



Introduction

Mediastinitis after cardiac surgery is defined as the infection of organs and spaces of the mediastinum, which may occur in 0.4% to 2.4% of cases. When this complication occurs, it increases the length of hospital stay and hospital costs, besides being possibly lethal. Mortality can vary between 10% and 47%. (*Cely et al, 2004*)

According to the criteria proposed by the **Center for Disease Control** (*Garner JS et al., 1988*), mediastinitis can be diagnosed by:

1. A positive bacterial culture from the mediastinal space.
2. Evidence of mediastinitis during surgery or in histology.
3. One of the following criteria: fever ($>38^{\circ}\text{C}$), chest pain, or sternal instability.
4. At least one of the following:
 - Purulent discharge from the mediastinal area.
 - Organisms cultured from blood.
 - Organisms cultured from mediastinal discharge.
 - Mediastinal widening in radiology.

(*Michael et al, 1998*)

Several studies have examined risk factors for development of mediastinitis which were classified as preoperative, intraoperative and postoperative:



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Preoperative risk factors include: body mass index >30, diabetes mellitus, smoking, hypertension, dyslipidemia, chronic obstructive pulmonary disease, previous use of corticoid or antibiotics, chronic or acute renal insufficiency, acute myocardial infarction up to 1 month before the surgery, low ejection fraction, previous sternotomy and others.

Intraoperative risk factors include: emergency surgery, long duration of the surgery, long perfusion time, long aorta cross clamping time, use of internal mammary arteries (unilateral or bilateral), intraoperative blood transfusion and others.

Postoperative risk factors include: reoperation (mainly for purposes of hemostasis), use of intra-aortic balloon, increased length of intensive care unit (ICU) stay, tracheostomy, increased duration of mechanical ventilation, use of inotropic drugs, and presence of infection at another site. (*Cely et al.*, 2004)

In spite of this, the key factor in preventing sternal dehiscence and sternal wound infection is a stable sternal approximation. Careful attention to hemostasis and meticulous surgical technique remain the mainstays of prevention and must include precise sternal alignment and stable closure. (*Schimmer et al.* 2008)

The treatment of mediastinitis after cardiac surgery has evolved over the past 35 years. The classic surgical therapy



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consisted of surgical debridement followed by open wound drainage. However, this technique was associated with high failure and mortality rates and has progressively been abandoned.

In 1963, Shumaker and Coworkers described a technique of wound debridement, primary sternal closure, and continuous mediastinal irrigation (*Shumaker et al. 1963*).

In 1989, Durandy and colleagues proposed a simple primary closed drainage technique using Redon catheters. The basis of this technique is, after meticulous wound debridement, to drain all infected areas with small catheters connected to bottles inside of which a strong negative pressure (-700 mm Hg) is created (Redon drainage device) (*Durandy et al., J Thorac Cardiovasc Surg 1989*). Studies have reported significantly reduced failure and 30-day mortality rates in patients treated with this technique in comparison to patients treated with the closed continuous irrigation technique. (*Calvat S et al., Ann Thorac Surg 1996*)

Evidence of antibiotic or iodine toxicity and poor results in earlier studies have stimulated the development of several plastic surgical procedures as alternatives to closed mediastinal irrigation (*Milano et al, 1999*). Thus, primary or delayed mediastinal closure using muscle flaps or the greater omentum have gained wide acceptance for the treatment of mediastinitis after cardiac surgery (*Gottlieb et al., Cardiac Surg 1996*).



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The vacuum-assisted closure (VAC) technique is a relatively new modality in wound-healing management. Local application of negative pressure to a wound results in improved tissue blood flow and increased granulation tissue formation (*Sjögren et al., 2005*).



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Aim of Work

This work aims at evaluation of the different recent modalities in the management of post-operative Mediastinitis.



Etiology and Risk Factors

Theoretical Mechanisms For The Development Of

Sternal Wound Infection And Dehiscence:

Several mechanisms have been proposed to explain the development of sternal wound dehiscence and infection. It has been suggested that localised ischaemic osteomyelitis is a primary event. Sternal wires become loose in the affected region and sternal instability follows, with subsequent dehiscence of the overlying skin incision. The osteomyelitic bone and open wound are an ideal focus for the development of infection. Other theories suggest that inadequate sternal fixation and the resulting instability lead to skin dehiscence as a primary event. The open wound becomes secondarily infected and infected material drains backwards into the pericardium and mediastinum (*Mills C and Philip Bryson., 2006*).

Alternatively, inadequate surgical drainage has been offered as a primary event. Insufficient drainage of the mediastinum in the postoperative period results in collection of blood and serous fluid in the mediastinum that forms an excellent culture medium for bacteria. Once a focus of infection is formed, infected material can then track forwards and discharge through the skin wound. Although inadequate sternal fixation or mediastinal drainage probably does increase risk, there are many instances of wound dehiscence where neither of



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these factors can be shown to be present (*Mills C and Philip Bryson., 2006*).

