Rationale for a Conservative Approach and Arguments Against Aggressive Surgical Approaches

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Abstract

Type A dissection is one of the few remaining true surgical emergencies. Without surgery, the risk of death is still as high as 50 % at 48 h. Patients must therefore have a repair as early as possible to have a chance of survival. In the last four to five decades, surgeons have gained experience of more complex operations for the aortic root and arch in the elective setting. Encouraged by their success, some have proposed performing the same complex operations in cases of type A dissection. In this chapter we put forward a number of arguments against these aggressive approaches. The fundamental counter-argument is that the emergency dissection patient is quite different from the elective aneurysm patient. In aneurysm cases, the patient arrives in theatre in a stable situation and any malperfusion that may exist has been compensated for. Patients presenting as emergencies with type A aortic dissection are unstable and ill-prepared for the insult of the operation. The accompanying systemic inflammatory response to arterial dissection will be compounded by extra-corporeal circulation and hypothermia if used. The "instantaneous risk to life" imposed by type A aortic dissection immediately follows this tearing though the arterial media and only falls below risks offered by surgery after 14 days. Considering these issues, operations for these patients must be prompt and swift in order to minimise the additional insult of surgery. This chapter summarises the evidence in support of this position.

Keywords

Type A aortic dissection • Aggressive vs. non-aggressive approaches • Goals of surgery

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Establishing the Goals of Surgery for Acute Type A Dissection

To establish the goal of surgery, the immediate risk of the disease must be appreciated. In type A dissection, the immediate risk to life is due to intra-pericardial rupture and cardiac tamponade, myocardial ischaemia through coronary involvement or acute heart failure through involvement of the aortic valve and regurgitation. Therefore the goal of surgery in the acute phase must be to eliminate these three sequelae. However some centres advocate aggressive surgery to correct all the anatomical defects caused by the dissection. To illustrate the point, it is well-recognised that malperfusion syndromes such as stroke, renal impairment and peripheral limb ischaemia occur in Type A dissection. Revascularisation of these territories is one of the justifications given for extending the operation beyond the three goals mentioned above. However, these malperfusion syndromes do not represent an immediate risk to the patient's life and may be corrected through establishing true lumen perfusion by surgical obliteration of the false lumen in the ascending aorta. Rather than reversing ischaemia, a complex operation actually adds further injury by necessitating prolonged extra-corporeal circulation or deeper hypothermia. These interventions increase the risk of complications to a patient who is in an evolving pathological state, potentially increasing the risk of early death. In contrast a conservative operation by diverting blood from the false lumen back to the true lumen will shelter the blood elements from the aortic media and limit the activation of the inflammatory cascades. In this chapter we will examine the arguments that have been put forward for aggressive operations in acute aortic dissection, many supported by excellent surgical results from expert aortic surgeons in high volume centres. Our counter-argument is twofold. Firstly, given that risk of death rises hourly, the recommended surgery for Type A dissection must be within the ability of all cardiac surgeons (not just aortic specialists) so that the patient may be treated locally and as soon as possible after onset and recognition of their dissection. Secondly, aggressive surgery for acute type A dissection prolongs bypass and may necessitate hypothermia, both of which 'add insult to injury' for no evidence based gain.

Definitions of 'Acute', 'Type A Dissection' and 'Conservative Surgery'

With the three goals of surgery foremost in our mind, it is useful to clarify the definitions of dissection 'age', 'type' and also to establish what is meant by conservative surgery.

Since DeBakey's seminal paper on aortic dissection in 1982 [1], a threshold of 14 days has been accepted internationally to distinguish acute from chronic dissection. The rationale for this derives from studies of the natural history of medically treated type A dissection, which showed that the cumulative mortality rises steeply during the first 1–2 weeks after onset of symptoms and then levels off. The initial steep rise in mortality is due to cardiac tamponade, acute severe AR or myocardial infarction with the ongoing mortality burden likely due to stroke, pneumonia and multi-organ failure. Beyond the 2 week point, the moment-to moment risk of dissection-related death is overtaken by the risk from operative death. It is interesting (although not surprising) to note that at the same point that the natural history of type A dissection changes, the dissected aortic wall is beginning to regain its strength through natural repair. It has been observed that at 2 weeks post-dissection, the aortic adventitia appears able to hold sutures almost as well as non-dissected aorta (Elefteriades JA, 12/07/12, personal communication). Research has corroborated that at 10–14 days post-dissection, fibroblast activity in the dissected aortic wall rises, beginning the process of scar formation (Elefteriades JA, 12/07/12, personal communication). Therefore the time-frame of 2 weeks to separate acute from chronic dissection is probably reasonable from the points of view of both patient and aorta. Interestingly, the behaviour of Type B (extra-pericardial) dissection is almost exactly the opposite. Studies have shown that for Type B dissection, medical management is 'safer or no more dangerous than

