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Review

Poststernotomy mediastinitis: a review of conventional surgical treatments, vacuum-assisted closure therapy and presentation of the Lund University Hospital mediastinitis algorithm

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Summary

Poststernotomy mediastinitis, also commonly called deep sternal wound infection, is one of the most feared complications in patients undergoing cardiac surgery. The overall incidence of poststernotomy mediastinitis is relatively low, between 1% and 3%, however, this complication is associated with a significant mortality, usually reported to vary between 10% and 25%. At the present time, there is no general consensus regarding the appropriate surgical approach to mediastinitis following open-heart surgery and a wide range of wound-healing strategies have been established for the treatment of poststernotomy mediastinitis during the era of modern cardiac surgery. Conventional forms of treatment usually involve surgical revision with open dressings or closed irrigation, or reconstruction with vascularized soft tissue flaps such as omentum or pectoral muscle. Unfortunately, procedure-related morbidity is relatively frequent when using conventional treatments and the long-term clinical outcome has been unsatisfying. Vacuum-assisted closure is a novel treatment with an ingenious mechanism. This wound-healing technique is based on the application of local negative pressure to a wound. During the application of negative pressure to a sternal wound several advantageous features from conventional surgical treatment are combined. Recent publications have demonstrated encouraging clinical results, however, observations are still rather limited and the underlying mechanisms are largely unknown. This review provides an overview of the etiology and common risk factors for deep sternal wound infections and presents the historical development of conventional therapies. We also discuss the current experiences with VAC therapy in poststernotomy mediastinitis and summarize the current knowledge on the mechanisms by which VAC therapy promotes wound healing. Finally, we suggest a structured algorithm for using VAC therapy for treatment of poststernotomy mediastinitis in clinical practice.

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1. Poststernotomy mediastinitis—the definition

Poststernotomy mediastinitis, also commonly called deep sternal wound infection, is one of the most feared complications in patients undergoing cardiac surgery. The definition of mediastinitis has been established by the Center for Disease Control and Prevention in the USA [1]. According to these guidelines, diagnosis of mediastinitis requires at least one of the following:

- (1) an organism isolated from culture of mediastinal tissue or fluid;
- (2) evidence of mediastinitis seen during operation;
- (3) one of the following conditions: chest pain, sternal instability, or fever ($>38^{\circ}\text{C}$), in combination with either purulent discharge from the mediastinum or an organism

isolated from blood culture or culture of mediastinal drainage.

2. Microbial etiology

The microbial etiology of sternal wound infections varies and includes Gram-negative and Gram-positive bacteria (Table 1) as well as fungi. However, the most common causative pathogen involved in sternal wound infections is *Staphylococcus epidermidis* (CoNS) and *Staphylococcus aureus*, both from the normal flora of the skin [2–5]. Previously, the finding of CoNS in the wound could be dismissed as contamination and the pathogen was regarded as a relatively benign pathogen. However, *S. epidermidis* (CoNS) is now well known to be one of the most important agents of healthcare associated infections especially when foreign material is implanted, such as prosthetic heart valves, prosthetic joints, peritoneal dialysis catheters, intravascular catheters, and cerebral spinal fluids shunts. Another foreign body, steel wires, is used in almost all cardiac

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Table 1
Culture-verified poststernotomy mediastinitis at Lund University Hospital, 1994–2003

Bacterial strains	VAC therapy		Conventional treatment	
	n	% ^a	n	% ^a
CoNS	34	56	27	68
<i>S. aureus</i>	8	13	2	5
<i>E. cloacae</i>	4	7	1	3
<i>Klebsiella oxytoca</i>	3	5	0	0
<i>Propionibacterium acnes</i>	2	3	0	0
<i>Escherichia coli</i>	1	2	0	0
<i>Bacterioides fragilis</i>	1	2	0	0
<i>Klebsiella pneumoniae</i>	1	2	2	5
CoNS + <i>S. aureus</i>	3	5	2	5
CoNS + <i>E. coli</i>	2	3	0	0
CoNS + <i>Proteus mirabilis</i>	1	2	1	3
CoNS + <i>Pseudomonas aeruginosa</i>	1	2	0	0
CoNS + <i>Enterococcus faecalis</i>	0	0	3	8
CoNS + <i>Enterobacter aerogenes</i>	0	0	1	3
<i>S. aureus</i> + <i>Citrobacter freundii</i>	0	0	1	3

CoNS: coagulase-negative Staphylococci strains; *S. aureus*: *Staphylococcus aureus*; *E. cloacae*: *Enterobacter cloacae*; *E. coli*: *Escherichia coli*.

^a The sum of the percentages exceeds 100% due to rounding.

surgery procedures when closing the sternotomy and CoNS has emerged as the most important pathogen in poststernotomy mediastinitis, responsible for 43% to 64% of all cases in recent studies [2,3]. In a study from Shafir et al., a shift from Gram-negative bacteria to Gram-positive bacteria, especially CoNS, was observed in postoperative mediastinitis [6]. Following initial colonization, large amount of extracellular polysaccharide is synthesized, forming a protective biofilm around the colony. Therefore, treatment of infections caused by CoNS frequently necessitates removal of the infected foreign body. Furthermore, the antibiotic treatment requires susceptibility testing, because *S. epidermidis* strains are often resistant to multiple antibiotics. Previous studies have demonstrated that approximately 75% of the CoNS strains were methicillin resistant [2,3]. Coagulase-negative staphylococcal infections generally have a slow onset and are often associated with relatively few clinical signs of mediastinitis compared to mediastinitis caused by other bacteria [5]. However, no difference in mortality was observed between sternal infections caused by CoNS, when compared to *S. aureus*, or Gram-negative pathogens [2]. The other major pathogen in poststernotomy mediastinitis is *S. aureus*, which may have a more aggressive nature and demonstrate more classical signs of infection. This bacteria has been increasingly associated with colonization of the nasal passages of the patient [4]. The incidence of nasal colonization with *S. aureus* in the normal population is reported to range from 10% to 15% and such colonization increases the risk of poststernotomy mediastinitis [7]. Perioperative application of nasal mupirocin eradicates 95–100% of *S. aureus* up to 1 year postoperatively and demonstrates a 67% reduction of infection [8].