The key factor in preventing sternal dehiscence and infection is a stable sternal approximation. All sternal closure techniques claim to maximize sternal stability, but it is difficult to differentiate between the merits of various techniques (*Christoph Schimmer et al., 2008*).

The question of whether sternal instability or infection is the primary underlying problem can not be answered with certainty. Circlage fixation under normal physiologic loads can prove to be inadequate and can lead to separation. Bacterial contamination in the face of sternal separation and instability can then progress to deep sterna wound infections. On the other hand, the initial instability derives from the basic mechanism of an osteomyelitis. This is the ground upon which the feared infection develops (*Song DH et al., 2004*).

Although several methods of sternal closure have been described in literature, for example, additional steel band at the third intercostal space (*Riess FC., et al 2004*), double crisscross (*Bottio T. et al., 2003*), and double wires (*Kiessling AH. et al., 2005*). **Khasati and colleagues** compared on the basis of 111 papers two different sternal closure techniques (simple wire and figure-of-eight technique). The authors concluded that the figure-of-eight technique revealed no significant advantage over the simple wire technique (*Khasati N. et al., 2004*).



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Nowadays, many other authors described the benefits of rigid plate fixations. These authors conclude that rigid plate fixation showed a significant decrease in the incidence of postoperative mediastinitis when compared with a similar population of patients whose sternums were closed with wire (*Raman J., 2006*).

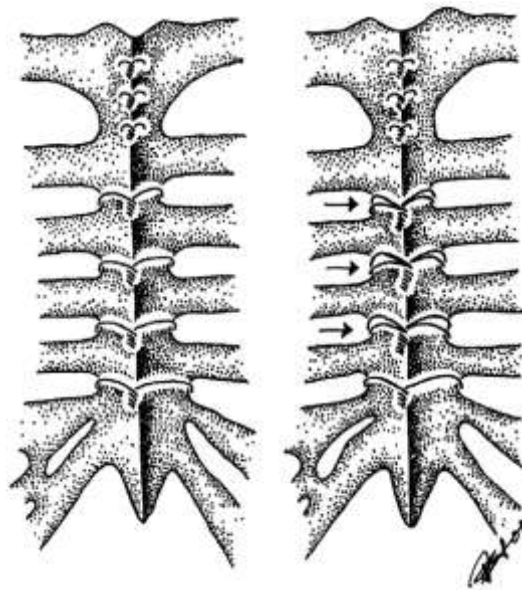
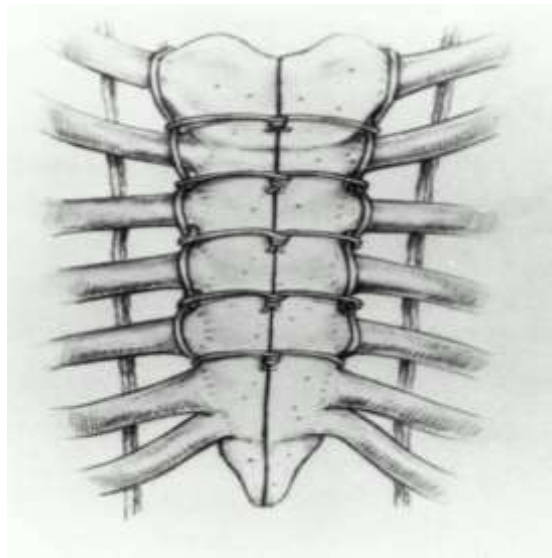


Fig. 1: Schematic drawing illustrating the two wiring methods. (Left) single peristernal with four single wire loops; (right) double peristernal with three double loops (arrows). Note a fourth, single wire loop at the bottom of the closure. Each manubrium is closed with three single wires (*Losanoff et al., 2007*).

The technique described by **Robicsek and coworkers in 1977** has several advantages: it stabilizes the sternum if it is fragile or broken; if subsequent instability develops, it prevents the wires cutting through the bone; it changes the site of

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pressure and provides wider support, especially if the sternum has been mishandled with the sternal retractor. This technique changes the point of contact from metal to bone to contact of metal to metal. The disadvantage of this technique is that it produces a constrictive weave that can disrupt the collateral blood supply of the sternum, and effective approximation of the top and bottom of a gaping sternum cannot be obtained.



Diagrammatic presentation of peristernal weave (Robicsek 1977).

In conclusion, the key factor in preventing sternal dehiscence and sternal wound infection is a stable sternal approximation. Careful attention to hemostasis and meticulous surgical technique remain the mainstays of prevention and must include precise sternal alignment and stable closure (*Christoph Schimmer et al., 2008*).



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The microbial etiology of sternal wound infections varies and includes Gram-negative and Gram-positive bacteria as well as fungi. However, the most common causative pathogens involved in sternal wound infections are *Staphylococcus Epidermidis* (Coagulase Negative Staph (CoNS)) and *Staphylococcus Aureus*, both from the normal flora of the skin (*Gardlund et al., 2002*).

