

Sternal wounds



THE V.A.C.[®]
VACUUM ASSISTED CLOSURE™

KCI
Wolfgang Imendörffer
Marketing Manager CEE
22 September, 2009

Der Stellenwert der V.A.C.[®]-Therapie bei Sternumosteomyelitis

O. Frerichs
S. Brüner
H. Fansa

Value of V.A.C.[®]-Therapy in the Treatment of Sternal Infections

Zusammenfassung

Die Sternumosteomyelitis ist eine gefürchtete Komplikation nach Sternotomie und geht mit einer hohen Mortalität einher. Die Infektbeherrschung ist ein Kernpunkt der erfolgreichen Therapie. Eine Thoraxinstabilität nach Sternektomie kann die Komplikationsrate deutlich erhöhen. Es erfolgte bei insgesamt 16 Patienten mit instabilem, infiziertem Sternum zunächst das radikale Debridement mit Entfernung des Sternums und der betroffenen Rippen. Bei 6 Patienten (Gruppe A) erfolgte eine primäre Defektdeckung durch einen gestielten Muskellappen. Bei 10 Patienten (Gruppe B) erfolgte zunächst die Anlage eines V.A.C.[®]-Systems und die Defektdeckung wurde dann bei sauberen Wundverhältnissen und nach Verbesserung des Allgemeinzustandes durchgeführt. Bis auf kleinere Wundheilungsstörungen kam es bei allen Patienten zu einer problemlosen Einheilung der Lappenplastiken. Fünf Patienten der Gruppe A hatten schwere Komplikationen mit Thoraxinstabilität sowie pulmonaler und kardialer Dekompensation. Diese Patienten mussten langfristig beatmet werden, zwei von ihnen verstarben aufgrund eines Multiorganversagens. In Gruppe B traten keine Todesfälle oder schwere Komplikationen auf. Alle Patienten waren innerhalb des Beobachtungszeitraums rezidivfrei. Die V.A.C.[®]-Therapie nach radikaler Sternektomie trägt entscheidend dazu bei, die Wundpflege zu erleichtern und den Infektherd zu sanieren. Gleichzeitig stabilisiert sie den Thorax und führt so zu einer Senkung der pulmonalen Komplikationen. Durch dieses Verfahren kann ein mehrzeitiges Vorgehen erfolgen und die erhebliche Belastung einer einzigen großen Operation bei multimorbiden Patienten reduziert werden.

Abstract

Osteomyelitis of the sternum is a dreaded complication after sternotomy and is related to high mortality. Control of infection by radical debridement is the key to successful treatment. Instability of the thoracic cage can lead to a high complication rate. 16 Patients with an infected and unstable sternum underwent radical debridement with resection of the sternum and adjacent ribs. 6 Patients (group A) received an immediate defect coverage with a pedicled muscle flap. 10 Patients (group B) were treated with a vacuum-assisted closure (V.A.C.[®])-therapy until stabilization of their general condition and underwent defect coverage in a second operation. Healing of the flaps was uneventful in all cases despite minor problems. 5 patients of group A had severe complications with pulmonary or cardiac failure and thoracic instability which lead to prolonged periods of mechanical ventilation. 2 patients of this group died due to multi organ failure. All patients of group B survived and there were no major complications. All of the patients were free of recurrence from their osteomyelitis during follow-up. V.A.C.[®]-therapy after radical resection of sternum osteomyelitis proved to be an effective measure to bridge time while optimizing the status of the patient and its wound. With this approach we believe to have lowered the rate of major complications in this multimorbid patient group by reducing the burden of one large operation and by improving thoracic stability.

Institutsangaben

Klinik für Plastische Wiederherstellungs- und Ästhetische Chirurgie, Handchirurgie, Klinikum Bielefeld

Korrespondenzadresse

Dr. Onno Frerichs · Klinik für Plastische, Wiederherstellungs- und Ästhetische Chirurgie · Handchirurgie · Klinikum Bielefeld · Teutoburger Str. 50 · 33604 Bielefeld · Tel.: 05 21/5 81 39 51 · E-mail: onno.frerichs@sk-bielefeld.de

Bibliografie

Zentralbl Chir 2006; 131: S120-S123 © J. A. Barth Verlag in Georg Thieme Verlag KG
DOI 10.1055/s-2006-921459
ISSN 0044-409X

Einleitung

Die Sternumosteomyelitis als Folge der Sternotomie bei Bypass-Operationen ist eine schwerwiegende Komplikation mit hoher Mortalität und langem Verlauf [5]. Traditionell erfolgte ein Debridement der Wunde und eine Spülbehandlung, gegebenenfalls mit Rezerklage. Alternativ erfolgte bei massiven Infekten auch eine Sternumresektion mit offener Wundbehandlung. Dieses Regime war häufig langwierig, und es bestand eine hohe Rezidivrate. Mit der Einführung von gestielten Muskellappenplastiken konnten auch ausgedehnte Defekte erfolgreich und sicher behandelt werden. Problematisch waren hier bei einzeitigem Vorgehen die möglichen Rezidive sowie die Belastung durch die ausgedehnte Operation bei den häufig multimorbiden Patienten. Durch ein zweizeitiges Vorgehen konnte die Rate an Wundkomplikationen verringert werden [6], es mussten im Intervall aber aufwändige und belastende Verbandwechsel erfolgen. Nach Sternumresektion musste zudem aufgrund der Thoraxinstabilität teilweise eine verlängerte Beatmung mit den damit verbundenen Risiken durchgeführt werden.

