

# Post-traumatic lesions of the aortic isthmus



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## Post-traumatic lesions of the aortic isthmus

*Lesions of the isthmus are the most frequent among post-traumatic lesions of the thoracic aorta (LTA): almost always secondary to closed thoracic traumas (road accidents, falls, crushing, and explosions), they are rarely iatrogenic (operator catheterisms) or caused by penetrating wounds. In the review of the literature concerned in the report, from the analysis of 89 bibliographic sources, we note that the etiopathogenesis and the pathophysiology of the LTA still entail a very high immediate mortality, but we also note that, in recent years, remarkable improvements have been made not only in prevention, first-aid, diagnostic definition and in the understanding of the development of the LTA, but above all in therapeutic results. The correct use of the conservative approach, particularly in the immediately post-traumatic phases, the increasingly wide-spread use of endovascular exclusion (T-EVAR), even if not without numerous technical difficulties, and the further improvement of open surgery, currently make it possible to guarantee the individual patient the treatment that can offer the best probabilities of success, at least immediately. Final development, and a more complete and rigorous assessment of the medium and long term results of TEVAR will allow the formulation of therapeutic strategies that are even better defined and increasingly simple to implement, on the basis of algorithms, such as the one proposed by the Authors.*

KEY WORDS: Aortic rupture, Evar, Isthmus, (T-EVAR), Trauma.

Lesions of the isthmus are most often the result of traumas of the thoracic aorta: almost always secondary to closed thoracic traumas (road accidents, falls, crushing, and explosions), they are rarely caused by penetrating wounds (knife and fire arm wounds) or are iatrogenic (operator catheterisms). Suffering from a high, often immediate mortality rate, they are difficult to treat even in the minority of patients who remain alive until they are hospitalised. In these cases, endovascular aortic exclusion (T-EVAR) is today a further therapeutic option compared with the, by now, classic alternative between at least initially conservative treatment, based mainly on controlled hypotension, and thoracotomic aortic replacement which, in emergency or urgency, in any case has a high mortality rate, also due to the associated lesions that are very frequent in these almost always multi-trau-

matized patients, and by specific morbidity – particularly serious in terms of the risk of cerebral haemorrhage and paraplegia.

## Incidence

In 1557 Andrea Vesalio described the first case of death from post traumatic thoracic aorta lesion (LTA) in a man who had fallen from his horse <sup>1</sup>. If we exclude direct lesions, moreover almost always fatal, the LTA is caused by a sudden deceleration of the thorax due to impact with a fixed obstacle: for this reason the incidence of immediately lethal cases, once modest, grew initially in a way that is directly proportional to the increase in the spread of high speed vehicles, to then decrease in an equally significant manner with the spread of modern car safety systems, introduced and subjected to on-going development on the basis of the definition of physiopathological LTA mechanisms, obtained from autoptic studies, data obtained from experimental animal models and finally from increasingly sophisticated computerised simulations. <sup>2-6</sup>.

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In the USA, the incidence of fatal LTAs grew rapidly from the 0.73% found in 1947, in 7000 autopsies on road accident victims, to an incidence of between 10 and 17% in various study groups at the end of the Sixties, to reach a peak of 26% in the review of the causes of road accident death carried out in 1988, when few vehicles were fitted with safety belts and even fewer drivers used them and, above all, airbag technology had not yet been introduced. The introduction of such protections, their progressive improvement (side and de-potentialised airbags, triple-anchored belts, side reinforcements) and the obligation to have them installed and to use them, have resulted in a significant decrease in mortality from LTA, up to 5.8% registered in 403 autopsies carried out during the period 1997-2002<sup>7-11</sup>. Moreover, in 2003<sup>12</sup> Richens and coll. estimated that around 20% of road accident deaths was associated to LTA. It shouldn't be forgotten that, at least in Italy, LTAs are often secondary to the use of motor-bikes, obviously not equipped with such safety systems, and finally that the second cause of LTA are traumas caused by falling (often from workplace accidents). In all, Fattori and coll. in 2007<sup>13</sup> assumed, on the contrary, that the incidence of patients with LTA who reached the hospital alive can be destined to increase, thanks also to the improvement in first-aid services.

### Anatomy, Pathophysiology and Types of LTAs

A short reference to the anatomy of the thoracic aorta is essential in order to be able to understand the localisation of LTAs, which only in 25-30% of cases occur at the level of the ascendant aorta and the arch (in these cases, patients who do not die immediately, or immediately after arriving at the hospital, are very rare), while the remaining 65-70% are observed at the level of the isthmus or at the origin of the descending aorta (and nearly all patients who at least initially survive the trauma are among these).

The isthmus, between the lower limit of the ostium of the left proximal subclavian and the origin of the third pairs of distal intercostals (the first two pairs of intercostals are branches of the thyrobricervicoscapular trunk of the subclavian), is the most mobile portion of the thoracic aorta since it is only in relation with the inferomedial pulmonary artery through the arterial ligament (residue of Botallo's foetal arterial duct). Instead the ascending aorta and the arch, on the whole travelling from the bottom towards the top and then transversally, from right to left and from the anterior to the posterior mediastinal and finally again downwards, are relatively fixed structures, thanks to the pericardio-heart unit on the bottom and the epicortic vessels at the top, just as the descending aorta is securely anchored to the costal and vertebral levels in giving origin not only to the bronchial, mediastinic and aoesophageal arteries but also to the last eight pairs of intercostal arteries.

We assume that closed trauma of the thorax causes the sudden increase of aortic endoluminal pressure, both due to the sudden squeezing of the endocardiac blood and due to acute obstruction of the aorta at the diaphragmatic hiatus (caused by angulation of the vessel caused at this level by a sudden deceleration) up to values of from 350 to 600 mmHg within 50-100 msec. of impact<sup>2</sup>. These pressing values, in themselves insufficient to cause rupture of the vessel, are, however, enough to cause its acute relaxation: the ascending aorta and the proximal arch, and the descending distal aorta, would change into a rigid and relatively fixed pipe which, acting as a fulcrum on the epiaortic vessels and particularly on the left subclavian securely anchored to the first rib, would create pressure waves which, with both an ascending and descending path, and associated to the partial dislocation of the heart and arch upwards, would in turn cause stretching, rotation and acute translation of the isthmus around its only rigid point, composed of the arterial ligament<sup>2-6</sup>. The latter is therefore the most frequent point of initial laceration, with a circumferential path, or transversal and with antero-lateral propagation, while the posterior wall may remain intact, or tamponed – at least temporarily – by the mediastinic tissue which surrounds the aorta itself starting from the third pair of intercostal arteries.

The conditions and extent of the trauma determine the various types of LTA, coded by Parmley and coll. 1958<sup>14</sup>, on the basis of the autoptic examination of 296 early LTAs:

- 1 Intimal haemorrhage;
- 2 Intimal laceration with haemorrhage;
- 3 Laceration of the median;
- 4 Complete laceration of the aorta (complete transection of the aorta);
- 5 Pseudoaneurysm;
- 6 Complete laceration with periaortic haemorrhage.

As well as the intimal always involved (probably due to its greater rigidity compared with that of the median and the adventitia) with immediate subintimal haemorrhage, the initial lesion nearly always also extends to the middle tunica, and it is calculated that in 80-90% of cases, after a free interval lasting from a few seconds to several years, parietal laceration of the whole thickness occurs, with a very high risk of immediate death caused by a massive haemorrhage.