surgical management for the first 14 days. This suggests that 'risk' in type A dissection is largely determined by the anatomy and specifically—by the confinement of the ascending aorta within the finite volume of the pericardial sac. Rupture of the aorta into the pericardium causes tamponade. In a single heartbeat, 50–60 ml of blood can potentially enter the pericardial space which is designed to hold 30 ml. Within a few heart beats, the pressure of extravasated blood in the pericardium will exceed the filling pressure of the ventricles and cardiac output will fail through inability to fill the compressed ventricles (tamponade). Myocardial ischaemia arises if the dissection flap extends to compromise blood flow into the coronary arteries. Whether one or both coronaries are affected, the patient may suffer myocardial infarction. In cases where aortic dissection extends in to the root and disrupts the suspension of the aortic valve leaflets, severe aortic regurgitation develops. The left ventricle becomes volume overloaded and may compensate poorly, resulting in acute left ventricular failure, which can only be reversed by restoration of aortic valve competence.

Complications that occur following dissection of the extra-pericardial aorta (stroke, renal or bowel ischaemia) may be disabling but are not immediately life-threatening for patients who have survived to hospital admission and diagnosis. Therefore we regard Type A dissections as those affecting the intrapericardial (rather than ascending) aorta and type B dissection as those affecting the extra-pericardial aorta.

Conservative surgery in this context is defined as the minimum surgery (and minimum bypass time) required to satisfy the three goals of surgery in acute dissection of the intrapericardial aorta. We do not agree that routine inspection of the arch, or beyond, for resection of the "primary intimal tear" (entry point) should form part of the surgical strategy, since this is a feature of Type B dissection, which we have already established is better managed by medical therapy in the acute phase. According to this definition conservative surgery would normally comprise replacement of the tubular part of the intrapericardial aorta along with restoration of a functional aortic valve and obliteration or replacement of any false lumen present in the aortic root. Techniques for the valve are replacement or resuspension minimising the cross-clamp and cardiopulmonary bypass times. Techniques for the root include a modified Bentall procedure or obliteration of the false lumen using biological glue. Aortic arch surgery and complex valve-sparing root reconstructions (VSRR) such as the David and Yacoub operations do not qualify therefore as conservative strategies. Our guiding principle is to perform the swiftest procedure to achieve the three goals of dissection surgery.

Rationale for the Three Goals of Surgery

The three goals of surgery in dissection reflect the three immediate risks to life imposed by type A dissection. Observational studies by DeBakey, Sato and others have suggested that severe AR, tamponade and myocardial infarction probably represent 60 % or so of deaths in those patients who reach hospital [1, 2], with the remainder of deaths resulting from other recognised complications of dissection. Studies from the IRAD (International Registry of Aortic Dissection) database and other authors have shown that in patients with dissection, the incidence of aortic valve regurgitation is 40–44 %, tamponade 21–37 % and shock 18–21 %. Therefore not only are these three diagnoses the most dangerous, they are also the most common presenting complications [3–5]. However, it is important to recognise that beyond these complications, the patient with acute dissection is faced with a systemic inflammatory response syndrome (SIRS) which can be equally injurious. Despite significant progress in our understanding and technology, cardiopulmonary bypass and hypothermia probably compound this acute inflammatory response. Many patients with dissection present with acidaemia secondary to either low cardiac output, malperfusion associated with an obstructed branch artery, or both. Cardiopulmonary bypass introduces coagulopathy and hypothermia is the final element in the so-called 'lethal triad' recognised as life threatening in trauma patients. In the context of aortic dissection this triad (hypothermia, acidaemia and coagulopathy) has been implicated in early deaths, with studies by

Apaydin and others showing that intra-operative blood loss >500 ml, clotting product transfusion and pre-operative malperfusion were all predictive of early mortality [6]. Until we are able to avert tamponade, MI and free AR without CPB, the next best strategy is to keep CPB time to a minimum and avoid deep hypothermia since both of these factors are associated with increased blood loss and transfusion requirement, renal failure, stroke and mediastinitis. Therefore inspection with or without resection of the arch cannot be justified.