Seit der Einführung der V.A.C.[®]-Therapie [8] können auch große Defektwunden mit relativ wenig Aufwand und sicher versorgt werden. Durch die kontinuierliche Sogbehandlung kommt es zu einer deutlichen Säuberung der Wunden. Die regelmäßigen Verbandwechsel können bei den meisten Patienten ohne Narkose erfolgen und erlauben eine verlässliche Beurteilung der Wundverhältnisse. Die V.A.C.[®]-Therapie scheint daher für die Behandlung der Sternumosteomyelitis prädestiniert zu sein.

Methodik

Seit 2003 wurden in unserer Klinik insgesamt 16 Patienten aufgrund einer Sternumosteomyelitis nach Bypass-OP behandelt. Es handelte sich um 5 Frauen und 11 Männer mit einem durchschnittlichen Alter von 67 Jahren. Bei allen Patienten lag primär ein instabiles Sternum (Abb. 1) mit Mediastinitis vor (Typ III–IV [3]). Bei 13 Patienten erfolgte bereits auswärts ein Debridement mit Teilentfernung des Sternums und eine V.A.C.[®]-Therapie. Alle Patienten waren in einem deutlich reduzierten Allgemeinzustand mit ASA III–IV. Die häufigste Begleiterkrankung, neben der immer vorliegenden koronaren Herzkrankheit und Arteriosklerose, war ein Diabetes mellitus (n = 12).

Bei allen Patienten erfolgte zunächst ein radikales Debridement mit Entfernung aller avitalen beziehungsweise entzündlichen Thoraxwandanteile (Abb. 2). Hierbei resultierte in 14 Fällen eine totale Sternumresektion, bei zwei Patienten konnte das Manubrium sterni erhalten werden. Bei den ersten 6 Patienten dieser Serie (Gruppe A) erfolgte nach der totalen Sternumresektion die sofortige Defektdeckung durch einen gestielten myokutanen Latissimus-dorsi-Muskellappen. Bei den weiteren 10 Patienten (Gruppe B) wurde nach dem Debridement eine V.A.C.[®]-Therapie

angewandt (Abb. 3), bei der alle 2–5 Tage ein Verbandwechsel, ggf. unter kurzfristiger Analgosedierung, erfolgte. In der Regel wurde der Schaum direkt in die Wunde eingelegt. Nur bei direkter Pleuraauflage oder über freiliegenden Gefäßen (A. thoracica interna bzw. Bypass) erfolgte die Einlage einer Silikongaze zur Verhinderung einer Schädigung der Strukturen. Der Sog wurde kontinuierlich mit 125 mm Hg appliziert. Bei diesen Patienten erfolgte zwischen Sternumresektion und Defektdeckung eine intensive Aufbauphase zur Verbesserung des Allgemeinzustandes. Hier wurde insbesondere auf eine möglichst optimale internistische Betreuung sowie vollständige Mobilisierung mit Krankengymnastik und Atemtherapie geachtet. Die Defektdeckung erfolgte dann bei infektfreien Wundverhältnissen und verbessertem Allgemeinzustand. Zur Defektdeckung wurde die schnellste, sicherste und einfachste zur Verfügung stehende Lappenplastik gewählt.

Ergebnisse

Die Patienten wurden im Durchschnitt 2,4-mal (inkl. Tracheotomie) operiert. Der stationäre Aufenthalt dauerte durchschnittlich 61 Tage (16–127). Zur Defektdeckung wurden 17 gestielte Muskellappen (3×Pectoralis, 2×VRAM, 12×Latissimus dorsi) und ein freier mikrovaskulärer M. latissimus dorsi benutzt. Die Lappenplastiken waren in allen Fällen komplikationsarm. Es traten lediglich kleine Wundheilungsstörungen an den Lappenrändern auf, die durch konservative Therapie und/oder Spalthauttransplantation abheilten. An 5 der 13 Latissimus- und bei jeweils einer Pektoralis- und VRAM-Entnahmestelle bestanden punktionwürdige Serome. Bei 3 Latissimus-Entnahmestellen kam es zu Wundheilungsstörungen, die alle unter konservativer Therapie abheilten. Ein Patient hatte ein postoperatives Pleuraleck, vermutlich durch Umlagerung im Bett, und musste revidiert werden.

Schwere Komplikationen mit verlängerter postoperativer Beatmung nach der Defektdeckung traten bei fünf der sechs Patienten aus Gruppe A auf. Bei diesen Patienten konnte direkt postoperativ eine deutliche Thoraxinstabilität mit erschwelter Atemtätigkeit beobachtet werden. Vier dieser Patienten mussten daher langzeitbeatmet werden und benötigten ein Tracheostoma. Die hierbei auftretenden Komplikationen waren pulmonale und kardiale Dekompensationen mit konsekutivem Multiorganversagen, Dialysepflicht, Pneumonie und Sepsis. Zwei dieser Patienten verstarben während dieser Zeit in Folge des Multiorganversagens, ein Patient wurde erfolgreich reanimiert. Die zweizeitig operierten Patienten hatten nach der Defektdeckung eine klinisch deutlich bessere Thoraxstabilität und mussten demzufolge kürzer postoperativ nachbeatmet werden. In dieser Patientengruppe gab es auch deutlich weniger schwere Komplikationen und keinen Todesfall.