Survival is usually established only by the intact condition of the adventitia and by the padding provided by the periaortic tissue<sup>15</sup>. Immediately after the trauma, a thrombus composed of fibrin and an emulsion of erythrocytes is created, followed by fibroblastic proliferation and with this the neovascularisation of the wall: in 2-3 weeks the thrombus organises itself and finally neointimalization of the lesion begins<sup>13</sup>. In addition to the rare *restitutio ad integrum* on one hand, only possible in the slightest cases, and to complete rupture of the aor-

ta on the other, a possible short term development is the pseudocoarctation syndrome, arising in around 10% of cases by the invagination, in the lumen, of a medio-intimal flap which, reducing the haematic flow in the descending aorta, can cause distal and spinal ischemias serious enough to require emergency surgery. The other typical developments are sacciform pseudoaneurysm in the case of transversal lesion, and fusiform pseudoaneurysm in the case of circumferential lesion: in late diagnosis, in these cases parietal calcification involving the periaortic tissue, is frequent with possible formation of strong adhesions with the pulmonary parenchyma and the pleura <sup>16</sup>.

### Prognosis

More than 85% of trauma patients with LTA die before they reach hospital. Without adequate treatment, patients who reach the hospital have a mortality rate of 30%, in the first 6 hours and of 50% in the first 24 hours after hospitalisation, but this worst prognosis is often in direct relation to the associated serious lesions almost always present, with a high incidence of cranial-encephalic traumas and abdominal parenchymas. If we exclude level 1 LTAs that often allow possible restitutio ad integrum, as we have already said, we believe that the evolution, with variable and unpredictable interval between trauma and final event, is almost always towards pseudocoarctation or, more frequently, towards rupture, in general through the formation of pseudoaneurysms and almost always immediately lethal <sup>2-12</sup>.

### Diagnosis

The very high incidence of LTAs caused by road accidents or by falls or crushing calls for the study of the thoracic aorta, and in particular the isthmus to be considered as obligatory and routine in multi-traumatised patients. The current level of definition of multi-slice CT Angiography methods and the associated "volume rendering" techniques in 3-D (since 2002-2003, thanks to the implementation of the number of detectors compared with the old instruments) in these cases used in any case compulsorily at "total body" extension, allows the acquisition of images in just a few seconds and extremely accurate identification of various types of the above-mentioned LTAs. Therefore it doesn't appear to be appropriate or useful to discuss any further the radiologic semeiotics of tests which were once the only ones available (e.g.: more or less accentuated changes in the mediastinic profile of the standard x-ray of the thorax <sup>17</sup>, but also the data provided by CTs of the first generations, and finally the same angiography findings, considered "gold standard" until about the end of the Nineties). Given the availability of adequate equipment,

we can state that, for this pathology also, the current diagnostic capacity is fundamentally operator-dependent, requiring a specific experience in the interpretation of vascular imaging which for these lesions, is, itself, absolutely accurate but, above all, in the forms at least initially less serious, at times not simple to recognise <sup>18-25</sup>. It should also be pointed out that most patients who reach hospital alive do not have active bleeding in the location of the aortic lesion, and that possible haemodynamic instability is more often caused not by the LTA, but by parenchymal laceration (mainly the liver and the spleen) or by frequent and widespread haemorrhage centres at the bone fracture; in any case, prompt CT-Angiography recognition of the direct or indirect signs of LTA (Table I), of the so-called signs of impending rupture (increase in dimensions or morphological worsening of the parietal lesion, increase in the mediastinic haematoma, haemothorax relapse) and of the already described pseudocoarctation syndrome <sup>13</sup> is fundamental. In addition to the multi-slice CT Angiography, and not for the first diagnosis but only as a monitoring method in cases that are addressed at least initially to a conservative treatment, the trans-oesophageal echo-cardiograph (TEE) may be taken into consideration, but with extreme caution: the fact that it allows the non-significant reduction of radiation to which the patient is exposed due to repeated CT-Angiography during any observation period

TABLE 1 – Radiological Imaging of LTAs (modified from Mirvis, et al. <sup>21</sup>)

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<i>PRIMARY FINDINGS</i>
– Pseudoaneurysm
– Mediastinal hemorrhage (active or stable)
– Hemotorax (active or stable; left / right / bilateral)
– Intimal Flaps and/or Pseudocoarctation
– Intramural Thrombosis
– Endoluminal Thrombosis (also if partial)
– Abnormalities of the parietal aortic contour
– Localized Variants of the aortic size
<i>SECONDARY FINDINGS</i>
– Mass effect with displacement of the oesophagus or nasogastric tube or trachea to the right
<i>ATYPICAL LOCATION</i>
– Aortic arch
– Ascending aorta
– Paradiaphragmatic aorta
– Congenital anatomic variants (lusoria subclavian artery, aortic coarctation, etc.)
<i>IMPENDING RUPTURE FINDINGS</i>
– Size and/or morphology variants of LTAs or hematoma
– FALSE DIAGNOSIS BECAUSE OTHER HEMORRHAGES
– Hemorrhage from periaortic mediastinal vessels
– Hemorrhage from branch vessels
<i>FALSE DIAGNOSIS BECAUSE CONGENITAL VARIANTS</i>
– Patent ductus arteriosus
– Aortic diverticulum
– Isthmic malformation
– Branch vessels infundibula

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must be considered as positive, but the potential risk of any further direct damage to the already damaged aortic wall – as far as further evident rupture, particularly in the case of plugged pseudoaneurysm – caused by inserting the instrument into the aesophageal lumen adjacent to the aortic isthmus, must not be ignored.

In Centres equipped with the latest generation of equipment, and above all operators with a high level of specific experience, dynamic Angio-MR is certainly an excellent alternative to the more invasive multi-slice CT-Angiography and TEE, since it can provide a morphological and haemodynamic definition of the LTA that theoretically can make it the gold standard of the years 2000<sup>26</sup>. Moreover, even in logistically favourable situations, the costs and, above all, the length of the test make its use more suitable for subsequent checks than for the first diagnosis<sup>27</sup>.

## Therapeutic strategies

There are three therapeutic options for LTAs: conservative medical treatments; open surgery correction; correction via T-EVAR.

### 1) CONSERVATIVE TREATMENT

In any case to be started as soon as possible where there is the suspicion of a possible LTA even only on the basis of trauma procedures and pending diagnostic investigations, this is based on hypotensive intensive therapy through beta blockers and nitrates, under constant monitoring (PAS ideally maintained around 100 mmHg)<sup>28-37</sup>. The premise for this conservative treatment consists in the fact that at least the adventitia and the periaortic mediastinic tissue of most patients who reach the hospital alive must be undamaged, making a massive haemorrhage unlikely. It is however fundamental to carry out seriated instrumental checks – at least every two days initially and then at increasing intervals later – to check for the absence of those radiological imaging findings, mentioned above – that predict impending rupture<sup>13,28</sup>.

Even when a surgical or endovascular decision has been reached, the time interval obtained in this way before carrying out the operation may make it possible to stabilise the patient and to take other therapeutic measures often necessary in these cases that are almost always complicated by often serious lesions, even in other areas (mainly cranio-encephalic, hepatic and splenic), that make emergency surgery, and in particular immediate open surgery, very high-risk.

Moreover, consolidation of the LTA in itself, and the already described process of re-absorption of the intraperitoneal haematoma almost always above and below the lesion and the periaortic haematoma, secondary not to the LTA but to the simultaneous laceration of small mediastinic vessels, make it possible to tackle the surgical correction

with greater chances of success, when the aortic wall has reacquired greater solidity, generally within 15-20 days, and if the even modest periaortic haemorrhages under-way have stopped.