The Arguments in Favour of Aggressive Procedures

The potential gains in augmenting an operation for dissection are threefold:

- To identify and resect the primary 'entry' tear in the intima to prevent immediate rupture and obliterate the false lumen.
- To halt or reverse the ischemic injury to downstream vascular beds (e.g. stroke, renal failure, limb ischaemia)
- To prevent a redo sternotomy and second operations for either the proximal or distal aorta.

The evidence on this topic is limited to low volume case series. This is due partly to the rarity of aortic dissection, but also to the rapid changes in surgical strategy over the last 50 years. Consequently, any new technique can only be applied to a small number of patients and so it is impractical to run randomised trials comparing arguably improved treatments or operations. Therefore it must be accepted that the evidence on both sides is anecdotal (class 2 or lower). Nonetheless, in the paragraphs that follow, we will put forward the evidence against these three 'secondary' goals of dissection surgery.

Arguments Against Resecting the Primary Tear

Since the 1970s, techniques for managing myocardial protection and hypothermia have improved steadily. Encouraged by these advances and probable improvement of cerebral protection, some groups have hypothesised that resecting the entry tear should improve long term outcomes for patients with Type A dissection. At face value, this appears logical—if blood were still able to enter the false lumen through the entry tear, the patient must be vulnerable to re-dissection or rupture. However, only 10–20 % of entry tears are located in the transverse arch in cases of acute dissection [7–9]. It is also recognised that there may be several intimal tears throughout the aorta and little indication as to which is "the entry point" to the false lumen of the dissected aorta. It would appear that routine aortic inspection is a low yield strategy and especially unattractive knowing that these adjuncts may be harmful. Nonetheless it could be considered worthwhile if resecting the tear resulted in clinical benefit in terms of either reduced risk of death from rupture or reduced risk of redo surgery due to subsequent aneurysm formation. Considering reduction of risk of rupture, Moon and others reviewed the outcomes of resecting entry tears in the arch. These workers demonstrated that this aggressive approach does not change early mortality or 10 year survival (early mortality was 17 % in the ascending replacement group versus 22 % in the ascending + arch surgery group, $p=0.7$). Ten year survival was 61 % with resection of the primary arch tear and 52 % without, $p>0.40$ [4]. This reiterates findings from the Stanford group who evaluated the same strategy with an operative mortality of 29 % after arch repair, 37 % without arch repair, $p=0.94$ and a 4 year survival of 71 % with arch repair, 44 % without arch repair [3, 10], but this was not statistically significant either. Therefore it is inferred that rupture of the aorta is not significantly affected by resection of an arch tear. Multiple centres have shown that a strategy of aggressive 'chasing' of the entry tear makes the operation considerably longer. Yun et al. published their retrospective analysis of acute dissection patients in 1991 [3] and demonstrated a mean CPB duration of 175 mins for patients undergoing arch surgery versus 137 min for those undergoing ascending aortic surgery alone. This is a worrying plan when considering that increased duration of cardiopulmonary bypass increases the risk of post-operative morbidity (respiratory failure, pneumonia,

renal failure etc.) as well as incurring coagulopathy and therefore increasing transfusion requirement. In addition deep hypothermia compounds the deleterious effects of CPB on coagulation, increases the risk of cerebral air emboli during cooling and rewarming and alters vascular permeability leading to cerebral oedema [11–14]. Nor is selective antegrade cerebral perfusion risk-free with under-perfusion leading to brain injury and over-perfusion leading to haemorrhage or oedema. In the face of such risks, operating on the arch in acute dissection has to be well justified. Many surgeons (including the authors) prefer to utilise DHCA in dissection for the purpose of performing the distal anastomosis with no cross-clamp in situ. This we believe encourages the construction of a secure and haemostatic distal anastomosis. In these situations the risks of DHCA is justified by the improved technical result of the distal suture line minimising the risk of haemorrhage, transfusion and malperfusion. However, resecting the primary tear has not been shown to reduce early or late mortality. It is established that non-resection of the primary tear is associated with an increased risk of persistence of false lumen patency and so, aneurysm formation. This will be discussed below.