Abb. 1 Präoperativer Status.

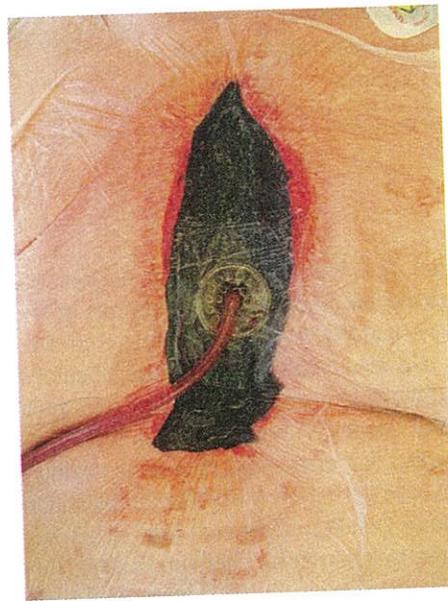


Abb. 3 V.A.C.[®]-Verband in situ.



Abb. 2 Zustand nach Sternumresektion.



Abb. 4 Zustand ein Jahr nach gestieltem Latissimus-dorsi-Lappen.

Alle überlebenden Patienten wurden nach Hause oder in eine Anschlussheilbehandlung entlassen und sind innerhalb des Beobachtungszeitraums (4 Monate–3 Jahre) rezidivfrei (Abb. 4) und ohne pulmonale Einschränkungen im Vergleich zum präoperativen Zustand. Ein Patient verstarb ein Jahr nach der Entlassung an einem Herzinfarkt. Alle Patienten waren mit dem Operationsergebnis sehr zufrieden, ein Patient klagte jedoch über Narbenschmerzen im Bereich der Latissimus-Entnahmestelle.

Diskussion

Die Defektdeckung nach Sternumresektionen ist heutzutage technisch in den meisten Fällen problemarm möglich. Es stehen verschiedene Muskellappenplastiken zur Verfügung, mit denen ein sicherer Defektverschluss nach radikalem Debridement durchgeführt werden kann. Die Anwendung von Fremdmate-

rialien und die damit erhöhte Komplikationsrate kann in den meisten Fällen vermieden werden [4]. Neben dem Latissimus-dorsi-Lappen [7] haben sich der Pektoralis-Lappen [2] und der vertikale Rectus-abdominis-Lappen (VRAM) [9] bewährt. Nichtsdestotrotz ist die Behandlung von Patienten mit Sternumosteomyelitis aufgrund der regelhaft bestehenden Begleiterkrankungen mit einer relativ hohen Morbidität und Mortalität von bis zu 20% verbunden [5].

Durch die Einführung der V.A.C.[®]-Therapie 1997 [8] wurde die Behandlung von ausgedehnten Komplexwunden entscheidend erleichtert. Unter dieser Therapie kommt es im Vergleich zu herkömmlichen Wundpflegeverfahren zu einer Reduzierung der Keimbeseidelung, Verbesserung der Durchblutung und Beschleunigung der Granulation sowie zu einer deutlichen Reduzierung der Belastung für das Pflegepersonal und den Patienten. Die V.A.C.[®]-Therapie wird daher als ideales Verbandssystem bei der Behandlung von Sternumwunden angesehen [1].

In der hier vorliegenden Untersuchung wurde festgestellt, dass die V.A.C.[®]-Therapie nach dem radikalen Debridement entschei-

end zur Reduzierung von Komplikationen in Folge der Thoraxinstabilität geführt hat. Klinisch konnte beobachtet werden, dass die kontinuierliche Sogbehandlung zu einer deutlichen Stabilisierung des Thorax führte. Während der Verbandwechsel kam es, vor allem in der frühen postoperativen Phase, zu einer deutlich stärkeren Exkursion der Wundränder und einer Instabilität des Thorax. Im Verlauf der zunehmenden Granulation des Wundbetts kam es dann durch die damit verbundene Narbenbildung zu einer weiteren Stabilisierung des Thorax. In allen Fällen mit zweizeitigem Vorgehen bestand schließlich nach der Defektdeckung eine ausreichende Stabilität des Brustkorbs. Zusätzlich hat die V.A.C.[®]-Therapie deutlich zur Erleichterung der Pflege für Arzt und Patienten beigetragen. Unter der Therapie bestanden bei allen Patienten durchgehend saubere Wundverhältnisse und eine gute Granulationstendenz der Weichteile. Es kam in keinem Fall zu Komplikationen durch die V.A.C.[®]-Therapie.

