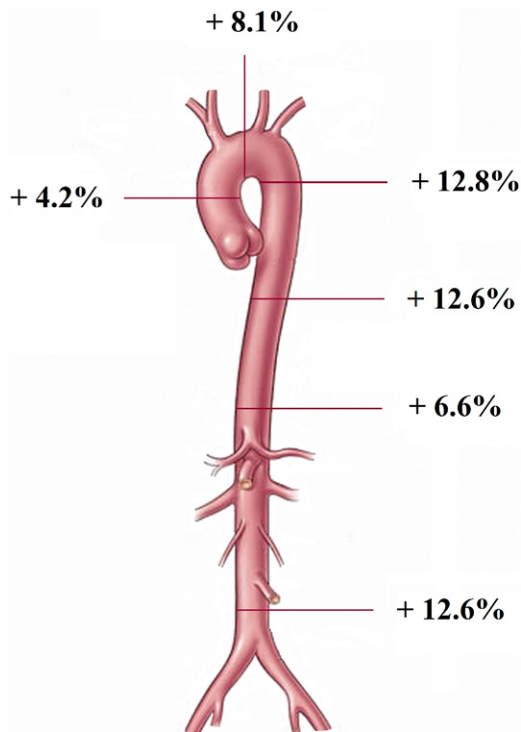


**Fig 2.** Mean increase in aortic diameter in patients with a pulse $\geq$130/min. The mean increase in aortic diameter was most consistent at the level of the mid descending thoracic aorta ($P$ = .003), and at the level of the infrarenal aorta ($P$ = .004), the mean increase in aortic diameter failed to reach statistical significance at the remaining levels.

pared with the control CT. In some cases, the aortic diameter was up to 40% larger on the control CT examination compared with the CT examination while hemodynamically unstable at admission. A theoretic explanation for this observation may be that substantial blood loss led to a shortage in circulating blood volume, which subsequently may have resulted in decreased pressure on the aortic wall, causing the aorta to collapse. However, reliable data regarding the circulating blood volume or the resuscitation status of the trauma patients were not available for this evaluation, so we cannot confirm this hypothesis. Van Prehn et al have previously described a hypovolemic patient with TTAI that successfully underwent TEVAR, but the postoperative CTA showed an increase in aortic diameter of 30%.[12] Except for this case report, no studies have previously investigated the influence of hemodynamic instability or hypovolemic shock on the aortic diameter to our knowledge, and the present patient series therefore represents the first study that has demonstrated significantly decreased aortic diameters in hemodynamically unstable trauma patients.

These findings may have implications for the endovascular management of acute thoracic aortic disease. Preoperative CTA is typically used for aortic measurements and endograft sizing in patients requiring TEVAR. However, considerably decreased aortic diameters in a hemodynamically unstable TTAI patient undergoing CTA likely result in inaccurate aortic measurements and undersizing of the endograft. Theoretically, an inadequate proximal seal of the endograft may lead to increased risks of type 1 endoleaks, although there is currently no evidence available that undersizing during TEVAR is associated with type 1 endoleak. Most endograft manufacturers recommend oversizing by 10% to 20%. In trauma patients admitted with a pulse $\geq$130/min, we found that the actual DTA diameter was on average about 13% larger than measured on the CT at admission. If similar aortic changes would occur in admitted TTAI patients with comparable vital signs, a physician could consider increasing the percentage of oversizing of the endograft when performing TEVAR on a hemodynamically unstable TTAI patient. However, excessive oversizing may increase risks of enfolding or collapse of the endograft,[19-21] a serious and potentially lethal complication, and further studies are therefore needed to confirm our findings, before such a recommendation can be made.

If the observed decrease in aortic diameter is related to a shortage of circulating volume, then an alternative to excessive oversizing of the endograft may be delaying the endovascular procedure until adequate resuscitation has been achieved. Recently, there has been a trend toward delayed repair of TTAI in more stable patients, which appears to improve survival.[2,22-26] Institution of antihypertensive medication can reduce the risk of aortic rupture in

these patients, resulting in a further decrease of in-hospital mortality.[2,26-28] Delayed repair may allow complete resuscitation, and additional CTA imaging can provide more reliable data regarding the actual aortic measurements for endograft sizing in these patients. However, this strategy is only possible in selected TTAI patients, and delaying TEVAR could be fatal in some hemodynamically unstable patients. Moreover, this approach would result in additional radiation exposure and treatment costs.