3. Risk factors

The pathogenesis of poststernotomy mediastinitis is complex and multifactorial. In previous literature, several

risk factors have been identified and in Table 2 a number of commonly observed risk factors are presented. However, even if a number of specific patient and procedure related risk factors have been delineated, inconsistency still remains regarding the contribution of individual factors to the development of sternal infections. Due to demographic changes in the Western countries the population undergoing cardiac surgery continuously grows older. At time of surgery a substantial part of this cohort also suffers from one or several co-morbidities, often related to their cardiovascular disease. Diagnoses such as obesity, diabetes mellitus, chronic obstructive pulmonary disorder, or heart failure are abundant. Previous literatures have demonstrated that the presence of one of these diagnoses, or risk factors, increase the risk for developing poststernotomy mediastinitis [9]. The mechanism by which obesity leads to this complication is not fully understood, however, perioperative antibiotics may be poorly distributed in adipose tissue, deep skin folds may cause the preoperative skin preparation to be inadequate, and it may also be difficult to diagnose mediastinitis in obese patients during the early phase of the infection [10]. Inadequate sterility may also be one of the mechanisms behind the finding of re-exploration for bleeding as a risk factor [5,11,12]. Diabetes mellitus is also a frequently reported risk factor for mediastinitis [9,13,14]. Elevated blood glucose levels may impair wound healing and the use of continuous, intravenous insulin has been shown to significantly reduce the incidence of deep sternal wound infection in diabetic patients [15]. Heart failure, a high NYHA Class, and a low ejection fraction (<30%) of the left ventricle have also been demonstrated to be associated with mediastinitis [10,12,16]. A reduced ejection fraction is included as a variable in the mediastinitis score proposed by the Northern New England Cardiovascular Disease Study Group and presented in the ACC/AHA Guidelines for CABG surgery [17]. Furthermore, the use of bilateral internal mammary arteries has been reported as a risk factor for sternal infection. The mechanism is probably related to a devascularization of the wound margins after harvesting both mammary arteries, which may delay proper wound healing [12,13]. Extensive coronary atherosclerosis and re-do surgery has also been associated with an increased risk of mediastinitis [10,18]. Again, it is not clear what the true primary cause is, however, this risk factor probably reflects, at least to some extent, a general atherosclerotic condition, which may predispose these patients to poor wound healing in combination with longer procedure times. Prolonged duration of the surgical procedure should intuitively increase the risk of intraoperative contamination and length of surgery has been demonstrated in previous studies to be a risk factor for poststernotomy mediastinitis [10,13].

4. Short- and long-term survival following poststernotomy mediastinitis

With modern hospital hygiene standards and the use of prophylactic antibiotics the overall incidence of poststernotomy mediastinitis is usually reported to be low, usually between 1% and 3% [12,16,19,20]. However, even if this complication is relatively rare it is associated with significant

Table 2
Independent risk factors for mediastinitis observed in previous studies

Risk factor	Odds ratio	Mediastinitis score AHA/ACC (Eagle and Guyton et al.)	Author	Year
Male gender	2.2		Borger et al.	1998
	NA		Demmy et al. [84]	1990
Obesity/severe obesity	6.49	2.5/3.5	Abboud et al. [82]	2004
	1.27		Bitkover et al. [83]	1998
	2.67		The Parisian Mediastinitis Group	1996
	3.59		Milano et al.	1995
	3.8		Nagachinta et al. [85]	1987
	2.65		Ridderstolpe et al.	2001
	3.46		Sjögren et al.	2005
Diabetes mellitus	5.82	1.5	Ridderstolpe et al.	2001
	2.6		Borger et al.	1998
	5.0		Lu et al	2003
	3.24		Sjögren et al.	2005
	2.6		Nagachinta et al.	1987
Smoking	3.27		Abboud et al.	2004
	1.8		Nagachinta et al.	1987
	2.41		Ridderstolpe et al.	2001
COPD	NA	3.5	Demmy et al.	1990
Heart failure/NYHA Class 3–4	3.36		Ridderstolpe et al.	2001
	1.33		Milano et al.	1995
Low LVEF	3.02	2	Sjögren et al.	2005
Renal failure	6.93	2.5	Sjögren et al.	2005
Peripheral vascular disease	3.7		Lu et al.	2003
	2.11		Ridderstolpe et al.	2001
Coronary disease/coronary surgery	2.67		The Parisian Mediastinitis Group	1996
	6.85		Sjögren et al.	2005
	3.2		Muñoz et al. [86]	1997
Use of BIMA	3.2		Borger at al.	1998
	4.23		Ridderstolpe et al.	2001
Length of surgery	1.0		Milano et al.	1995
Re-do surgery	2.2		Milano et al.	1995
Re-exploration	9.2		Muñoz et al.	1997
	NA		Ottino et al.	1987
	3.3		The Parisian Mediastinitis Group	1996
Transfusion	NA		Ottino et al.	1987
Prolonged mechanical ventilation	1.04		Lu et al.	2003
	1.004		Ridderstolpe et al.	2001
Prolonged use of inotropic drugs	2.37		The Parisian Mediastinitis Group	1996
	3.5		Muñoz et al.	1997
ICU stay > 2 days	4.5		Abboud et al.	2002

NA: not available; COPD: chronic obstructive pulmonary disease; NYHA: New York Heart Class; LVEF: left ventricular ejection fraction; BIMA: bilateral internal mammary artery; ICU: intensive care unit.

mortality, reported to vary between 10% and 35% [5,9,12,13,21]. Deep sternal wound infection does not only lead to high mortality, but the morbidity in surviving patients is also significant [5]. Furthermore, poststernotomy mediastinitis is associated with a prolonged length of hospital stay [13], an increased cost of care [3,16] and significant impairment in long-term survival [10,13,19,22,23].