Literatur

- ¹ Agarwal JP, Ogilvie M, Wu LC, Lohmann RF, Gottlieb LJ, Franczyk M, Song DH. Vacuum-assisted closure for sternal wounds: A first-line therapeutic management approach. *Plast Reconstr Surg* 2005; 15: 1035
- ² Arnold PG, Pairolero PC. Use of pectoralis major muscle flap to repair defects of anterior chest wall. *Plast Reconstr Surg* 1979; 63: 205
- ³ Fansa H, Handstein S, Schneider W. Treatment of infected median sternotomy wounds with a myocutaneous latissimus dorsi flap. *Scand Cardiovasc J* 1998; 32: 33
- ⁴ Frerichs O, Fansa H, Schneider W. Die Therapie der Thoraxwandosteomyelitis. *Chirurg* 2001; 72: 1020–1025
- ⁵ Gummert JF, Barten MJ, Hans C, Kluge M, Doll N, Walther T, Hentschel B, Schmitt DV, Mohr FW, Diegeler A. Mediastinitis and cardiac surgery: An updated risk factor analysis in 10373 consecutive patients. *J Thorac Cardiovasc Surg* 2002; 50: 87
- ⁶ Lidsey JT. A retrospective analysis of 48 infected sternal wound closures: Delayed closure decreases wound complications. *Plast Reconstr Surg* 2002; 109: 1882
- ⁷ McCraw JB, Penix JO, Baker JW. Repair of major defects of the chest wall and spine with the latissimus dorsi myocutaneous flap. *Plast Reconstr Surg* 1978; 62: 197
- ⁸ Morykwas MJ, Argenta LC, Shelton-Brown EI, McGiurt W. Vacuum assisted closure: A new method for wound control and treatment. Animal studies and basic foundation. *Ann Plast Surg* 1997; 38: 553
- ⁹ Reed WP, Spence RJ. Vertical rectus abdominis musculocutaneous flap for chest wall reconstruction after irradiation. *South Med J* 1987; 80: 287

6. Sitzung: Varia

36

Vacuum Assisted Closure (V.A.C.®) mit GranuFoam® Silver™ verbessert die Wundheilung bei komplizierten sternalen InfektionenI. Kutschka¹, T. Bisdas¹, S. Fischer¹, C. Hagl¹, N. Kaladj¹, P. Zardo¹, T. Peters², A. Haverich¹¹Herz-, Thorax-, Transplantations- und Gefäßchirurgie, Medizinische Hochschule Hannover, Deutschland²Klinik für Plastische, Hand- und Wiederherstellungschirurgie, Medizinische Hochschule Hannover, Deutschland

Zusammenfassung: Silberbeschichtete Polyurethanschwämme sollen die Effizienz der V.A.C.® Therapy von Wunden mit resistenter Keimbeseidung verbessern. Wir haben dieses Verfahren bei 3 Patienten mit schwerer sternaler Wundinfektion eingesetzt. Im Gegensatz zu unserer bisherigen Erfahrung mit ORSA/MRSA/MRSE oder sonstigen Problemkeimen besiedelten Wunden, konnten wir eine erfolgreiche lokale Dekontamination erzielen. Die resistenten Keime konnten mikrobiologisch nicht mehr nachgewiesen werden. Bei allen Patienten konnte ein sekundärer Wundverschluss mit bilateraler Pektoralisplastik erzielt werden. Die mittlere Behandlungsdauer mit V.A.C.® betrug 23 ± 7 Tage.

Schlüsselwörter: V.A.C.®, Silber-Schwamm, ORSA/MRSA, Sternuminfektion

Vacuum Assisted Closure (V.A.C.®) with GranuFoam® Silver™ improves healing of complicated sternal wounds

Summary: Recently Vacuum Assisted Closure (V.A.C.®) Therapy using silver impregnated foams has been introduced for wound infections with resistant bacteria. We used this technique in 3 patients with severe sternal wound infection. In contrast to our previous experience with oxacillin/-methicillin resistant bacteria (ORSA/MRSA) we observed a successful decontamination of all sternal wounds. Microbiological testing became negative.

All patients underwent secondary wound closure using bilateral pectoralis muscle flaps. Mean duration of V.A.C.® was 23 ± 7 days.

Keywords: V.A.C.®, silver impregnated sponge, ORSA/MRSA, sternal wound infection

Einleitung: Sternal Wundinfektionen nach Herzoperationen führen zu hoher Morbidität und Mortalität [1]. Primäre Therapie für diese Komplikation ist in der Regel ein chirurgisches Debridement mit Einlage von Spüldrainagen und direkter Reverdrahtung. Dieses Vorgehen ist jedoch mit einer hohen Rezidivrate behaftet. Dies trifft vor allem auf Infektionen mit ausgeprägter Knochennekrose, tief greifende mediastinaler Infektionen und Wunden mit multiresistenten Keimen zu [2, 3]. Deshalb wird zunehmend ein zweistufiges Verfahren zur Behandlung von Sternuminfektionen favorisiert. Die Anwendung von Vacuum Assisted Closure (V.A.C.®) gilt hier als optimales Verfahren zur primär „offen“ chirurgischen Wundtherapie und zur Vorbereitung für einen sekundären Wundverschluss. Für Wundinfektionen mit resistenten Erregern wurde ein silberbeschichteter Schwamm (V.A.C. GranuFoam Silver®, KCI®, Germany) eingeführt. Die desinfizierende Wirkung von Silberoberflächen ist hinreichend bekannt [4] und kann nun mit der durchblutungsfördernden und sekretentlastenden Wirkung des Vacuums kombiniert werden [5]. Wir haben diese neue Methode in 3 Fällen mit komplizierter sternaler Infektion eingesetzt und beschreiben den Verlauf dieser Patienten.