Take Home Message

The entry tear is only present in the arch in 10–20 % of cases of Type A dissection. Routine resection of arch tears does not improve early, mid-term (4 year) or long term (10 year) mortality.

Can Aggressive Surgery Halt or Reverse Down-Stream Ischaemia?

Observational studies have shown that as many as a third of patients (28–33 %) with Type A dissection have some form of end-organ ischaemia on presentation [4, 15–17]. This may be coronary (5–26 %), cerebral (6–52 %) [4, 15, 17, 18], renal (6.9–16 %) [2, 15] Sato, 16 % Shiiya), mesenteric (3–16 %) [15, 17] or peripheral limb ischaemia (11.6–52 %) [15, 17, 18]. These studies all demonstrated that patients presenting with any kind of malperfusion syndrome had a higher risk of death following surgery (42–58 % versus 8–14 %, $p<0.001$) [6, 18]. It has been hypothesised that this may be lessened by performing aggressive revascularisation procedures for the affected organs. These procedures include concomitant arch repair/replacement to improve cerebral perfusion, coronary artery bypass grafting to address coronary malperfusion and endovascular or surgical fenestration to revascularise the abdominal viscera or limbs. Shiiya et al. considered that end-organ ischaemia is either caused by obstruction of branch vessels by the aortic dissection flap (‘aortic type malperfusion’) or by dissection into the branch artery (‘branch type malperfusion’) [15]. In their paper, 30 patients were reviewed with Type A dissection and malperfusion. All patients were all treated with ‘central aortic surgery’ including resection of the primary tear. Direct extra-anatomical bypass grafting was performed to address any pre-operatively malperfused organ (coronary artery bypass grafts for five patients with coronary ischaemia, arch vessel reconstruction for eight patients with cerebral malperfusion, bypass grafting of the celiac axis and superior mesenteric artery for two cases of bowel ischaemia and femorofemoral crossover grafting for two cases of unilateral lower limb ischaemia). In this series, central aortic surgery with resection of the entry tear was not effective in seven of eight cases of branch type malperfusion, which represents 81 % of cases of malperfusion. Staged stenting of the affected visceral arteries, however, proved to be very effective in these cases and probably represents a lower risk adjunct that avoids prolonged bypass time or hypothermia In the case of coronary malperfusion, with its implications for separating from CPB, it seems logical that surgical CABG would be preferred to staged stenting since it adds only a short CPB time but can improve delivery of cardioplegia to protect the ischaemic myocardium during surgery. However, in the case of cerebral malperfusion the risk-benefit analysis is more complex. Several groups have advocated aggressive surgery to restore true lumen flow to the cerebral circulation since the head and neck vessels can be accessed through the median sternotomy. Pre-operative ischaemic stroke is known to be a

risk factor for mortality in patients with Type A dissection [19] and is thought to have an incidence of 6–16 % [10, 19, 20]. Ischaemic strokes associated with Type A dissection are most frequently (69–71 %) bihemispheric [21, 22]. However, dissection of the arch vessels is only seen in 43 % of these cases [21]. Furthermore only 22 % of patients with dissection of the arch vessels suffer an ischaemic stroke representing 8 % of the Type A dissection population. This is probably due to the protection offered by collateral flow in the Circle of Willis. Other mechanisms for ischaemic stroke include thromboembolism or hypotensive episodes in these patients who are likely to have some degree of vasculopathy. Despite these data separating stroke from arch or arch vessel dissection, it could be argued that the potential implications of stroke are so severe as to warrant an attempt to secure the cerebral circulation by means of arch replacement—30 day mortality after surgery is 20–40 % for those with neurological impairment [21, 23] versus 0 % for those without in one small study [23]. Furthermore, 12 % of patients with a post-operative neurological deficit will require permanent care and 14 % will need some assistance with daily living [21]. Unfortunately reversal of cerebral injury is by no means guaranteed by arch surgery. Morimoto et al. reviewed a series of 41 consecutive patients with acute type A dissection complicated by cerebral malperfusion [23]. This study also analysed predictors of neurological recovery, which was seen in 63 % of patients post-operatively. The authors found that neither extent of aortic resection nor method of cerebral protection were predictors of neurological recovery. Tanaka et al. published their series of 16 patients with cerebral malperfusion and corresponding neurological deficit due to dissection involving the arch vessels [24]. All of these patients underwent some form of arch surgery (partial, hemi-arch or total arch) with intra-operative cerebral perfusion (either retrograde or antegrade) in an effort to restore normal cerebral blood flow. However, the mortality rate was 56 %, with six deaths (37.5 %) due to severe brain injury. Estrera et al. have contributed to the debate with their series of 16 patients with pre-operative stroke [25]. The operative strategy was to replace the ascending aorta, inspect the arch under DHCA with retrograde cerebral perfusion and resect any entry tears in the proximal arch. Only two patients (14 %) returned to normal neurological function. The paper's authors state that some degree of neurological improvement was seen in six patients but do not qualify this with a review of functional status. These sparse and low volume case series have demonstrated that arch surgery in type A dissection at least does not worsen neurological function. However, the authors suggest that the hypothermia and/or circulatory arrest required to perform arch surgery incur a risk of prolonged CPB time, coagulopathy, and transfusion requirement that *is* associated with increased mortality and morbidity. This cannot be justified since it cannot be shown to reliably salvage cerebral function.