Finally, LTAs, which at initial radiological imaging can be attributed to those of Level 1-2 sec. Parmley, if of an extremely limited extension, can evolve spontaneously until they even reach *restitutio ad integrum*, which does not exclude the need for long term instrumental follow up to check for the absence of late degenerations which may be possible, such as pseudocoarctation and pseudoaneurysm.

Various experiences show that the risk of complete rupture is limited and therefore acceptable if the patient is haemodynamically stable, and especially in cases where the lesion is not circumferential<sup>34-37</sup>. The length of conservative treatment is extremely variable, to be modulated according to the patient's conditions, morphological characteristics and any changes in the imaging checks of the individual case, also taking into account the technical considerations discussed before. After the first indication of Akins and coll.<sup>32</sup> who, at the beginning of the Seventies, before proceeding with surgical correction, began a hypotensive treatment, without finding any case of acute evolution of LTAs, the experience of Pate and coll.<sup>30</sup> who in 1995 referred that in none of the 41 pseudoaneurysms kept at pressure values lower than 140 mmHg did they find rupture before the surgical operation, carried out between 12 hours and 24 weeks of the trauma, appears to be very important. Since then, most surgeons have adopted this approach, with very significant experiences also in Italy, such as the one published in 2000 by Pierangeli and coll.<sup>35</sup>, discussed in detail later.

This decision must, in any case, be well assessed in relation to various risks<sup>38-41</sup>:

- Sudden death, from acute and uncontrollable evolution of the LTA during medical treatment: even in apparently stable situations, various Authors have recorded the death of 2-5% of patients, within the first week of the trauma, even if such data must be assessed critically in relation to the implementation methods of the necessary radiological monitoring mentioned above;
- Gradual dilation of the aortic sector involved by the LTA, until compressive phenomena on the trachea and the main left bronchus;
- Fibro-calcific organisation of the aortic wall that can make any future endovascular treatment difficult;
- Cerebral hypoperfusion induced by the hypotensive treatment itself, in patients who frequently present cranial traumas associated to the LTA.

### 2) OPEN SURGERY

Generally carried out via left thoracotomy and unilateral right ventilation, CEC and relative heparinization (only partially reducible although using current heparinated circuits), also in Centres with greater cardiosurgi-

cal and vascular experience – not always easily reachable from the place of the traumatic event – open emergency surgery for LTA has a mortality rate of from 15 to 30%, and central neurological morbidity (cerebral haemorrhage, especially in frequent cases of patients with associated cranial trauma) and peripheral neurological morbidity (paraplegia) that have a 15% influence on cases<sup>42,43</sup>. Therefore, currently this procedure must be considered in emergency cases, when an endovascular procedure is logistically or anatomically impossible, and above all only after having ascertained that conservative treatment is impossible.

In fact, for the reasons already mentioned, this last option makes it possible to carry out replacement, in open surgery, of the aortic section where the LTA is located with a lower rate of complications, below 5% including mortality plus neurological morbidities<sup>26-37,39,40</sup>. Therefore, unlike in emergency cases, thoracotomy surgery of the LTA carried out electively, or even in deferred urgency, is still to be considered an initial possible choice for final treatment of the LTA, and is still probably the gold standard for the correction of its late complications.

Therefore the synthesis of Mattox and Wall who, in 2000<sup>16</sup>, identified three clinical situations with regard to open surgery of LTAs, is still valid:

- 1: Patients with massive and exsanguinating LTAs, for whom every surgical attempt is, in any case, pointless;
- 2: Haemodynamically unstable and only temporarily compensable patients: there may be time to carry out diagnostic tests and proceed with the surgical attempt, but mortality, in any case, remains very high;
- 3: Stable haemodynamic patients with LTA and tamponed haematoma, often with diagnosis deriving from diagnostic investigations prescribed only on the recent anamnestic trauma data, capable of causing an aortic lesion caused by deceleration. These patients usually do not require emergency treatment, and it rarely results in LTA-correlated death.

After the absolute emergency phase, both the length of deferment of the surgical correction and the surgical technique to be adopted are still the object of discussion. With regard to the first aspect, Schumacher and coll.<sup>43</sup> opt for deferred urgency, maintaining that the best timing after initial stabilisation of the patient is within 72 hours of the trauma, before fibrosis of the mediastinic haematoma has developed. Most Authors, on the other hand, favour much longer intervals between trauma and open surgery, and in any case, after having treated the various comorbidities that are often the primary cause of the very bad results of immediate open surgery (with particular reference to the treatment of serious respiratory deficits caused by pulmonary contusion, and stabilisation of cranial traumas): for example, Pierangeli e coll.<sup>35</sup>, after observing a mortality of 19% in 21 patients who underwent immediate surgery, paraplegia in 3 cas-

es and permanent dialysis in 1 of the survivors, in another 29 patients, they carried out surgical correction at as long as 8.6 months (on average) from the LTA, after prolonged hypotensive treatment and treatment of the pulmonary complications associated to the trauma, bringing mortality and major complications to 0%.

With regard to the more purely technical aspects of the open surgery of isthmus LTAs<sup>42,43</sup>, it should be remembered that the methods used are mainly either simple clamp and sew, or correction with aortic distal perfusion, via Gott shunt or left atrio-femoral bypass or again with total femoro-femoral CEC. In favour of distal protection, moreover without significant advantages in Literature of one particular type of perfusion with respect to the others<sup>44</sup>, is discovery of a significant reduction in paraplegia compared with the simple clamp and sew (moreover, not always feasible), especially thanks to the possibility of lengthening clamping times in often complex situations, and reducing to the minimum the spinal insult from hypotension that can occur if intraoperative bleeding becomes significant. Moreover, spinal protection also becomes crucial in relation to the frequent advisability of clamping the aorta in safe conditions between the common carotid artery and the left subclavian (thus further reducing spinal circulation starting from the vertebral artery); this manoeuvre, obligatory in the case of LTA at less than 1 cm from the subclavian, is perhaps advisable in any case in order to be able to proceed with extreme caution in the circumferential isolation of the aorta at the arch, in this way reducing the risks of iatrogenic lesions of the oesophagus, thoracic duct and recurrent nerve and, above all, without dissecting the posterior wall of the vessel, or penetrating the context of the perilesional haematoma or the LTA itself, with massive and often fatal intraoperative haemorrhage<sup>42-44</sup>.

### 3) ENDOVASCULAR THERAPY (T-EVAR)

In theory, endovascular exclusion of the aorta with LTA solves most if not all the problems mentioned up to here: the T-EVAR is positioned with mini-invasive accesses and specifically does not expose the patient to the risks of proximal aortic clamping, does not require heparinization, rarely entails significant risks of paraplegia and, finally, can be completed within just a few dozen minutes. For all of these reasons, it also makes it possible to avoid resorting to the stabilisation period and the relevant risks of very evident delayed rupture, allowing, rather, the treatment of associated lesions in one surgical operation (the case of LTA associated to abdominal haemorrhage caused by parenchymatous lesion is typical).

For the first time used as treatment of LTAs by Semba and coll. in 1997<sup>45</sup>, after more than a decade of constant perfecting of materials and the various types of endoprostheses, it has become perhaps the most commonly used technical option in these patients, and

Literature at this point contains numerous experiences with dozens of treated cases: all agree in underlining a technical success of the stated procedure in around 95% of treated cases, with very low LTA-correlated mortality and paraplegia, almost always in total below 3% (Table 2<sup>13,46-71</sup>).