Methodik: Im Zeitraum zwischen September 2007 und Januar 2008 erhielten 3 Patienten (2 ♂; 1 ♀) mit schwerer sternaler Wundinfektion (ORSA [$n=2$] und Klebsiella oxytoca und E. coli [$n=1$]) eine V.A.C.® Therapy mit Silber-Schwamm. Bei 2 Patienten wurde als initialer Eingriff eine koronare Bypassoperation und bei einem Patient eine Aortenklappenersatz durchgeführt. Das Risi-

Korrespondenz: PD Dr. Ingo Kutschka, Oberarzt, Herz-, Thorax-, Transplantations- und Gefäßchirurgie, Medizinische Hochschule Hannover, Carl-Neuberg-Strasse 1, 30625 Hannover, Deutschland
Fax: ++49-511-532 5404
E-mail: kutschka.ingo@mh-hannover.de

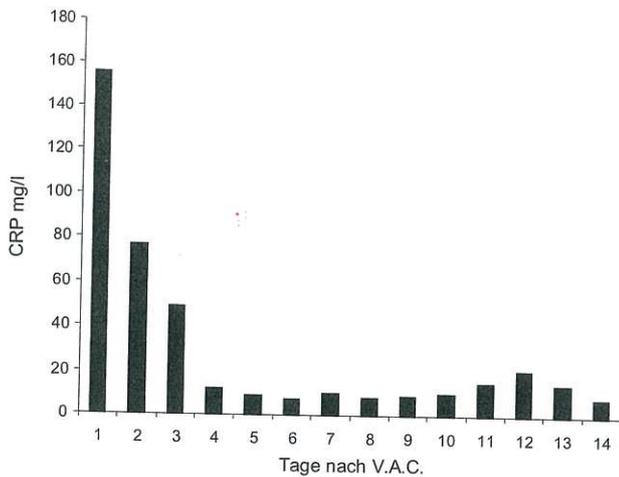


Abb. 1: Serum-CRP Verlauf nach V.A.C.®-Anlage mit Silber-Schwamm (Fall 3)

koprofil bzgl. Wundheilungsstörung war bei allen Patienten deutlich erhöht (s.u.). Der mittlere Body Mass Index (BMI) betrug $37,3 \pm 9 \text{ kg/m}^2$.

Die Erstanlage des V.A.C.®-Verbandes erfolgte im Operationssaal unter Allgemeinanästhesie. Alle weiteren Verbandwechsel wurden in 3-tägigen Intervallen unter Analgetikatherapie auf der Station durchgeführt. Serum-CRP und Blutbild wurde alle 2–3 Tage bestimmt. Vor jeder V.A.C.®-Neuanlage wurde ein Wundabstrich genommen.

Ergebnisse: Die mittlere Dauer der V.A.C.® Therapy mit Silber-Schwamm war 23 ± 7 Tage. Zwei Patienten (Fall 1, 3) konnten direkt nach dem Eingriff extubiert werden. Ein Patient (Fall 2) musste bei Sepsis längerfristig beatmet und tracheotomiert werden. Die systemischen Infektparameter waren bei allen Patienten zügig rückläufig (Abb. 1). Bei allen Patienten verblieb ein grösserer Knochen- bzw. Weichteildefekt, der sekundär durch eine Pectoralisplastik gedeckt wurde.



Abb. 2: Fall 1: (a) Wunde nach 14 Tagen V.A.C.® mit Silberschwamm. (b) Wunde nach Verschluss mit bilateraler Pectoralisplastik, Fall 2: (c) Wunde 10 Tage nach Beginn der Vakuumtherapie bei Reinfektion nach Omentoplastik. (d) Wunde 8 Tage nach bilateraler Pectoralisplastik, Fall 3: (e, f) Wunde 14 Tage nach V.A.C.® Therapy vor Pectoralisplastik

Fallberichte: Patient 1 (67 Jahre): Im August 2007 wurde eine koronare Bypassoperation mit Dor-Plastik bei linksventrikulärem Vorderwandaneurysma durchgeführt. Es bestand folgendes Risikoprofil: insulinpflichtiger Diabetes mellitus Typ II, Alkoholabusus mit Zustand nach toxischer Pankreatitis und akutem Nierenversagen, renale Anämie, Adipositas (BMI 27 kg/m²). Postoperativ kam es zu einem Kammerflimmern mit zweimaliger suffizienter kardiopulmonaler Reanimation. In der Folge entwickelte sich eine sternale Wundinfektion, die am 36. p.op. Tag ein Wunddebridement mit Sternumreverahtung und Einlage von Spüldrainagen erforderlich machte. Nach initial guter Wundheilung kam es zur Ausbildung einer chronischen Infektion mit Fistelbildung. In der Wunde konnte ORSA nachgewiesen werden. Zehn Wochen nach der Sternumreverahtung erfolgte eine erneute Eröffnung der Wunde mit partieller Entfernung der Cerclagen und Anlage eines V.A.C.®-Verbandes. Es folgten Verbandwechsel alle 3 Tage. 29 Tage nach der ersten V.A.C.®-Anlage zeigte sich weiterhin schlechte Wundheilungstendenz. Die Abstriche erbrachten schliesslich eine ORSA-Besiedlung der Wunde. Deshalb wurde die Therapie auf V.A.C.® mit Silber-Schwamm umgestellt. Im Verlauf zeigte sich eine gute Wundheilungstendenz und sterile Wundabstriche, sodass die Wunde nach weiteren 4 Wochen sekundär mit Pektoralisplastik verschlossen werden konnte (Abb. 2a, b).