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 surgery procedures when closing the sternotomy and CoNS has emerged as the most important pathogen in post-sternotomy mediastinitis, responsible for 43% to 64% of all cases in recent studies (*Johan Sjögren 2006*).



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

Bacterial strains	VAC therapy		Conventional treatment	
	<i>n</i>	% ^a	<i>n</i>	% ^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.

(Johan Sjögren 2006).

Mediastinitis caused by coagulase negative staphylococci was more often associated with sternal instability than other bacterial etiology. Sternal instability itself is strongly associated with mediastinitis and it is likely that mediastinitis often develop from a minor skin or subcutaneous infection in the sternal wound and that the infection may spread inwards to the mediastinal space if sternal dehiscence breaks the mechanical



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barrier between the pre-sternal tissue and the mediastinum. A pre-sternal wound infection with coagulase negative staphylococci would otherwise be expected to be benign and self-contained and not to pose a clinical problem.

In a study from **Shafir et al.**, a shift from Gram-negative bacteria to Gram-positive bacteria, especially CoNS, was observed in postoperative mediastinitis (**Shafir R, et al., 1994**). 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 (**Gardlund B. et al., 2002**). 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 (**Grossi EA. Et al., 1985**). However, no difference in mortality was observed between sternal infections caused by CoNS, when compared to *S. Aureus*, or Gram-negative pathogens (**Gardlund B. et al., 2002**). 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 (**Baskett RJ. et**



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al. 1999). 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 (*Jakob HG. et al., 2000*). Perioperative application of nasal mupirocin eradicates 95—100% of *S. aureus* up to 1 year postoperatively and demonstrates a 67% reduction of infection⁸ (*Johan Sjögren 2006*).

The third major group of bacteria identified in postoperative mediastinitis is aerobic gram negative rods. The typical pathogenic mechanisms could be entirely different from that of the staphylococci. Perioperative contamination of the sternal wound with gram-negatives appears unlikely considering the known bacterial flora in the chest region and that the isolation of gram-negative bacteria from air in the operating theater or from the wound after a long operation is rare (*Bitkover et al., 2000*).

The use of a vein graft harvested from a contaminated donor site in the groin or leg in coronary by-pass surgery could be a way of introducing gram-negative bacteria in the sternal wound. However, coronary artery bypass grafting did not carry an increased risk for gram-negative mediastinitis either in this or in previously published studies (*Sta•hle E. et al., 1997*), or in bypass surgery with vein grafts compared to other bypass conduits (*Loop FD. et al., 1990*). Instead, gram-negative mediastinitis has been shown to be associated with concomitant infections, mainly pneumonia, with gram-negative bacteria in



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the immediate postoperative period and is typically associated with a more complicated postoperative course with prolonged mechanical ventilation, which increases the risk for nosocomial infections with gram-negatives (*Wouters R. et al., 1994*). Furthermore, outbreaks of mediastinitis caused by specific gram-negatives like *Pseudomonas* or *Serratia* may suggest that nosocomial spread of gram-negative infections in the postoperative period is important for the development of gram-negative mediastinitis (*Gardlund et al., 2002*).

In conclusion to that three basically different types of postoperative mediastinitis can be distinguished:

- (1) Mediastinitis associated with obesity and sternal dehiscence, sometimes also with chronic obstructive pulmonary disease, and often caused by coagulase negative staphylococci;
- (2) Mediastinitis following perioperative contamination of the mediastinal space often caused by *S. aureus*, and
- (3) Mediastinitis caused by spread from concomitant infections in other sites than the mediastinum in the postoperative period, often caused by gram negative rods.

(Gardlund et al., 2002).

This classification is of course not strict, but may be helpful in understanding the discrepancies and sometimes contradictory results from studies on risk factors in postoperative mediastinitis. The classification of post-operative



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mediastinitis into the three major groups with partly different pathogenic mechanisms may also be useful in designing infection control programs. If gram-negative mediastinitis is a major problem in the institution, strict enforcing of hygienic barrier routines to reduce the spread of nosocomial infections in the postoperative ward seems most important. If *S. aureus* mediastinitis were a major problem, investigations aimed at identifying and eradicating sources of bacteria in individuals in the operating theater and to make sure the antibiotic prophylaxis is administered in a timely fashion would seem more appropriate. In addition, routines for hair removal, skin preparation and also the hygienic and ventilation standard of the operating rooms should be scrutinized. In mediastinitis caused by coagulase negative staphylococci, appropriate preventive measures are not as evident (*Gardlund et al., 2002*).

Several authors have described various host risk factors that might contribute to the development of deep surgical site infections. These have been described extensively in the literature and include obesity, diabetes mellitus, the use of internal thoracic artery grafts (especially bilateral), advanced age, female sex, chronic lung disease, active smoking, prolonged mechanical ventilation, the use of steroids, and preoperative hospital stay longer than 5 days (*El. Oakley and Wright 1996*).

Obesity, particularly in diabetic women, is considered a major risk factor (*Zacharias et al., 1996*). It was found that