Moreover, in the first systematic review and meta-analysis of works published on the comparison between T-EVAR and open surgery, which appeared in the Journal of Vascular Surgery in May 2008 and coordinated by Ouriel K.<sup>72</sup>, we read that in the case of LTA – unlike other pathologies of the thoracic aorta even though also treated with T-EVAR – no statistically significant differences emerge, neither of mortality nor of paraplegia (although recording a certain reduction of the latter in patients undergoing T-EVAR). Although not underestimating the well-known bias inherent in this type of statistical analysis, these findings are in any case proof of the need to reduce probably excessive enthusiasm, which probably may lead to tackling and treating through T-EVAR the patient with LTA even without the support of specialists in cardio-surgery or vascular surgery, and perhaps also to widening recommendations for the operations, as we can imagine by observing a significant number of LTAs recently treated in Centres which, previously, had not indicated interest in this type of pathology.

On the other hand, numerous other Authors have already emphasized that the T-EVAR, in this as in other applications, presents numerous problems that still have not been solved<sup>13,43,51,54,60,70</sup>. To date, we are still a long way from producing an ideal endoprosthesis for the thoracic aorta, as proved by the considerable structural differences between the various types on sale today, and by the constant changes made by all the manufacturers. The structural heterogeneity that characterizes the different and numerous endoprostheses on the market (material, thickness, dimensions and disposition of the exo and endoskeleton through stent; conformation of the ends with or without the presence of uncovered stents or of extraflexions flush with the tissue; any transparietal anchorage systems, etc.) and the resulting difficulties in assessing their different performance in the different anatomical situations, has also been demonstrated recently by Canaud L. and coll., with their study published in June 2008 in the Journal of Endovascular Therapy<sup>73</sup>. In this experimental study, endoprostheses belonging to the four different types, currently the most used, were inserted into removed from cadavers normal-sized aortas, without any lesions (moreover above all similar to the aorta usually suffering from LTA) testing several oversizing sizes for each brand. Subjecting the aorta to increasing angulations, the aorta-endoprosthesis system

TABLE II: *Recent Results of > 10 T-EVAR for LTAs from Uni-Center surveys.*

	No. cases	% Early technical succes	% Mortality within 30 days	% Paraplegia and/or stroke
Alric et all., 2002 <sup>46</sup>	10	100	10	0
Lachat et all., 2002 <sup>47</sup>	12	100	8	0
Kato et all., 2003 <sup>48</sup>	13	100	46	0
Karmy-Jones e coll., 2003 <sup>49</sup>	11	81,1	27,3	0
Scheinert e coll., 2003 <sup>50</sup>	10	100	0	0
Amabile e coll., 2004 <sup>51</sup>	13	100	0	0
Dunham e coll., 2004 <sup>52</sup>	16	100	6,3	0
Melnitchouk e coll., 2004 <sup>53</sup>	15	100	0	6,6
Morishita e coll., 2004 <sup>54</sup>	18	100	17	0
Neuhauser e coll., 2004 <sup>55</sup>	13	92	0	0
Doss e coll., 2005 <sup>56</sup>	18	100	0	0
Eggebrecth e coll., 2005 <sup>57</sup>	17	65	24,5	0
Michelet e coll., 2005 <sup>58</sup>	10	100	0	0
Peterson e coll., 2005 <sup>59</sup>	11	100	0	0
Rousseau e coll., 2005 <sup>60</sup>	29	96,5	0	0
Agostinelli e coll., 2006 <sup>61</sup>	15	100	13	0
Hoorweg e coll., 2006 <sup>62</sup>	28	100	0	0
Marcheix e coll., 2006 <sup>63</sup>	33	100	0	3
Pratesi e coll., 2006 <sup>64</sup>	11	100	9,1	0
Reed e coll., 2006 <sup>65</sup>	13	100	23	0
Fattori e coll., 2007 <sup>13</sup>	27	100	0	3,8
Neschis e coll., 2007 <sup>66</sup>	20	100	0	0
Orend e coll., 2007 <sup>67</sup>	34	100	8,8	0
Raupach e coll., 2007 <sup>68</sup>	10	100	10	10
Steingruber e coll., 2007 <sup>69</sup>	22	86,3	4,5	0
Buz e coll., 2008 <sup>70</sup>	39	97,4	3,7	0
Canaud e coll., 2008 <sup>71</sup>	27	100	3,7	0

was then assessed with the emission of pulsatile flow: the observed performances, relating both to the seal of the graft to the aortic wall which on maintaining perviety showed obvious differences among the various endoprostheses, also with photographic documentation, to say the least, impressive, especially if imagined in a living person, and curiously never in favour of the endoprosthesis used the most by the first Author <sup>71</sup>.

### Problems of the T-EVAR specific to LTAs

An initial series of technical problems derives from the particular type of patients with LTA who, being generally victims of road accidents or accidents on the workplace, are mostly young, with, moreover, an undamaged vascular system. This entails certain specific physiopathological characteristics that are typical of these patients compared with patients with degenerative aortic pathologies. In particular <sup>67</sup>: a considerable pulsatility and accentuated movements of the aorta in both a longitudinal and transversal direction, with variation in size between systoles and diastoles of at least 15-20%, and in any case generally over that of patients with degenerative pathology; a normal vasal diameter frequently not over 20-22 mm, sometimes between 16 and 20 mm, very different from the widely ectatic vessels associated to aneurysms; an isthmus often located at the tip of an extremely acute angle between the arch and the descending thoracic aorta, this being a less frequent condition in patients affected by atherosclerosis which generally also results in lengthening of the vessel and a less accentuated angulation of the arch.

In the case of LTA, therefore, and above all in the young patient, any choice in favour of the T-EVAR must therefore be made bearing well in mind the following summarised considerations:

– The greater range between systoles and diastoles makes even more difficult pre-operative radiologic planning that is indispensable in order to be able to choose the endoprosthesis most suitable to the individual case: imaging analysis is always difficult to carry out at aortic arch level, due to the complex changes in path already mentioned above, and therefore equipment of the latest generation is required and, above all, operators with specific experience. One of the major difficulties consists in the choice of the oversizing that is in any case necessary to obtain an adequate anchorage to the aortic wall, both for endoprostheses with uncovered proximal stents, designed to stabilise the system and to make its radial structure more effective and for those also, or only, equipped with transparietal fixture systems. There is a real risk <sup>74-83</sup> of choosing either an excessive oversizing (resulting incomplete aperture or at least partial infolding of the endoprosthesis with type I endoleak and also collapse of the endoprosthesis or, again, excessive tension on the aorta, with secondary

dissection or rupture of the vessel, often also involving the oesophagus and the pulmonary parenchyma) or an insufficient oversizing (possible migration of the endoprosthesis and type I repermeation of the LTA, or even its accentuation from laceration secondary to the migration itself);

– The smallest endoprosthesis for the thoracic aorta currently available measures 22 mm, supplied, however, by only three brands, but the 24 mm size can already cause an excessive oversizing for patients with LTA, with the relative risks of complications mentioned above (with an endoprosthesis of 24 mm in an aorta 20 mm in diameter, there would be, for example, a 20% oversizing, about double that currently recommended by the manufacturers for the T-EVAR of LTAs in the healthy aorta);