Keimspektrum und Antibiotika

ORSA

Antibiotika: Clindamycin, umgestellt auf Linezolid

Patient 2 (77 Jahre): Als initiale Operation wurde ein Aortenklappenersatz mit septaler Myektomie durchgeführt. Risikofaktoren waren insulinpflichtiger Diabetes mellitus Typ IIb, Adipositas (BMI 42 kg/m²) und kompensierte Niereninsuffizienz. Am 20. p.op. Tag entwickelte die Patientin eine Wundheilungsstörung mit instabilem Sternum und einer Sternumdehiszenz, sodass eine Wundrevision durchgeführt werden musste. Es zeigte sich eine ausgedehnte Nekrose des Sternums, sodass ein Wunddebridement erfolgte und ein Omentumhochzug zur Deckung des Defektes durchgeführt wurde. Nach Verlegung auf die Intensivstation komplizierte sich der weitere Verlauf durch das Auftreten einer Reinfektion mit Sepsis. Drei Tage nach der Omentumplastik musste die Wunde komplett eröffnet werden. Nach erneutem Debridement bei partieller Omentumnekrose wurde die Anlage eines V.A.C.® mit Silber-Schwamm durchgeführt. Im weiteren Verlauf zeigte sich eine gute Wundheilungstendenz, die Wundabstriche waren steril, sodass die Wunde 24 Tage nach der ersten V.A.C.®-Anlage sekundär durch die Kollegen der plastischen Chirurgie mit grossflächiger Pektoralisplastik verschlossen werden konnte (Abb. 2c, d). Bei Entlassung 77 Tage nach der Herzoperation war die Wunde reizlos.

Keimspektrum und Antibiotika

Klebsiella oxytoca, Escherichia coli.

Antibiotika: Clindamycin und Flucloxacillin

Patient 3 (47 Jahre): Als initialen Eingriff wurde 6/2004 eine koronare Bypassoperation durchgeführt. Risikofakto-

ren waren ein insulinpflichtiger Diabetes mellitus, Adipositas (BMI: 43 kg/m²) und ORSA Besiedlung der Nase. 15 Monate nach dem ersten Eingriff stellte sich der Patient mit instabilem Sternum vor, und es wurde eine Sternumreverahtung durchgeführt. Hierbei kam es zu einer Verletzung des rechten Ventrikels, die eine Übernähung unter HLM-Einsatz erforderlich machte. Der Patient wurde nach 14 Tagen mit unauffälligen Wundverhältnissen entlassen. Nach weiteren 27 Monaten erfolgte die erneute Aufnahme des Patienten aufgrund eines chronischen prästernalen Seroms. Es wurde ein ausgedehntes prästernales Wunddebridement mit Entfernung aller Drahtcerclagen, Einlage von 2 Drainagen und direktem Wundverschluss durchgeführt. Zwei Wochen später zeigte sich eine Wundinfektion, die ein erneutes Eröffnen der Wunde erforderlich machte. Bei ORSA-Besiedlung der Wunde wurde eine V.A.C.® Therapy mit Silber-Schwamm begonnen. Es folgten weitere Verbandwechsel alle 2–3 Tage (Abb. 2e, f). Die Wundabstriche waren nach 14 Tagen steril. Im Verlauf zeigte sich gute Wundheilungstendenz, sodass die Wunde 19 Tage nach der ersten V.A.C.®-Anlage sekundär mit Pektoralisplastik verschlossen werden konnte. Die Wunde stellte sich im Weiteren reizlos dar.

Keimspektrum und Antibiotika

ORSA, Staphylococcus epidermidis, Staphylococcus haemolyticus

Antibiotika: Clindamycin, Flucloxacillin, keine spezielle Therapie gegen ORSA

Diskussion: Etwa 20% der Population sind ständige oder intermittierende Träger von multiresistenten Keimen wie oxacillin-/methicillinresistenten Staphylokokken (ORSA/MRSA). Die Wahrscheinlichkeit der perioperativen Entwicklung einer Sepsis bei einer Besiedlung der Nase mit MRSA ist signifikant erhöht [6]. Auch das Risiko für eine Wundinfektion ist bei MRSA Kolonisierung in der Nase um das 3,8 fache erhöht [7].

Prävalenzuntersuchungen zeigen besonders hohe MRSA-Besiedlungsraten bei Personen mit insulinpflichtigem Diabetes mellitus oder Niereninsuffizienz, Patienten, die häufig auch im herzchirurgischen Krankengut zu finden sind.

Tiefe sternale Wundinfektionen mit mediastinaler Beteiligung, insbesondere bei Nachweis multiresistenter Bakterien, besitzen ein hohes Mortalitätsrisiko von bis zu 30% [8]. In erster Linie muss deshalb die Ausbreitung der Infektion mit Entwicklung einer Mediastinitis verhindert werden [8]. Im Vergleich zu anderen Therapiemöglichkeiten ermöglicht hier die V.A.C.® Therapy eine schnelle Infektkontrolle durch einen kurzen und wenig belastenden Eingriff. Dies umfasst die Entfernung von seröser Wundflüssigkeit [8] sowie die Reduktion von Ödem und bakterieller Besiedlung. Gleichzeitig verhindert sie eine Wunddehydration und fördert die Bildung von Granulationsgewebe [5].