In this evaluation, the increase in aortic diameter on the control CT was not consistent in all patients, and in some cases, no increase in aortic diameter or even a slightly smaller aortic diameter was seen on the control CT examination. There are several theoretic explanations for the inconsistent findings. The few patients in which no decreased aortic diameters were seen on the initial CT at the trauma bay, may have been hemodynamically unstable due to other causes than blood loss, or may have been more stable during CT examination due to fluid resuscitation. The exact vital signs and adequacy of resuscitation of the patient at the moment of the CT examination are difficult to determine retrospectively, which is an important weakness of the present study. There may have been inaccurate aortic measurements in some patients, or the aorta may have been measured at a slightly different level on the second CT scan by the single observer. Additionally, the aorta exhibits significant pulsation with each heart cycle,[11,29,30] and the aortic diameter is typically larger in systole than in diastole, which may have contributed to the relatively wide range of the increase in aortic diameter among individual patients.

A considerable percentage of patients with ruptured abdominal aortic aneurysms[31-33] and ruptured thoracic aortic aneurysms[34,35] are admitted with hemodynamic instability and/or hypovolemic shock, which could result in aortic diameter changes in these patients as well. However, potential aortic changes may be less spectacular in hemodynamically unstable patients with ruptured aortic aneurysms than in TTAI patients, since patients with aortic aneurysms typically are much older and the aorta may be more stiff and calcified.

We observed even more remarkable increases of the diameters of the IVC, used as a marker for adequacy of resuscitation. In particular, the minimum diameter decreased dramatically on the CT examination while hemodynamically unstable, which suggests that the IVC changed shape as well during hemodynamic instability, from an approximately round shape to a more elliptic shape. Decreasing diameters of the IVC during hemodynamic instability and/or hypovolemic shock has been described before,[36-39] and some have opted that the IVC diameter is more sensitive than the blood pressure alone for identifying a hypovolemic state in trauma patients.[36]

**Limitations.** The findings of the present study should be viewed within its limitations. The median time interval between CT and the recorded vital signs was 30 minutes, and due to the retrospective design of the study, the exact vital signs and adequacy of resuscitation of the patient at the

moment of the CT examination are difficult to determine. Consequently, the increase in aortic diameter may be even underestimated. Additionally, the cardiovascular circulation is a complex balance. Many factors may affect the aortic diameter, including anti-hypertensive medication, hormones, pre-existing comorbidities such as atherosclerosis, diabetes mellitus, and others that may have confounded our results. Therefore, we could only conclude that aortic changes occur in hemodynamically unstable trauma patients, but we could not derive a model from our data to predict actual aortic diameters in admitted trauma patients. Additional research, preferably a prospective multicenter study is needed. ECG-gated CT would be most suitable for such a study, since this imaging modality considers the normal aortic dynamics. However, since most trauma patients will not undergo two ECG-gated CT examinations of the thorax and abdomen, it may be very difficult to realize such a study, and an experimental animal study may be more practical.

## CONCLUSION

We observed that trauma patients that are admitted with hemodynamic instability have a significantly smaller aortic diameter on the CT at admission compared with a control CT examination. The decrease in aortic diameter was observed in the entire aorta, from the ascending thoracic aorta to the infrarenal abdominal aorta. This decrease in aortic diameter could theoretically lead to inaccurate aortic diameter measurements and undersizing of the endograft in hemodynamically unstable TTAI patients requiring TEVAR. Further research is needed to better predict the actual aortic diameters in individual hemodynamically unstable patients requiring endovascular aortic repair.

## AUTHOR CONTRIBUTIONS

Conception and design: FJ, HV, FM, BM
Analysis and interpretation: FJ, HV, FM, BM
Data collection: FJ, HM, KD
Writing the article: FJ, KD
Critical revision of the article: HV, HM, KD, FM, BM
Final approval of the article: FJ, HV, HM, KD, FM, BM
Statistical analysis: FJ
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Overall responsibility: BM

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