– In order to continue to adhere to the aortic wall inside a path with a particularly acute angle <sup>67,78-83</sup>, the endoprosthesis must, above all, have considerable conformability, indispensable both in order to progress within such angulation at the time it is positioned (even if facilitated in these cases by the use of extra-stiff guides as well as the by now specific classical manoeuvres, such as the combined brachial or anonymous and femoral approach, etc), and in order to adhere to the aortic wall following its winding path, and without causing an excessive straightening thrust on the vessel. In these cases, in fact, the risk of a collapse of the aortic wall with formation, starting from both ends of the endoprosthesis, of an ante or retrograde dissection, or of a pseudoaneurysm caused by laceration of the aortic wall, often involving the adjacent oesophagus and pulmonary parenchyma <sup>74-77</sup> becomes high, immediate but also at short-medium term. At the same time, the expansive radial force of the endoprosthesis, itself a conflicting characteristic with respect to conformability, must be sufficient so that the prosthesis itself does not stenosed (thus creating an iatrogenic pseudocoarctation), and, thanks also to any transparietal anchorage systems, does not tend to migrate, pushed both by endoaortic pressure and by the same parietal motility and by the already mentioned changes in diameter of the native vessel during the systole-diastole cycle <sup>67,78-83</sup>. Above all in cases of an arch with an unsatisfactory angle, a possible solution is to widen the sector of the healthy aorta covered by the endoprosthesis (landing zone) to the proximal level, to increase the sealing extension and therefore the efficacy, and also at distal level, bringing the caudal limit of the endoprosthesis to the rectilinear sector of the descending thoracic aorta, so as to also further exploit columnary strength <sup>84</sup>. However, the result of these solutions may be that there will be proximal interference with the ostium of the left subclavian, with problems that will be discussed later, and that, distally to the LTA, create greater oversizing, with the relevant possible complications, caused by the physiology and gradual decrease in caudal sense

of the diameter of the descending thoracic aorta <sup>71</sup>. Again distally, in this way there is coverage of a greater number of intercostal arteries, with at least a theoretical increase in the risk of paraplegia, and of the oesophageal and bronchial arteries, which, according to some Authors, could, through ischemic damage, create the premises for aorto-oesophageal and aorto-bronchial lacerations and fistulizations that can also occur even a long time after the implant <sup>74-77</sup>;

– The calibre of the supporting systems currently on the market for endoprostheses for the thoracic aorta (variable in relation to the sizes of the endoprosthesis used, but also for smaller endoprostheses with a minimum size of 7-8 mm) may not be compatible with the diameter of the common femoral artery and the external iliac: but this aspect must be borne in mind, when there is a risk of causing deep dissection and also laceration of such areas during release of the system. The solution of the problem is to make an access to the common iliac extraperitoneally which, even if more invasive compared with the crural access, is, on the other hand, generally quite easy (if made initially and not only after such complications arise), so that it is often selected by Authors <sup>62,63,66,69</sup>.

### T-EVAR and coverage of the left subclavian in TALSS

As for other cases discussed for open surgery, with the T-EVAR there is also the problem of the involvement or non-involvement of the left subclavian in the procedure <sup>84-86</sup>. The ostium of the vessel is often very close to the istmic LTA, which makes accidental or necessary coverage with the endoprosthesis frequent in order to obtain a sector of the aorta proximal to the lesion extensive enough to obtain a more secure sealing of the endoprosthesis. Most Authors maintain that this does not entail real risks for the patient, given the efficacy, in almost all cases, of the collaterality of the arterial circulation towards the arm, and having established the absence of cerebellar complications from diversion or interruption in the flow of the left vertebral artery.

On the basis of equally significant personal case histories, other Authors, on the other hand, underline that the risk of neurological complications after T-EVAR with coverage of the subclavian is not unimportant, both at cerebellar and spinal level, and not necessarily in the immediate post-procedural period <sup>13,85,86</sup>. It is, on the other hand, known from neurosurgical experiences <sup>87</sup> that occlusion of the left vertebral, dominating with respect to the right one in about 55% of individuals, can be compensated by the collateral circulation, but that, in about 5% of cases, can cause cerebellar infarcts with often serious functional deficits – up to the vegetative state of the patient as recently observed in a case by Fattori and coll. within the consolidated experience of the Bologna Centre <sup>13</sup>. Still with

regard to possible complications affecting the encephalic area, it was pointed out that, if the prosthesis is inserted near the subclavian, the fact that the endoprosthesis and the common left carotid are so close (and also its direct involvement in the case of endoprostheses with uncovered proximal stents) may result in an emboligenic source for supratentorial infarcts, found even a long time after the implant. Finally, we must not forget the importance of the vertebral circulation in the descending collaterality essential for maintaining spinal irrigation, which in these operations is always at risk due both to the obligatory coverage of a variable number of intercostal arteries by the endoprosthesis and in relation to the frequent episodes of systemic hypotension to which multi-traumatised patients may be exposed for various reasons.

Very briefly, with regard to coverage of the origin of the left subclavian, it is a matter of choosing among the various strategies, all reliably supported in Literature <sup>13,46-71,84-86,88</sup>:

- to limit their use as much as possible: some Authors currently maintain that the need to cover the ostium of the subclavian should arise less frequently than thought since for prosthetic sealing, at least in young patients with a healthy aortic wall, a landing zone proximal to the LTA only 4-5mm long would be enough, much shorter than that required for T-EVARs that must interact with atherosclerotic aortas;
- to consider as the first choice, open surgery for all patients in whom the subclavian coverage manoeuvre would be indispensable;
- in all cases in which the need for subclavian coverage is predicted, carry out preliminarily a carotid-subclavian revascularization, associated to the suture or section or endo-occlusion of the subclavian moncone proximal to the vertebral ostium to prevent type II endoleak;
- to believe that covering the subclavian ostium, particularly useful in severely angled arches, is without substantial risks, possibly carrying out a carotid-subclavian revascularization only later, in rare cases where an ischemic symptomatology in the brachial area or in the subtentorial area arises due to vertebro-subclavian diversion.

Finally, the fundamental grounds for which clear caution in using the T-EVAR in istmic LTAs still appears appropriate consists in the almost total absence, unlike what we can now state for thoracotomy replacement, of consolidated medium-long term results, a detail that should under no circumstances be neglected especially in relation to the average age of these patients. Too many cases appear lost at follow-up due to the lack of compliance both of the patient himself and the implementing Centre itself.

All the proposed endoprostheses have been modified several times over just a few years, if not even completely redesigned, as, on the other hand, is logical and

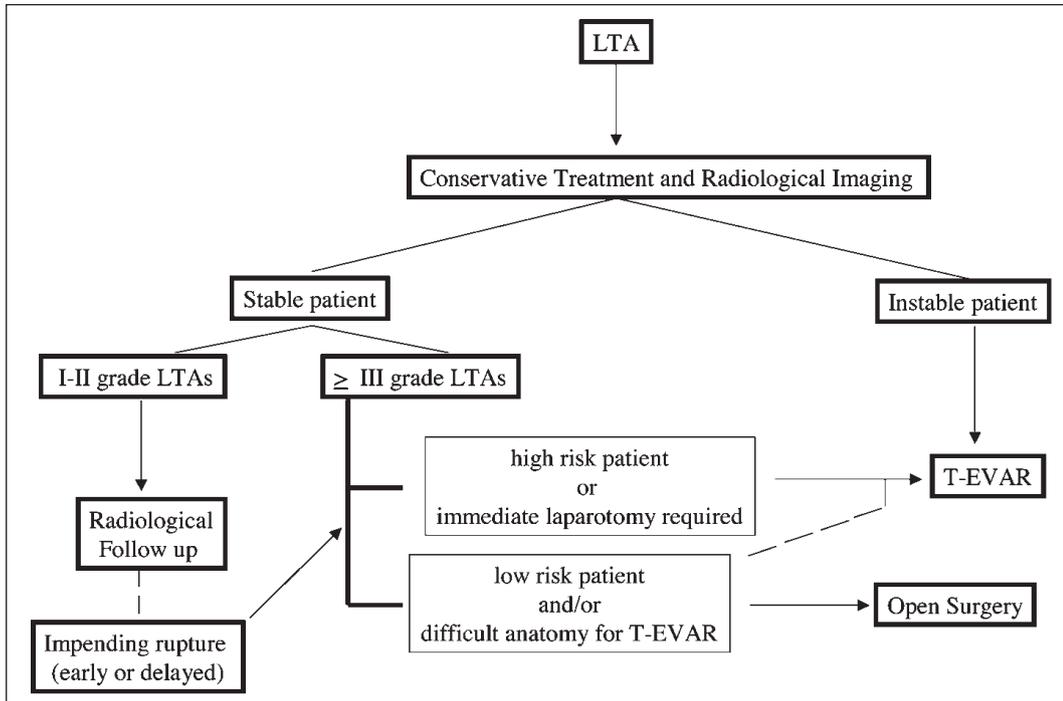


Fig. 1: LTA: A proposal of Therapeutic algorithm.