5. Conventional treatment in poststernotomy mediastinitis

During the development of modern cardiac surgery a number of wound-healing strategies have been established

for the treatment of poststernotomy mediastinitis. Conventional forms of treatment usually involve surgical revision with open dressings or closed irrigation, or reconstruction with vascularized soft tissue flaps such as omentum or pectoral muscle. These wound-healing techniques may be used as a single-line therapy or in combination with other procedures. At the present time, there is little consensus regarding the appropriate surgical approach to mediastinitis following open-heart surgery.

Poststernotomy mediastinitis was initially treated with surgical revision, with or without multiple open dressing changes, followed by sternal re-wiring or secondary healing. However, a high mortality rate up to 45% has been reported following this strategy [24]. One major disadvantage of open

dressings is thoracic instability, which requires mechanical ventilation. Prolonged immobilization may increase the risk of additional complications such as pneumonia, thrombosis and muscular weakening. Another devastating complication when leaving the sternum open is right ventricular laceration, which is associated with high mortality rates [25–27]. An important advance in mediastinitis treatment was made in 1963 with continuous irrigation in combination with drainage and a closed sternum [28]. Bryant et al. achieved further development with antibiotic irrigation [29]. Surgical revision with re-wiring or closed irrigation offers an expeditious procedure with the advantage of a closed wound and a stable sternum, but several studies have reported unsatisfactorily high rates of failure [25,30,31] and mortality [5]. The explanation to these failures may be an increased bacterial resistance and a relative devascularization of the left chest wall after harvesting the left mammary artery, which is nowadays a routine procedure. Therapeutic failure during poststernotomy mediastinitis treatment with recurrent infections is known to aggravate an already difficult situation, and results in very high early mortality [32]. Another alternative technique for healing wound infections, without continuous irrigation, is closed drainage with Redon catheters. This technique is based on surgical debridement in combination with multiple small catheters connected to bottle with a strong (–700 mmHg) negative pressure inside. The successful use of this therapy in poststernotomy mediastinitis was demonstrated in 1989 by Durandy et al. [33] and has been confirmed in more recent studies [34,35].

A commonly accepted wound-healing approach is primary, or delayed, wound closure with vascularized soft tissue flaps. The use of pectoral muscle flaps was initially described in 1980 [36]. Recent studies have reported varying results with pectoral muscle flaps in poststernotomy mediastinitis [37–39]. Other authors have advocated the technique employing omentum flaps first described by Lee et al. for closure of mediastinal defects [24,40,41], or reversed rectus abdominis muscle flaps [30,42]. Reconstruction with soft tissue flaps has a relatively low mortality rate according to some reports [37,41]. However, there are disadvantages, including additional surgical trauma and late flap-related morbidity such as pain, weakness and hernias [41,43,44]. Furthermore, there are several reports demonstrating poor long-term outcome with these techniques as mentioned previously.

6. Development of vacuum-assisted closure therapy

Vacuum-assisted closure (VAC) is a recent technical innovation in wound care with a growing number of applications. This wound-healing system was developed in the U.S. by Argenta and Morykwas in the mid 1990s [45]. During approximately the same time period a similar system using sub-atmospheric pressure in wound healing was evaluated by Fleischmann in Germany [46]. VAC therapy has been available in North America since 1995 and the system was introduced in Europe in 1997. This wound-healing technique is based on the application of local negative pressure to a wound. This is achieved by placing polyurethane

foam with an open pore structure of 400–600 μm in the wound. One end of a non-collapsible tube is then connected to the foam and the other end is connected to a vacuum-source in a closed system via connected to a fluid container. The foam and the entire wound are covered with an adhesive drape thus ensuring an air-tight system. Finally, a predetermined, continuous or intermittent, negative pressure is applied to the wound. The foam dressing collapses on application of the negative pressure and transmits an even distribution of pressure across the wound [47].

7. Mechanisms of vacuum-assisted closure therapy

During the application of negative pressure to a sternal wound several advantageous features of conventional surgical treatment are combined. Vacuum-assisted closure allows open drainage that continuously removes exudate with simultaneous stabilization of the chest and isolation of the wound. By maintaining a moist environment, this therapy stimulates granulation-tissue formation [48] in combination with an increased blood flow in the adjacent tissue [49,50]. Furthermore, VAC therapy approximates the wound edges and provides a mass filling effect with a low degree of surgical trauma, without establishing a new wound (e.g. abdominal wound in omental flaps). Finally, due to sternal stabilization and wound isolation, patients can be mobilized early and receive physiotherapy in order to minimize further complications. Morykwas et al. also demonstrated a decrease in bacterial count during VAC therapy [48]. However, there have been conflicting results regarding the bacterial burden during VAC treatment [51,52]. Whether the arbitrary critical level of 1×10^5 CPU/g still is valid or not when using VAC in an infected wound is debatable [51].

The exact mechanism by which VAC exerts its wound-healing mechanism is not fully understood. It has been postulated that the increased perfusion results from an increased hydrostatic pressure gradient along an arteriole, thus directly drawing blood along the vessel [53]. An alternative explanation is the reduction of tissue edema by removing osmotically active molecules and mediators, thus preventing capillary compromise [48,54]. The tissue oedema in the wound margins creates a situation similar to a localized compartment syndrome [55]. A complimentary mechanism aiding the healing process would be the removal of inhibitory molecules. Bennet al. reported a decrease in inflammatory phase cytokines and a simultaneous increase in stimulating cytokines [56]. However, so far no quantitative studies have been reported supporting the theory of a reduction in interstitial fluid. Vacuum-assisted closure also approximates the wound margins, and therefore, exerts a mechanical force on the surrounding tissue. Tissue expansion is known to stimulate angiogenesis and increase the mitotic activity in skin [57,58]. It has also been hypothesized that mechanical stress (transduction) caused by VAC stimulates wound healing through 'reversed' tissue expansion [59,60]. Mechanical stimulation is known to stimulate several physiological mechanisms, such as ion transport, release of second messengers, alterations in gene expression, and increased protein synthesis. Shear stress has been demonstrated to increase cell division by

up-regulation of the second messengers causing phosphorylation [61].