Durch Einsatz der silberbeschichteten Schwämme konnte eine zusätzliche wunddesinfizierende Wirkung erzielt werden. In allen Fällen konnte ein sekundärer operativer Wundverschluss durchgeführt werden. Trotz

der ausgeprägten Risikokonstellationen wurde eine vollständige Wundheilung erzielt.

Zusammenfassend stellt die V.A.C.® Therapy ein effektives Verfahren zur primären Behandlung von Patienten mit komplizierter Sternuminfektion dar. Bei Patienten mit multiresistenten Erregern kann der Einsatz des antimikrobiellen, silberbeschichteten Schwammes die Wundheilung weiter begünstigen.

Literatur

- [1] Kutschka I, Frauendorfer P, Harringer W (2004) Vacuum assisted closure therapy improves early postoperative lung function in patients with large sternal wounds. *Zentralbl Chir* 129: 33–34.
- [2] Domkowski PW, Smith ML, Gonyon DL Jr, Drye C, Wooten MK, Levin LS, Wolfe WG (2003) Evaluation of vacuum-assisted closure in the treatment of poststernotomy mediastinitis. *J Thorac Cardiovasc Surg* 126: 386–390.
- [3] Fleck TM, Fleck M, Moidl R, Czerny M, Koller R, Giovanoli P, Hiesmayer MJ, Zimpfer D, Wolner E, Grabenwoger M (2002) The vacuum-assisted closure system for the treatment of deep sternal wound infections after cardiac surgery. *Ann Thorac Surg* 74: 1596–1600.
- [4] Castellano JJ, Shafii SM, Ko F, Donate G, Wright TE, Mannari RJ, Payne WG, Smith DJ, Robson MC (2007) Comparative evaluation of silver-containing antimicrobial dressings and drugs. *Int Wound J* 4: 114–122.
- [5] Luckraz H, Murphy F, Bryant S, Charman SC, Ritchie A (2003) Vacuum-assisted closure as a treatment modality for infections after cardiac surgery. *J Thorac Cardiovasc Surg* 125: 301–303.
- [6] Boyce JM (1997) Epidemiology and prevention of nosocomial infections. In: Croosley KB, Archer GL (eds) *The Staphylococci in human disease*, Churchill Livingstone, New York, Edinburgh, London, Madrid, Melbourne, San Francisco, Tokyo.
- [7] Pujol M, Peña C, Pallares R, Ariza J, Ayats J, Dominguez MA, Gudiol F (1996) Nosocomial *Staphylococcus aureus* bacteremia among nasal carriers of methicillin-resistant and methicillin-susceptible strains. *Am J Med* 100: 509–516.
- [8] Kutschka I, Dziadzka S, El Essawi A, Flory P, Harringer W (2006) Vacuum assisted closure for sternal wound infections – initial therapy and bridging to reconstructive surgery. *Zentralbl Chir* 131: 129–132.

Therapie thorakaler Problemwunden bei immunsupprimierten Patienten mit GranuFoam Silver® und Vacuum-Assisted Closure®

Therapy of Infected Thoracic Wounds with GranuFoam Silver® and Vacuum-Assisted Closure® in Immunosuppressed Patients

P. Zardo, P. Weishäupl-Karstens, C. Hagl, N. Khaladj, A.R. Simon, S. Fischer, A. Haverich, I. Kutschka
 Medizinische Hochschule Hannover, Herz-, Thorax-, Transplantations- und Gefäßchirurgie

Zusammenfassung

Einleitung

Wundinfektionen nach herz- bzw. thoraxchirurgischen Eingriffen stellen, gerade bei immunsupprimierten Patienten, eine große Herausforderung im klinischen Alltag dar und erfordern den Einsatz innovativer Therapiestrategien.

Patienten und Methoden

Wir präsentieren insgesamt 4 Fälle immunsupprimierter Patienten nach herz- bzw. thoraxchirurgischen Eingriffen, die im postoperativen Verlauf eine tiefe Wundinfektion mit Nachweis von Problemkeimen entwickelten.

Ergebnisse

Unter konsequenter V.A.C.®-Therapie mit Einsatz neuartiger silberbeschichteter Polyurethan Schwämme (GranuFoam Silver®) kam es in den beschriebenen Fällen zu einer rapiden Besserung der Wundverhältnisse mit signifikanter Reduktion der Keimlast und einer Normalisierung der Infektparameter. Nach einer medianen Behandlungszeit von 48 Tagen konnten 3 Wunden sekundär verschlossen werden, die letzte heilte spontan aus.

Schlüsselwörter

V.A.C.®-Therapie, GranuFoam Silver®, Transplantation, Immunsuppression, Wundheilung

Summary

Background

The treatment of severe wound infections after cardiothoracic surgery still poses a major challenge in the clinical setting. Innovative therapeutic strategies are required, especially in immunosuppressed patients.

Patients and Methods

We present 4 cases of immunosuppressed patients who underwent cardio-

thoracic surgery and developed secondary wound infections with multiresistant bacteria.

Results

Implementation of V.A.C.® (Vacuum-Assisted Closure) -therapy with newly developed silver-coated Polyurethane sponges (GranuFoam Silver®) led to rapid wound decontamination and improved wound healing. After a median V.A.C.® treatment of 48 days, we achieved 3 secondary surgical wound closures with subsequent complete wound healing. The remaining wound healed spontaneously.