advisable for very recently conceived instruments for clinical use. The result, precisely, is this “non-datum”, obvious not only in retrospective studies, but also in the more bulky multi-centre registers and in the prospective studies themselves (the 25% of patients also lost in the FDA Acceptance Trial in the United States for one of the currently most used thoracic endoprosthesis<sup>89</sup> is a glaring example of it). Unlike what can be done today for the follow-up of abdominal EVAR (less use of CT-Angiography in favour of ultrasound methods, associated or not, to the use of echo contrast means), the problem of the costs and risks connected to imaging, which for now is still based on CT-Angiography, still remains for T-EVAR carrier patients, and will continue for a long time, because no-one is yet able to foresee the duration of the structural seal of endoprosthesis or the actual incidence of the various complications that may occur in the whole morpho-functional aorto-endoprosthesis complex: a typical example consists of the increasingly indicated aorto-aesophageal and aorto-bronchial fistulas, more as Case Reports at Conferences than in Literature<sup>74-77</sup>.

In any case, the datum according to which, above all in unstable patients and patients who are not candidates for open surgery, T-EVAR is currently a life-saving method, unimaginable only ten years or so ago, remains certain: we hope that it will soon be possible to further simplify the technique and to dispose of materials that have the same long-term reliability as that currently recognised for open surgery, whose limit, as already mentioned, is in the fact that it is difficult to put into practice in emergencies.

## Conclusions

Patients who are still alive when they reach hospital are still the minority of those suffering from LTA, but recently remarkable improvements have been made not only in prevention, first aid, diagnostic definition and in the understanding of the natural history of this event, but also in therapeutic results. The correct use of the conservative approach, above all in the immediately post-traumatic phases, the prudent use of the endovascular exclusion technique and the further development of open surgery, currently make it possible to guarantee the individual patient the treatment that can offer the best probabilities of success, at least immediately. Only the final development of T-EVAR and a more complete and rigorous assessment of medium and long term results will be able to allow the formulation, in coming years, of increasingly better defined therapeutic strategies, in algorithms that can modify the trends mainly followed today (Fig. 1).

## References

- 1) Scarpa A: *A Treatise on the Anatomy, Pathology, and Surgical Treatment of Aneurysm*. Edinburgh: Mundell Doig & Stevenson, 1808.
- 2) Siegel JH, Smith JA, Shabana Q, et al.: *Change in Velocity and Energy Dissipation on Impact in Motor Vehicle Crashes as a Function of the Direction of Crash: Key Factors in the Production of Thoracic Aortic Injuries, Their Pattern of Associated Injuries and Patient Survival. A Crash Injury Research Engineering Network (CIREN) Study*. J Trauma, 2004; 57:760-78.

- 3) Schmoker JD, Lee CH, Taylor RG, et al.: *A Novel Model of Blunt Thoracic Aortic Injury: A Mechanism Confirmed?* J Trauma, 2008; 64:923-31.
- 4) Nikolic S, Atanasijevic T, Mihailovic Z, et al.: *Mechanisms of Aortic Blunt Rupture in Fatally Injured Front-Seat Passengers in Frontal Car Collisions. An Autopsy Study.* Am J Forensic Med Pathol, 2006; 27:292-95.
- 5) Pearson R, Philips N, Hancock R, Hashim S, et al.: *Regional wall mechanics and blunt traumatic aortic rupture at the isthmus.* Eur J Cardiothorac Surg 2008; 34:616-22.
- 6) Siegel JH, Yang KH, Smith JA, et al.: *Computer Simulation and Validation of the Archimedes Lever Hypothesis as a Mechanism for Aortic Isthmus Disruption in a Case of Lateral Impact Motor Vehicle Crash: A Crash Injury Research Engineering Network (CIREN) Study.* J Trauma, 2006; 60:1072-82.
- 7) Dischinger PC, Siegel JH, Ho SM, et al.: *The effect of impact velocity on development of injury complications in multiple trauma motor vehicle crash patients.* Proc 39th AAAM, 1995; 39:367-79.
- 8) Dischinger PC, Cowley RA, Shankar BS, et al.: *The incidence of ruptured aorta among vehicular fatalities.* Proc AAAM; 1988; 32:15-23.
- 9) Katyal D, McLellan BA, Brenneman FD, et al.: *Lateral impact motor vehicle collisions: significant cause of blunt traumatic rupture of the thoracic aorta.* J Trauma, 1997; 42:769-72.
- 10) Strassman G.: *Traumatic rupture of the aorta.* Am Heart J, 1947; 33:508-15.
- 11) Sutuoius DJ, Schreiber JT, Helmsworth JA, et al.: *Traumatic disruption of the thoracic aorta.* J Trauma, 1973; 13:583-89.
- 12) Richens D, Kotidis K, Neale M, et al.: *Rupture of the aorta following road traffic accidents in the United Kingdom 1992-999. The results of the Co-operative Crash Injury Study.* Eur J Cardiothorac Surg, 2003; 23:143-48.
- 13) Fattori R., Buttazzi K., Russo V., et al.: *Evolving concepts in the treatment of traumatic aortic injury.* J Cardiovasc Surg, 2007; 48:625-31.
- 14) Parmley LF, Mattingly TW, Manion WC, et al.: *Nonpenetrating traumatic injury of the aorta.* Circulation, 1958; 17:1086-101.
- 15) Pate JW: *Is traumatic rupture of the aorta misunderstood?* Ann Thorac Surg, 1994; 57:530-31.
- 16) Mattox K, Wall MJ: *Historical review of blunt injury to the thoracic aorta.* Chest Surg Clin North Am, 2000; 10:167-82.
- 17) Ungar TC, Wolf SJ, Haukoos JS, Dyer DS, et al.: *Derivation of a clinical decision rule to exclude thoracic aortic imaging in patients with blunt chest trauma after motor vehicle collisions.* J Trauma, 2006; 61:1150-155.
- 18) Roisinblit JM, Allende NG, Neira JA, et al.: *Local Thrombus as an Isolated Sign of Traumatic Aortic Injury.* Echocardiography, 2002; 19:63-65.
- 19) Zissimopoulos I, Tsoukas A, Koliandris I, et al.: *Traumatic aortic transection.* Echocardiography, 2005; 22:35-38.
- 20) Sammer M, Wang EC, Blackmore C, et al.: *Indeterminate CT Angiography in blunt thoracic trauma: Is CT Angiography enough?* AJR 2007; 189:603-08.
- 21) Mirvis SE, Shanmuganathan K: *Diagnosis of blunt traumatic aortic injury 2007: Still a nemesis.* Eur Journ Radiol, 2007; 64:27-40.
- 22) Steenburg SD, Ravenel JG: *Multi-detector computed tomography findings of atypical blunt traumatic aortic injuries: a pictorial review.* Emerg Radiol, 2007; 14:143-50.
- 23) Ellis JD, Mayo JR: *Computed Tomography Evaluation of Traumatic Rupture of the Thoracic Aorta: An Outcome Study.* Can J Assoc Radiol, 2007; 58:22-26.
- 24) Bruckner BA, DiBardino DJ, Cumbie TC, et al.: *Critical evaluation of chest computed tomography scans for blunt descending thoracic aortic injury.* Ann Thorac Surg, 2006; 81:1339-47.
- 25) Fisher RG, Sanchez-Torres M, Whigham CJ et al.: *"Lumps" and "bumps" that mimic acute aortic and brachiocephalic vessel injury.* Radiographics, 1997; 17:825-34.
- 26) Fattori R., Celletti F., Bertaccini P., et al.: *Delayed surgery of traumatic aortic rupture: Role of magnetic resonance imaging.* Circulation, 1996; 94:2865-70.
- 27) Maggisano R., Natheus A., Alexandrova NA, et al.: *Ruptures trumatiques de l'aorte thoracique: l'intervention d'urgence est-elle toujours indiquée?* Ann Chir Vasc, 1995; 9:44-52.
- 28) Chip-Jin NG, Jih-Chang C, Li-Jen W: *Diagnostic value of the helical CT scan for traumatic aortic injury: correlation with mortality and early rupture.* J Emerg Med, 2006; 30:277-282.
- 29) Fabian TC, Davis KA, Gavant ML, et al.: *Prospective study of blunt aortic injury: helical CT is diagnostic and antihypertensive therapy reduces rupture.* Ann Surg, 1998; 227:666-76.
- 30) Pate JW, Gavant ML, Weiman DS, et al.: *Traumatic rupture of the aortic isthmus: Program of selective management.* World J Surg, 1999; 23:59-63.
- 31) Wahl WL, Michaels AJ, Wang SC, et al.: *Blunt thoracic aortic injury: delayed or early repair?* J Trauma, 1999; 47:254-59.
- 32) Akins CW, Buckley MJ, Daggett W, et al.: *Acute traumatic disruption of the thoracic aorta: A ten-year experience.* Ann Thorac Surg, 1981; 31:305-9.
- 33) Borman KR, Aurbakken CM, Weigelt JA.: *Treatment priorities in combined blunt abdominal and aortic trauma.* Am J Surg, 1982; 144:728-32.
- 34) Dake MD, Miller DC, Semba CP, et al.: *Transluminal placement of endovascular stent-grafts for the treatment of descending thoracic aortic aneurysms.* N Engl J Med, 1994; 331:1729-734.
- 35) Pierangeli A, Turinetto B, Galli R, et al.: *Delayed treatment of isthmus aortic rupture.* Cardiovascular Surgery, 2000; 8:280-83.
- 36) Pacini D, Angeli E, Fattori R, et al.: *Traumatic rupture of the thoracic aorta: Ten years of delayed management.* J Thorac Cardiovasc Surg, 2005; 129:880-84.
- 37) Kipfer B, Leupi F, Schepbach P, et al.: *Acute traumatic rupture of the thoracic aorta: immediate or delayed surgical repair?* Eur J Cardiothorac Surg, 1994; 8:30-33.
- 38) Von Oppell UO, Dunne TT, De Groot MK, et al.: *Traumatic aortic rupture: Twenty-year metaanalysis of mortality and risk of paraplegia.* Ann Thorac Surg, 1994; 58:585-95.
- 39) Holmes JH, Bloch RD, Hall RA, et al.: *Natural history of traumatic rupture of the thoracic aorta managed nonoperatively: A longitudinal analysis.* Ann Thorac Surg, 2002; 73:1149-54.