8. Vacuum-assisted closure therapy in poststernotomy mediastinitis

Several recent studies have reported the clinical benefits of VAC treatment in poststernotomy mediastinitis. In these studies, the VAC technique has been successful, either as a single-line therapy, or as a procedure for providing optimal conditions for second-line treatment with delayed flap closure [33,62–71]. Also successful data including high-risk patients and patients with infectious complications after assist device implantation or transplantation have been reported [72–75].

Our research group has previously presented a concept for VAC as a single-line therapy followed by sternal re-wiring without the use of additional tissue flaps [76]. We have used a structured approach with VAC, but without additional tissue flaps, for poststernotomy mediastinitis at our department in Lund since 1999 with promising results (Fig. 1). The survival data in Fig. 1 is based on 65 patients (mean age 67.9 ± 9.9) undergoing VAC therapy for poststernotomy mediastinitis between January 1999 and March 2004. The number of days from primary cardiac surgery procedure until mediastinitis diagnosis was 16.4 ± 10.6 . The patients had VAC therapy (continuous suction) for 11.9 ± 9.0 days and the patients underwent a mean of 3.4 ± 2.2 procedures including initial debridement, VAC-changes, and final re-wire. All procedures were made in the operating theatre. All patients (100%) were re-wired without the use of tissue flaps and the total length of stay at our hospital was 24.6 ± 16.4 days. Our group has recently demonstrated an improved 90-days survival and a lower failure rate when comparing VAC therapy to conventional treatments [77]. We have also compared the long-term survival between patients undergoing CABG without mediastinitis to VAC-treated patients suffering from poststernotomy mediastinitis after CABG [78]. No difference in late survival was demonstrated, even after adjusting the Kaplan–Meier plot for differences in EuroSCORE. These results are contradictory to previously published reports regarding long-term survival after mediastinitis [13,25,26]. The reason for

negative survival effects when using conventional treatments for poststernotomy mediastinitis is unclear. However, a severe systemic infection with repeated septic episodes due to ineffective treatments might cause irreversible effects in vulnerable organs such as the heart, kidneys, and bypass grafts [13]. We believe the successful long-term results found in our study may be due to the use of vacuum-assisted closure therapy as a wound-healing strategy. The effective combination of several wound-healing principles provided by VAC seems to result in minimal sequelae from the poststernotomy mediastinitis.

Catarino et al. performed an early, small retrospective study in order to compare VAC to continuous irrigation [33]. They reported a significantly greater number of treatment failures with continuous irrigation than with VAC. Domkowski et al. conducted an observational study using VAC therapy as a single-line treatment or as a bridge to tissue flap surgery, which showed very low (3.7%) early mortality [62]. However, they included both superficial and deep sternal wound infections. Fleck et al. reported lower rates of recurrent mediastinitis with VAC followed by delayed primary closure or pectoral muscle flaps compared to revision and primary closure [79]. Doss et al. reported good results when comparing VAC to conventional wound management, but they used VAC as a bridge to tissue flap treatment in 20% of their patients [65]. They also reported a shorter length of hospital stay and treatment duration following VAC therapy [65]. The shorter hospital stay with VAC treatment was verified in a recent study by Fuchs et al., however, some of their patients were discharged with an open sternum relying on secondary wound healing without re-wiring [80]. Moidl et al. recently reported lower costs when using VAC therapy for poststernotomy mediastinitis [81]. In our retrospective comparison all patients underwent sternal re-wiring without the use of soft tissue flaps and no significant difference in length of stay or treatment duration was observed between VAC therapy and conventional treatment (Ref. [77]). One explanation may be that differences in health care systems affect the routines regarding discharge to the patients' home or transfer of patients to the referring hospital. Furthermore, treatment duration varies considerably between different surgical techniques, and therefore, results between different studies should be interpreted with caution.

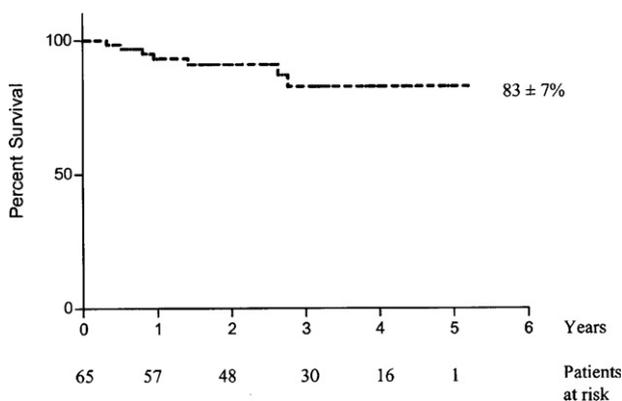


Fig. 1. Overall survival after vacuum-assisted closure therapy for poststernotomy mediastinitis following cardiac surgery.

9. The Lund University Hospital mediastinitis algorithm

One important factor in successful outcome following poststernotomy mediastinitis is early referral to a surgical center with a structured approach and a well-known experience in wound-healing management. Delayed diagnosis and therapy will most likely lead a deteriorating patient resulting in increased morbidity and mortality. In our region all our referring hospitals discuss, and eventually, send patients with diagnosed, or suspected, mediastinitis back to our cardiothoracic center. We have adopted VAC therapy for poststernotomy mediastinitis in a structured fashion and here we describe the wound-healing concept used at Lund University Hospital (Fig. 2). While applying this algorithm all patients with mediastinitis have received vacuum-assisted closure therapy followed by successful delayed primary