Take Home Message

Six to fifty-two percent of Type A dissection patients may present with anatomical cerebral malperfusion, but only a fifth of these will go on to develop ischaemic stroke. Arch surgery only returns 14 % of these patients to normal neurology, but increases the risk of mortality to 16–34 % from 4.5 % to 11.1 % if only the proximal aorta is operated upon.

Aggressive Surgery to Avoid Reoperation: The Issue of the Patent False Lumen (Pfl)

Several studies have indicated that non-resection of entry tears in patients with Type A dissection is associated with an increased risk of long-term patency of the false lumen in the residual aorta. The incidence of patent false lumen (PFL) after any surgical repair of type A dissection is in the region of 47–60 % [26]. In one study by Ergin et al., resection of the arch in the presence of an arch tear was associated with a lower incidence of patent false lumen (23 % versus 60 %) at mean follow-up of 4.4 years [26], and this has been

corroborated by others. Retrospective data published by Kimura et al. [27] showed that in the presence of a patent false lumen, aneurysmal expansion is accelerated in all aortic segments compared to cases where the false lumen was obliterated (OFL)—1.1 mm/year versus−0.41 mm/year in the arch, 1.9 mm/year versus−0.71 mm/year in the proximal descending aorta and 1.3 mm/year versus−0.7 mm/year in the distal descending aorta. Similar findings (5.6 mm/year PFL versus 1.1 mm/year OFL, $p<0.05$) have been published by others [28]. The aortic dilatation witnessed in cases of PFL is mainly due to expansion of the false lumen itself and is probably due to the impaired stress distribution in the weakened aortic wall. PFL has been described by many authors as one of the determinants of late mortality, along with age, male sex and connective tissue disorders [29–31]. Ergin et al. [26] demonstrated a possible survival disadvantage at 5 years with failure to obliterate the false lumen (76 % PFL patients alive versus 95 % OFL patients) although this failed to achieve statistical significance. Even longer term data was provided by Fattouch et al. [32], showing that survival at 10 years is severely compromised for patients with PFL (59.8 % versus 89.8 %, $p=0.001$). Freedom from re-operation in this study was 63.7 % at 10 years for patients with PFL, compared to 94.2 % for patients with OFL. These would seem to be arguments in favour of aggressive surgery at the time of the initial dissection. However the authors propose the following counter-arguments. Firstly, the incidence of reoperation is low (circa 10 %) [33, 34] and the mortality risk from elective re-operation is less than that incurred by an extensive emergency operation at first presentation with Type A dissection. Secondly, the evidence that aggressive aortic resection prevents re-operation is controversial. To consider this issue in further detail we will divide the issue of late re-operations into re-operations for the proximal aorta and re-operations for the distal aorta.