- 40) Cook J, Salerno C, Krishnadasan B: *The effect of changing presentation and management on the outcome of blunt rupture of the thoracic aorta*. J Thorac Cardiovasc Surg, 2006; 131:594-600.
- 41) Bortone AS, Schena S, D'Agostino D, et al.: *Immediate versus delayed endovascular treatment of post-traumatic aortic pseudoaneurysms and type B dissections: Retrospective analysis and premises to the upcoming European trial*. Circulation, 2002; 106:234-40.
- 42) Carter Y, Mark Meissner M, Bulger E: *Anatomical considerations in the surgical management of blunt thoracic aortic injury*. J Vasc Surg, 2001; 34:628-33.
- 43) Schumacher H, Böckler D, von Tengge-Kobligk H: *Acute traumatic aortic tear: open versus stent-graft repair*. Semin Vasc Surg, 2006; 19:48-59.
- 44) Weiman DS, Gurbuz AT, Andrei Gursky A, et al.: *Comparison of spinal cord protection utilizing left atrial-femoral with femoral-femoral bypass in patients with traumatic rupture of the aortic isthmus*. World J Surg, 2006; 30:1638-641.
- 45) Semba CP, Kato N, Kee ST, et al.: *Acute rupture of the descending thoracic aorta: repair with use of endovascular stent-grafts*. J Vasc Interv Radiol, 1997; 8:337-42.
- 46) Alric P, Berthet JP, Branchereau P, et al.: *Endovascular repair for acute rupture of the descending thoracic aorta*. J Endovasc Ther, 2002; 9(suppl 2):II51-II59.
- 47) Lachat M, Pfammatter T, Witzke H, et al.: *Acute traumatic aortic rupture: early stentgraft repair*. Eur J Cardiothorac Surg, 2002; 21:959-63.
- 48) Kato N, Dake MD, Miller DC, et al.: *Traumatic thoracic aortic aneurysm: treatment with endovascular stent-grafts*. Radiol, 1997; 205:657-62.
- 49) Karmy-Jones R, Hoffer E, Meissner MH, et al.: *Endovascular stent-grafts and aortic rupture: a case series*. J Trauma, 2003; 55:805-10.
- 50) Scheinert D, Krankenberg H, Schmidt A, et al.: *Endoluminal stentgraft placement for acute rupture of the descending thoracic aorta*. Eur Heart J, 2004; 25:694-700.
- 51) Amabile P, Collart F, Gariboldi V, et al.: *Surgical versus endovascular treatment of traumatic thoracic aortic rupture*. J Vasc Surg, 2004; 40:873-9.
- 52) Dunham MB, Zygun D, Petrasek P, et al.: *Endovascular stent-grafts for acute blunt aortic injury*. J Trauma, 2004; 56:1173-178.
- 53) Melnitchouk S, Pfammatter T, Kadner A, et al.: *Emergency stent-graft placement for hemorrhage control in acute thoracic aortic rupture*. Eur J Cardiothorac Surg, 2004; 25:1032-38.
- 54) Morishita K, Kurimoto Y, Kawaharada N, et al.: *Descending thoracic aortic rupture: role of endovascular stent-grafting*. Ann Thorac Surg, 2004; 78:1630-34.
- 55) Neuhauser B, Czermak B, Jaschke W, et al.: *Stent-graft repair for acute traumatic thoracic aortic rupture*. Am Surg, 2004; 70:1039-44.
- 56) Doss M, Wood JP, Balzer J, et al.: *Emergency endovascular interventions for acute thoracic aortic rupture: four-year follow-up*. J Thorac Cardiovasc Surg, 2005; 129:645-51.
- 57) Eggebrecht H, Schmermund A, Herold U.: *Endovascular stent-graft placement for acute and contained rupture of the descending thoracic aorta*. Cath and Cardiovasc Interv, 2005; 66:474-82.
- 58) Michelet P, Roch A, Amabile P, et al.: *Endovascular treatment of isthmus aortic rupture: use of second generation stent grafts*. Ann Fr d'Anesthésie et de Réanimation, 2005; 24:355-60.
- 59) Peterson BG, Matsumura JS, Morasch MD, et al.: *Percutaneous endovascular repair of blunt thoracic aortic transection*. J Trauma, 2005; 59:1062-65.
- 60) Rousseau H, Dambrin C, Marcheix B, et al.: *Acute traumatic aortic rupture: A comparison of surgical and stent-graft repair*. J Thorac Cardiovasc Surg, 2005; 129:1050-55.
- 61) Agostinelli A, Stefano Sacconi S, Bruno Borrello B, et al.: *Immediate endovascular treatment of blunt aortic injury: Our therapeutic strategy*. J Thorac Cardiovasc Surg, 2006; 131:1053-57.
- 62) Hoorweg LL, Dinkelman MK, Goslings JC, et al.: *Endovascular management of traumatic ruptures of the thoracic aorta: A retrospective multicenter analysis of 28 cases in The Netherlands*. J Vasc Surg, 2006; 43:1096-102.
- 63) Marcheix B, Dambrin C, Bolduc JP, et al.: *Endovascular repair of traumatic rupture of the aortic isthmus: Midterm results*. J Thorac Cardiovasc Surg, 2006; 132:1037-41.
- 64) Pratesi C, Dorigo W, Nicola Troisi N, et al.: *Acute traumatic rupture of the descending thoracic aorta: Endovascular treatment*. Am J Surg, 2006; 192:291-95.
- 65) Reed AB, Thompson JK, Crafton CJ, et al.: *Timing of endovascular repair of blunt traumatic thoracic aortic transections*. J Vasc Surg, 2006; 43:684-88.
- 66) Neschis DG, Moaine S, Gutta R, et al.: *Twenty consecutive cases of endograft repair of traumatic aortic disruption: Lessons learned*. J Vasc Surg, 2007; 45:487-92.
- 67) Orend KH, Zarbis N, H. Schelzig H, et al.: *Endovascular treatment (EVT) of acute traumatic lesions of the descending thoracic aorta: 7 Years' Experience*. Eur J Vasc Endovasc Surg 2007; 34:666-72.
- 68) Raupach J, Ferko A, Lojik M.: *Endovascular Treatment of Acute and Chronic Thoracic Aortic Injury*. Cardiovasc Intervent Radiol, 2007; 30:1117-123.
- 69) Steingruber IE, Czermak BV, Chemelli A, et al.: *Placement of endovascular stent grafts for emergency repair of acute traumatic aortic rupture: a single-center experience*. Eur Radiol, 2007; 17:1727-737.
- 70) Buz S, Zipfel B, Mulahasanovic S, et al.: *Conventional surgical repair and endovascular treatment of acute traumatic aortic rupture*. Eur J Cardio-thoracic Surg, 2008; 33:143-51.
- 71) Canaud L, Alric P, Branchereau P, et al.: *Lessons learned from midterm follow-up of endovascular repair for traumatic rupture of the aortic isthmus*. J Vasc Surg, 2008; 47:733-38.
- 72) Ouriel K Editor, Walsh SR, Tang TY, et al.: *Endovascular stenting versus open surgery for thoracic aortic disease: Systematic review and meta-analysis of perioperative results*. J Vasc Surg, 2008; 47:1094-98.
- 73) Canaud L, Alric P, Laurent M, et al.: *Proximal fixation of thoracic stent-grafts as a function of oversizing and increasing aortic arch angulation in human cadaveric aortas*. J Endovasc Ther, 2008; 15:326-34.
- 74) Eggebrecht H, Baumgart D, Radecke K, et al.: *Aortoesophageal*