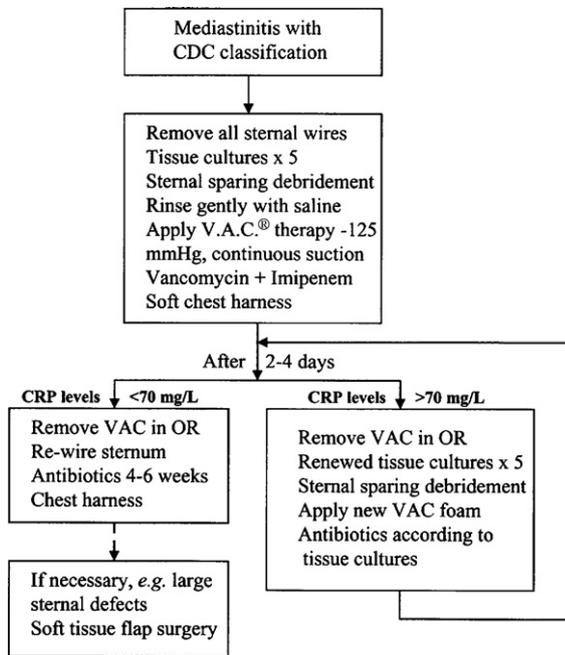


Fig. 2. Algorithm for vacuum-assisted closure therapy in poststernotomy mediastinitis.

closure, including sternal re-wire, without the use of vascularized tissue flaps.

10. Conclusion and future perspectives

Poststernotomy mediastinitis is still one of the most feared complications after cardiac surgery and during the last 50 years several conventional treatments have been developed in order to manage these devastating infections. During the last decade a new treatment modality using topical negative pressure, or VAC, in the wound has evolved. Vacuum-assisted closure combines several advantageous features of conventional surgical treatments in addition to the unique features of topical negative pressure. During the last years VAC therapy has been adopted for use in poststernotomy mediastinitis and the use of this wound-healing therapy is currently spreading throughout the cardiothoracic community. Knowledge and experience regarding VAC in poststernotomy mediastinitis is constantly expanding and an increasing number of institutions report improved results when using this wound-healing strategy. However, several of these reports are relatively small studies and of an observational character including limited populations with short follow-up times. Therefore, although the use of VAC treatment in poststernotomy mediastinitis has presented promising clinical results, there is still need for further knowledge, e.g. large multi-center studies and randomized trials. Another way to ensure large enough amount of data is to initiate a European VAC Therapy Database Register. However, without a structured clinical approach at the individual center one may fail to secure all the clinical benefits of VAC therapy. Therefore, we present an algorithm for use in VAC-treated poststernotomy mediastinitis after cardiac surgery.

References

- [1] Mangram AJ, Horan TC, Pearson ML, Silver LC, Jarvis WR. Guideline for Prevention of Surgical Site Infection, 1999. Centers for Disease Control and Prevention (CDC) Hospital Infection Control Practices Advisory Committee. *Am J Infect Control* 1999;27:97–132.
- [2] Gardlund B, Bitkover CY, Vaage J. Postoperative mediastinitis in cardiac surgery—microbiology and pathogenesis. *Eur J Cardiothorac Surg* 2002;21:825–30.
- [3] Tegnell A, Aren C, Ohman L. Coagulase-negative staphylococci and sternal infections after cardiac operation. *Ann Thorac Surg* 2000;69:1104–9.
- [4] Baskett RJ, MacDougall CE, Ross DB. Is mediastinitis a preventable complication? A 10-year review. *Ann Thorac Surg* 1999;67:462–5.
- [5] Grossi EA, Culliford AT, Krieger KH, Kloth D, Press R, Baumann FG, Spencer FC. A survey of 77 major infectious complications of median sternotomy: a review of 7,949 consecutive operative procedures. *Ann Thorac Surg* 1985;40:214–23.
- [6] Shafir R, Weiss J, Gur E, Herman O, Siegman-Igra Y, Sorkine P, Rudick V. Sternal wound infection: our experience with 200 cases. *J Cardiovasc Surg (Torino)* 1994;35(6 Suppl. 1):103–4.
- [7] Jakob HG, Borneff-Lipp M, Bach A, von PS, Windeler J, Sonntag H, Hagl S. The endogenous pathway is a major route for deep sternal wound infection. *Eur J Cardiothorac Surg* 2000;17:154–60.
- [8] Cimochoowski GE, Harostock MD, Brown R, Bernardi M, Alonzo N, Coyle K. Intranasal mupirocin reduces sternal wound infection after open heart surgery in diabetics and nondiabetics. *Ann Thorac Surg* 2001;71:1572–8.
- [9] Borger MA, Rao V, Weisel RD, Ivanov J, Cohen G, Scully HE, David TE. Deep sternal wound infection: risk factors and outcomes. *Ann Thorac Surg* 1998;65:1050–6.
- [10] Milano CA, Kesler K, Archibald N, Sexton DJ, Jones RH. Mediastinitis after coronary artery bypass graft surgery. Risk factors and long-term survival. *Circulation* 1995;92:2245–51.
- [11] Ottino G, De PR, Pansini S, Rocca G, Tallone MV, Comoglio C, Costa P, Orzan F, Morea M. Major sternal wound infection after open-heart surgery: a multivariate analysis of risk factors in 2,579 consecutive operative procedures. *Ann Thorac Surg* 1987;44:173–9.
- [12] Lu JC, Grayson AD, Jha P, Srinivasan AK, Fabri BM. Risk factors for sternal wound infection and mid-term survival following coronary artery bypass surgery. *Eur J Cardiothorac Surg* 2003;23:943–9.
- [13] Loop FD, Lytle BW, Cosgrove DM, Mahfood S, McHenry MC, Goormastic M, Stewart RW, Golding LA, Taylor PCJ. Maxwell Chamberlain memorial paper. Sternal wound complications after isolated coronary artery bypass grafting: early and late mortality, morbidity, and cost of care. *Ann Thorac Surg* 1990;49:179–86.
- [14] Crabtree TD, Codd JE, Fraser VJ, Bailey MS, Olsen MA, Damiano Jr RJ. Multivariate analysis of risk factors for deep and superficial sternal infection after coronary artery bypass grafting at a tertiary care medical center. *Semin Thorac Cardiovasc Surg* 2004;16:53–61.
- [15] Furnary AP, Zerr KJ, Grunkemeier GL, Starr A. Continuous intravenous insulin infusion reduces the incidence of deep sternal wound infection in diabetic patients after cardiac surgical procedures. *Ann Thorac Surg* 1999;67:352–60.
- [16] Ridderstolpe L, Gill H, Granfeldt H, Ahlfeldt H, Rutberg H. Superficial and deep sternal wound complications: incidence, risk factors and mortality. *Eur J Cardiothorac Surg* 2001;20:1168–75.
- [17] Eagle KA, Guyton RA, Davidoff R, Ewy GA, Fonger J, Gardner TJ, Gott JP, Herrmann HC, Marlow RA, Nugent WC, O'Connor GT, Orszulak TA, Rieselbach RE, Winters WL, Yusuf S, Gibbons RJ, Alpert JS, Eagle KA, Garson Jr A, Gregoratos G, Russell RO, Smith Jr SC. ACC/AHA Guidelines for Coronary Artery Bypass Graft Surgery: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1991 Guidelines for Coronary Artery Bypass Graft Surgery). *American College of Cardiology/American Heart Association. J Am Coll Cardiol* 1999;34:1262–347.
- [18] The Parisian Mediastinitis Study Group. Risk factors for deep sternal wound infection after sternotomy: a prospective, multicenter study. *J Thorac Cardiovasc Surg* 1996;111:1200–7.
- [19] Stahle E, Tammelin A, Bergstrom R, Hambreus A, Nystrom SO, Hansson HE. Sternal wound complications—incidence, microbiology and risk factors. *Eur J Cardiothorac Surg* 1997;11:1146–53.
- [20] Douville EC, Asaph JW, Dworkin RJ, Handy Jr JR, Canepa CS, Grunkemeier GL, Wu Y. Sternal preservation: a better way to treat most sternal wound complications after cardiac surgery. *Ann Thorac Surg* 2005;78:1659–64.