In the context of the proximal aorta (i.e. the aortic valve and root), 'aggressive surgery' can be understood to mean complex valve sparing root reconstruction such as the modified David or Yacoub procedures. Various expert centres across the world have published reports suggesting that such procedures can be performed for elective aneurysm cases with no increase in cardiopulmonary bypass or cross-clamp times compared to Bentall procedures or implantation of valved conduits [35]. However, this is not matched by other centres. In other institutional reports, VSRR can be expected to add 30 min to CPB time and 40 min to cross-clamp time when compared against composite grafts [36] *in the elective setting*. A recent study showed that VSRR in the emergency setting adds a further 33 min to CPB time and 18 min to cross-clamp time (Leshnower). This 'cost' in terms of prolonged extra-corporeal circulation has to be justified. Expert centres have published reports that valve-sparing root reconstruction does not compromise survival in acute type A dissection (Subramanian) but unfortunately, they do still increase the risk of late reoperations (Concistre). These late re-operations for the proximal aorta are mostly (82 %) elective procedures [37] at a median of 69 months [38] after the original operation for dissection. The dominant indications are severe aortic valve regurgitation (27 %), root aneurysm (45 %), or a combination of the two, with a small proportion (3 %) of re-operations performed for infected prostheses. The risk of mortality from re-operative surgery for the non-infected proximal aorta is 4.5–11.1 % [38, 39] and is a procedure that can be undertaken by any competent general cardiac surgeon. 1, 5 and 10 years survival is reportedly 82, 74 and 62 %. In counterpoint, the risk of mortality from valve-sparing root reconstruction in dissection in an expert centre is of the order of 7–20 %. Therefore the authors recommend that conservative (widely reproducible) procedures should remain the gold standard for type A dissection accepting that if the patient survives the initial episode, there is a chance that a second elective operation may become necessary a few years later. It must be remembered that the role of stenting has not been established clearly in patients presenting late with complications following emergency surgery for type A dissection.

Late re-operations for the distal aorta are usually performed for aneurysm of either the arch,

the descending aorta or both. Moon et al. [4] and Tan et al. [40] both performed multivariate risk analysis showing that aggressive arch replacement at the time of dissection surgery does not reduce rate of late reoperation. Bekkers et al. reported their series of late re-operations after surgical repair of type A dissection. This centre had adopted an aggressive approach to the primary operation with a high frequency of arch replacement. Nonetheless, the re-operation rate for complications of the distal aorta was still 7.3 % in this study at a mean follow-up of 7.2 years. The peri-operative mortality rate was 17 % (3 of 17 patients), but this small population included a patient who had distal aortic replacement on Day 1 after the primary operation due to excessive bleeding. In most series, the mortality risk for distal reoperations is lower (0–4 %) than that for proximal reoperation (4.5–11.1 %)—presumably due to the alternative approach through the lateral chest rather than through a resternotomy. Kimura et al. [27] reported a 0 % mortality rate for their series of late reoperations on the distal aorta. In Kobuch et al's series [33] of late reoperations, the mortality rate was only 4.3 %, although it is not clear whether this was a proximal or distal re-operation. In contrast, the mortality rates quoted from centres who aggressively replace the arch in the primary operation is 16–34 % [41–44]. In light of these facts, the authors propose that aggressive arch replacement in dissection probably does increase the risk of patency of the false lumen, thereby increasing the rate of aneurysm formation. However, we contend that the risk associated with surveillance imaging and elective reoperation is much lower than the risk of hypothermia and arch replacement in the initial operation.

Conclusions

We re-emphasise that the patient on first presentation with Type A dissection has suffered a systemic insult that continues to evolve over a course of days to weeks after the event. The more complex the initial operation, the greater the risk of both morbidity and mortality. The nature of Type A dissection is such that patients require surgery as soon as possible to reduce the risk of tamponade, myocardial ischaemia and acute heart failure through aortic valve incompetence. So although the impressive results achieved by some centres performing aggressive complex repairs for Type A dissection are to be commended, it is not logical to base recommendations on these handful of small series. The only incontestable argument for aggressive surgery is that resection of the primary tear reduces the incidence of false lumen patency and in turn reduces the rate of growth of aneurysm in the residual aorta. This we believe can be better remedied by a comprehensive follow up with surveillance imaging and elective re-operation for aneurysm of the residual aorta rather than complex emergency surgery. We propose that aggressive emergency surgery probably equates to an overall greater risk to life than separating essential and conservative emergency surgery and subsequent operative procedures for late complications. Evidence for this suspicion is not yet available.

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