- fistula secondary to stent-graft repair of the thoracic aorta.* J Endovasc Ther, 2004; 11:161-67.
- 75) Bell RE, Taylor PR, Aukett M, et al.: *Results of urgent and emergency thoracic procedures treated by endoluminal repair.* Eur J Vasc Endovasc Surg, 2003; 25:527-31.
- 76) Kirkpatrick C Santo, FRCS, Peter Guest, FRCP, FRCR, Ian McCafferty: *Aorto-esophageal fistula secondary to stent-graft repair of the thoracic aorta after previous surgical coarctation repair.* J Thorac Cardiovasc Surg, 2007; 134:1585-86.
- 77) Hance KA, Hsu L, Eskew T, et al.: *Secondary aorto-esophageal fistula after endoluminal exclusion because of thoracic aortic transection.* J Vasc Surg, 2003; 37:886-88.
- 78) Idu MM, Reekers JA, Balm R, et al.: *Collapse of a stent-graft following treatment of a traumatic thoracic aortic rupture.* J Endovasc Ther, 2005; 12:503-07.
- 79) Mohan IV, Laheij RJF, Harris PL.: *Risk factors for endoleak and the evidence for stent-graft oversizing in patients undergoing endovascular aneurysm repair.* Eur J Vasc Endovasc Surg, 2001; 21:344-49.
- 80) Malina M, Lindblad B, Ivancev K, et al.: *Endovascular AAA exclusion: will stents with hooks and barbs prevent stent-graft migration?* J Endovasc Surg, 1998; 5:310-17.
- 81) Leurs LJ, Bell R, Degrieck Y, et al.: *Endovascular treatment of thoracic aortic diseases: combined experience from the EUROSTAR and United Kingdom Thoracic Endograft registries.* J Vasc Surg, 2004; 40:670-80.
- 82) Liffman K, Sutalo ID, Lawrence-Brown MM, et al.: *Movement and dislocation of modular stent-grafts due to pulsatile flow and the pressure difference between the stent-graft and the aneurysm sac.* J Endovasc Ther, 2006; 13:51-61.
- 83) Muhs BE, Balm R, White GH, et al.: *Anatomic factors associated with acute endograft collapse after Gore TAG treatment of thoracic aortic dissection or traumatic rupture.* J Vasc Surg, 2007; 45:655-61.
- 84) Noor N, Sadat U, Hayes PD.: *Management of the Left Subclavian Artery During Endovascular Repair of the Thoracic Aorta.* J Endovasc Ther, 2008; 15:168-76.
- 85) Schoder M, Grabenwoger M, Holzenbein T, et al.: *Endovascular repair of the thoracic aorta necessitating anchoring of the stent-graft across the arch vessels.* J Thorac Cardiovasc Surg, 2006; 131:380-87.
- 86) Woo EY, Bavaria JE, Pochettino A, et al.: *Techniques for preserving vertebral artery perfusion during thoracic aortic stent-grafting requiring aortic arch landing.* Vasc Endovascular Surg, 2006; 40:367-73.
- 87) Steinberg GK, Drake CG, Peerless SJ.: *Deliberate basilar or vertebral artery occlusion in the treatment of intracranial aneurysms. Immediate results and long-term outcome in 201 patients.* J Neurosurg, 1993; 79:161-73.
- 88) Reece TB, Gazoni LM, Cherry KJ, et al.: *Reevaluating the need for left subclavian artery revascularization with thoracic endovascular. Aortic Repair Ann Thorac Surg, 2007; 84:1201-05.*
- 89) Makaroun MS, Dillavou ED, Wheatley GH, et al.: *Five-year results of endovascular treatment with the Gore TAG device compared with open repair of thoracic aortic aneurysms.* J Vasc Surg, 2008; 47:912-18.