- [21] Krabatsch T, Hetzer R. Poststernotomy mediastinitis treated by transposition of the greater omentum. *J Card Surg* 1995;10:637–43.
- [22] Braxton JH, Marrin CA, McGrath PD, Morton JR, Norotsky M, Charlesworth DC, Lahey SJ, Clough R, Ross CS, Olmstead EM, O'Connor GT. 10-year follow-up of patients with and without mediastinitis. *Semin Thorac Cardiovasc Surg* 2004;16:70–6.
- [23] Toumpoulis IK, Anagnostopoulos CE, Derose Jr JJ, Swistel DG. The impact of deep sternal wound infection on long-term survival after coronary artery bypass grafting. *Chest* 2005;127:464–71.
- [24] Sarr MG, Gott VL, Townsend TR. Mediastinal infection after cardiac surgery. *Ann Thorac Surg* 1984;38:415–23.
- [25] Cartier R, Diaz OS, Carrier M, Leclerc Y, Castonguay Y, Leung TK. Right ventricular rupture. A complication of postoperative mediastinitis. *J Thorac Cardiovasc Surg* 1993;106:1036–9.
- [26] Piwnica A, Abdelmeguid I, Mesnildrey P, Laborde F, Menasche P, Romano M, Guedon C. Rupture of the right ventricular free wall. An unusual complication of mediastinitis after cardiac surgery. *Eur J Cardiothorac Surg* 1988;2:172–5.
- [27] Arbulu A, Gursel E, Camero LG, Asfaw I, Stephenson LW. Spontaneous right ventricular rupture after sternal dehiscence: a preventable complication? *Eur J Cardiothorac Surg* 1996;10:110–5.
- [28] Shumacker Jr HB, Mandelbaum I. Continuous antibiotic irrigation in the treatment of infection. *Arch Surg* 1963;86:384–7.
- [29] Bryant LR, Spencer FC, Trinkle JK. Treatment of median sternotomy infection by mediastinal irrigation with an antibiotic solution. *Ann Surg* 1969;169:914–20.
- [30] Catarino PA, Chamberlain MH, Wright NC, Black E, Campbell K, Robson D, Pillai RG. High-pressure suction drainage via a polyurethane foam in the management of poststernotomy mediastinitis. *Ann Thorac Surg* 2000;70:1891–5.
- [31] Rand RP, Cochran RP, Aziz S, Hofer BO, Allen MD, Verrier ED, Kunzelman KS. Prospective trial of catheter irrigation and muscle flaps for sternal wound infection. *Ann Thorac Surg* 1998;65:1046–9.
- [32] DeFeo M, De Santo LS, Romano G, Renzulli A, Della CA, Utili R, Cotrufo M. Treatment of recurrent staphylococcal mediastinitis: still a controversial issue. *Ann Thorac Surg* 2003;75:538–42.
- [33] Durandy Y, Batisse A, Bourel P, Dibie A, Lemoine G, Lecompte Y. Mediastinal infection after cardiac operation. A simple closed technique. *J Thorac Cardiovasc Surg* 1989;97:282–5.
- [34] Calvat S, Trouillet J-L, Nataf P, Vuagnat A, Chastre J, Gibert C. Closed drainage using Redon catheters for local treatment of poststernotomy mediastinitis. *Ann Thorac Surg* 1996;61:195–201.
- [35] Kirsch M, Mekontso-Dessap A, Houël R, Giroud E, Hillion M-L, Loisanse DY. Closed drainage using Redon catheters for poststernotomy mediastinitis: results and risk factors for adverse outcome. *Ann Thorac Surg* 2001;71:1580–6.
- [36] Jurkiewicz MJ, Bostwick III J, Hester TR, Bishop JB, Craver J. Infected median sternotomy wound. Successful treatment by muscle flaps. *Ann Surg* 1980;191:738–44.
- [37] Jones G, Jurkiewicz MJ, Bostwick J, Wood R, Bried JT, Culbertson J, Howell R, Eaves F, Carlson G, Nahai F. Management of the infected median sternotomy wound with muscle flaps. The Emory 20-year experience. *Ann Surg* 1997;225:766–76.
- [38] Wettstein R, Erni D, Berdat P, Rothenfluh D, Banic A. Radical sternectomy and primary musculocutaneous flap reconstruction to control sternal osteitis. *J Thorac Cardiovasc Surg* 2002;123:1185–90.
- [39] Klesius AA, Dzemali O, Simon A, Kleine P, bdel-Rahman U, Herzog C, Wimmer-Greinecker G, Moritz A. Successful treatment of deep sternal infections following open heart surgery by bilateral pectoralis major flaps. *Eur J Cardiothorac Surg* 2004;25:218–23.
- [40] Lee Jr AB, Schimert G, Shaktin S, Seigel JH. Total excision of the sternum and thoracic pedicle transposition of the greater omentum; useful strategies in managing severe mediastinal infection following open heart surgery. *Surgery* 1976;80:433–6.
- [41] Milano CA, Georgiade G, Muhlbaier LH, Smith PK, Wolfe WG. Comparison of omental and pectoralis flaps for poststernotomy mediastinitis. *Ann Thorac Surg* 1999;67:377–80.
- [42] Shibata T, Hattori K, Hirai H, Fujii H, Aoyama T, Seuhiro S. Rectus abdominis myocutaneous flap after unsuccessful delayed sternal closure. *Ann Thorac Surg* 2003;76:956–8.
- [43] Ringelman PR, Vander Kolk CA, Cameron D, Baumgartner WA, Manson PN. Long-term results of flap reconstruction in median sternotomy wound infections. *Plast Reconstr Surg* 1994;93:1208–14.
- [44] Yuen JC, Zhou AT, Serafin D, Georgiade GS. Long-term sequelae following median sternotomy wound infection and flap reconstruction. *Ann Plast Surg* 1995;35:585–9.
- [45] Argenta LC, Morykwas MJ. Vacuum-assisted closure: a new method for wound control and treatment: clinical experience. *Ann Plast Surg* 1997;38:563–76.
- [46] Fleischmann W, Strecker W, Bombelli M, Kinzl L. Vacuum sealing as treatment of soft tissue damage in open fractures. *Unfallchirurg* 1993;96:488–92.
- [47] Thomas GPL, Banwell PE. Topical negative-pressure therapy in wound management. In: Teot L, Banwell PE, Ziegler UE, editors. *Surgery in wounds*. Heidelberg: Springer-Verlag; 2004. p. 109–24.
- [48] Morykwas MJ, Argenta LC, Shelton-Brown EI, McGuirt W. Vacuum-assisted closure: a new method for wound control and treatment: animal studies and basic foundation. *Ann Plast Surg* 1997;38:553–62.
- [49] Wackenfors A, Sjögren J, Gustafsson R, Algotsson L, Ingemansson R, Malmjö M. Effects of vacuum-assisted closure therapy on inguinal wound edge microvascular blood flow. *Wound Repair Regen* 2004;12:600–6.
- [50] Wackenfors A, Gustafsson R, Sjögren J, Algotsson L, Ingemansson R, Malmjö M. Blood flow responses in the peristernal thoracic wall during vacuum-assisted closure therapy. *Ann Thorac Surg* 2005;79:1724–30.
- [51] Moues CM, Vos MC, van den Bemd GJ, Stijnen T, Hovius SE. Bacterial load in relation to vacuum-assisted closure wound therapy: a prospective randomized trial. *Wound Repair Regen* 2004;12:11–7.
- [52] Weed T, Ratliff C, Drake DB. Quantifying bacterial bioburden during negative pressure wound therapy: does the wound VAC enhance bacterial clearance? *Ann Plast Surg* 2004;52:276–9.
- [53] Banwell PE. Topical negative pressure therapy in wound care. *J Wound Care* 1999;8:79–84.
- [54] Morykwas MJ, David LR, Schneider AM, Whang C, Jennings DA, Canty C, Parker D, White WL, Argenta LC. Use of subatmospheric pressure to prevent progression of partial-thickness burns in a swine model. *J Burn Care Rehabil* 1999;20:15–21.
- [55] Morykwas MJ. Sub-atmospheric pressure therapy: research evidence. In: Banwell PE, Teot L, editors. *Topical negative pressure (TNP) therapy*. Faringdon: TXP Communications; 2004. p. 39–44.
- [56] Bennett DC, Komorowska-Timek E, Garbriel A, Gupta S. Characterizing the changing gene statement of chronic wounds treated with sub-atmospheric pressure therapy. *Plastic Surgery Research Council 47th Annual Meeting*, 2002, 4-17-0020.
- [57] Olenius M, Dalsgaard CJ, Wickman M. Mitotic activity in expanded human skin. *Plast Reconstr Surg* 1993;91:213–6.
- [58] Cherry GW, Austad E, Pasyk K, McClatchey K, Rohrich RJ. Increased survival and vascularity of random-pattern skin flaps elevated in controlled, expanded skin. *Plast Reconstr Surg* 1983;72:680–7.
- [59] Banwell PE, Teot L. Topical negative pressure (TNP): the evolution of a novel wound therapy. *J Wound Care* 2003;12:22–8.
- [60] Saxena V, Hwang CW, Huang S, Eichbaum Q, Ingber D, Orgill DP. Vacuum-assisted closure: microdeformations of wounds and cell proliferation. *Plast Reconstr Surg* 2004;114:1086–96.
- [61] Chen KD, Li YS, Kim M, Li S, Yuan S, Chien S, Shyy JY. Mechanotransduction in response to shear stress. Roles of receptor tyrosine kinases, integrins, and Shc. *J Biol Chem* 1999;274:18393–400.
- [62] Domkowski PW, Smith ML, Gonyon Jr DL, Drye C, Wooten MK, Levin LS, Wolfe WG. Evaluation of vacuum-assisted closure in the treatment of poststernotomy mediastinitis. *J Thorac Cardiovasc Surg* 2003;126:386–90.
- [63] Luckraz H, Murphy F, Bryant S, Charman SC, Ritchie AJ. Vacuum-assisted closure as a treatment modality for infections after cardiac surgery. *J Thorac Cardiovasc Surg* 2003;125:301–5.
- [64] Fleck TM, Fleck M, Moidl R, Czerny M, Koller R, Giovanoli P, Hiesmayer MJ, Zimpfer D, Wolner E, Grabenwoger M. The vacuum-assisted closure system for the treatment of deep sternal wound infections after cardiac surgery. *Ann Thorac Surg* 2002;74:1596–600.
- [65] Doss M, Martens S, Wood JP, Wolff JD, Baier C, Moritz A. Vacuum-assisted suction drainage versus conventional treatment in the management of poststernotomy osteomyelitis. *Eur J Cardiothorac Surg* 2002;22:934–8.
- [66] Song DH, Wu LC, Lohman RF, Gottlieb LJ, Franczyk M. Vacuum assisted closure for the treatment of sternal wounds: the bridge between debridement and definitive closure. *Plast Reconstr Surg* 2003;111:92–7.
- [67] Tang AT, Ohri SK, Haw MP. Novel application of vacuum assisted closure technique to the treatment of sternotomy wound infection. *Eur J Cardiothorac Surg* 2000;17:482–4.

- [68] Obdeijn MC, de Lange MY, Lichtendahl DH, de Boer WJ. Vacuum-assisted closure in the treatment of poststernotomy mediastinitis. *Ann Thorac Surg* 1999;68:2358–60.
- [69] Hersh RE, Jack JM, Dahman MI, Morgan RF, Drake DB. The vacuum-assisted closure device as a bridge to sternal wound closure. *Ann Plast Surg* 2001;46:250–4.
- [70] Immer FF, Durrer M, Muhlemann KS, Erni D, Gahl B, Carrel TP. Deep sternal wound infection after cardiac surgery: modality of treatment and outcome. *Ann Thorac Surg* 2005;80:957–61.
- [71] Agarwal JP, Ogilvie M, Wu LC, Lohman RF, Gottlieb LJ, Franczyk M, Song DH. Vacuum-assisted closure for sternal wounds: a first-line therapeutic management approach. *Plast Reconstr Surg* 2005;116:1035–40.
- [72] Cowan KN, Teague L, Sue SC, Mahoney JL. Vacuum-assisted wound closure of deep sternal infections in high-risk patients after cardiac surgery. *Ann Thorac Surg* 2005;80:2205–12.
- [73] Routledge T, Saeb-Parsy K, Murphy F, Ritchie AJ. The use of vacuum-assisted closure in the treatment of post-transplant wound infections: a case series. *J Heart Lung Transplant* 2005;24:1444.
- [74] Yuh DD, Albaugh M, Ullrich S, Conte JV. Treatment of ventricular assist device driveline infection with vacuum-assisted closure system. *Ann Thorac Surg* 2005;80:1493–5.
- [75] Sjögren J, Gustafsson R, Dobre M, Koul B, Ingemansson R, Algotsson L. Vacuum-assisted closure therapy in mediastinitis after heart transplantation. *J Heart Lung Transplant* 2004;23:506–7.
- [76] Gustafsson RI, Sjögren J, Ingemansson R. Deep sternal wound infection: a sternal-sparing technique with vacuum-assisted closure therapy. *Ann Thorac Surg* 2003;76:2048–53.
- [77] Sjögren J, Gustafsson R, Nilsson J, Malmsjö M, Ingemansson R. Clinical outcome after poststernotomy mediastinitis: vacuum-assisted closure versus conventional treatment. *Ann Thorac Surg* 2005;79:2049–55.
- [78] Sjögren J, Nilsson J, Gustafsson R, Malmsjö M, Ingemansson R. The impact of vacuum-assisted closure on long-term survival after post-sternotomy mediastinitis. *Ann Thorac Surg* 2005;80:1270–5.
- [79] Fleck TM, Koller R, Giovanoli P, Moidl R, Czerny M, Fleck M, Wolner E, Grabenwoger M. Primary or delayed closure for the treatment of post-sternotomy wound infections? *Ann Plast Surg* 2004;52:310–4.
- [80] Fuchs U, Zittermann A, Stuetzgen B, Groening A, Minami K, Koerfer R. Clinical outcome of patients with deep sternal wound infection managed by vacuum-assisted closure compared to conventional therapy with open packing: a retrospective analysis. *Ann Thorac Surg* 2005;79:526–31.
- [81] Moidl R, Fleck T, Giovanoli P, Grabenwoger M, Wolner E. Cost effectiveness of V.A. C. (R) therapy after poststernotomy mediastinitis. *Zentralbl Chir* 2006;131(S 1):189–90.
- [82] Abboud CS, Wey SB, Baltar VT. Risk factors for mediastinitis after cardiac surgery. *Ann Thorac Surg* 2004;77:676–83.
- [83] Bitkover CY, Gardlund B. Mediastinitis after cardiovascular operations: a case-control study of risk factors. *Ann Thorac Surg* 1998;65:36–40.
- [84] Demmy TL, Park SB, Liebler GA, Burkholder JA, Maher TD, Benckart DH, Magovern Jr GJ, Magovern Sr GJ. Recent experience with major sternal wound complications. *Ann Thorac Surg* 1990;49:458–62.
- [85] Nagachinta T, Stephens M, Reitz B, Polk BF. Risk factors for surgical-wound infection following cardiac surgery. *J Infect Dis* 1987;156:967–73.
- [86] Munoz P, Menasalvas A, Bernaldo de Quiros JC, Desco M, Vallejo JL, Bouza E. Postsurgical mediastinitis: a case-control study. *Clin Infect Dis* 1997;25:1060–4.

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