Keywords

V.A.C.®-Therapy, GranuFoam Silver®, Transplantation, Immunsuppression, Wound healing

Einleitung

Tiefe Wundinfektionen nach herzthoraxchirurgischen Eingriffen verursachen neben erheblicher Morbidität und Mortalität für die betroffenen Patienten (1) auch erhebliche Kosten für die behandelnden Kliniken (2). Gerade multire-

sistente Keime (MRSA/VRE/MRSE) erweisen sich in diesem Kontext als große therapeutische Herausforderung. Da konventionelle Behandlungsstrategien in derartigen Fällen häufig nicht zielführend sind, wenden wir konsequent ein Zweistufenkonzept zur Versorgung komplizierter thorakaler Wunden an. Nach ausgiebigem chirurgischen Débridement führen wir eine offene Wundbehandlung mit V.A.C.®-Therapie, gegebenenfalls unterstützt durch die Verwendung von Silber-beschichteten Polyurethan Schwämmen (GranuFoam Silver®) durch. Erst nach negativem Wundabstrich und vollständiger Wundreinigung wird eine sekundäre Wundnaht durchgeführt (3).

Basierend auf der nachgewiesenen desinfizierenden Wirkung von silberbeschichteten Oberflächen (4) kommt es zu einer Dekontamination der Wunde, wobei die Wundheilung zusätzlich durch die durchblutungsfördernde und sekretentlastende Wirkung der V.A.C.®-Therapie unterstützt wird.

Die Anwendung von GranuFoam Silver® verspricht vor allem Vorteile bei einer Superinfektion mit nosokomialen Problemkeimen sowie bei Infektionen von Patienten mit deutlich reduzierter Immunabwehr. Gerade dieses Patientenkollektiv stellt im klinischen Alltag eine besondere Herausforderung an die Therapiestrategie dar (5, 6). Als Schwerpunktzentrum für thorakale Organtransplantationen sind wir auch mit Wundinfektionen bei immunsupprimierten Patienten konfrontiert. An dieser Stelle berichten wir über unsere Erfahrungen in der Therapie tiefer sternaler und thorakaler Wunden bei diesem Patientenkollektiv.

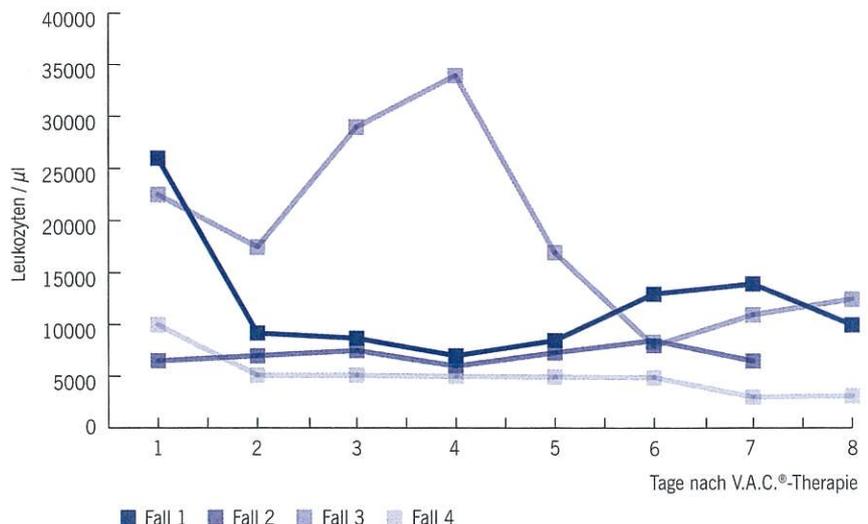


Abb. 1: Verlauf der Infektparameter nach Einleitung der V.A.C.®-Therapie

Material und Methodik

Im Zeitraum zwischen März 2008 und Januar 2009 wurden n=4 Patienten (2 Frauen 2 Männer,) mit tiefer sternaler (n=1) oder thorakaler (n=3) Wundinfektion (n=1: ORSA, n=1: S. aureus, n=1: VRE, n=1: Pseudomonas aeruginosa) und Immunsuppression (n=3: Mycophenolatmofetil, n=3: Tacrolimus, n=1: Cyclosporin A, n=4: Prednison) nach Herz-Lungen-, Doppellungen- (n=2) und Pankreas-Nierentransplantation (n=1) mit GranuFoam Silver®-V.A.C.-Therapie behandelt. Die Wundinfektionen traten nach medianer Sternotomie zur kombinierten Herz-Lungentransplantation, beidseitiger anterolateraler Minithorakotomie zur Doppellungen- und Pankreas-Nierentransplantation (n=2) sowie posterolateraler Minithorakotomie zur Lungenbiopsie bei Verdacht auf pulmonale Filiae auf.

Die initiale V.A.C.-Anlage erfolgte unter sterilen Bedingungen nach ausgiebigem Wunddébridement im Operationssaal in Intubationsnarkose. Alle weiteren Verbandswechsel fanden unter sterilen Kautelen und analgetischer Behandlung in 2-3-tägigen Abständen auf Normalstation statt. Neben regelmäßigen Laborkontrollen einschließlich CRP, Blutbild und Procalcitonin entnahmen wir bei jedem Verbandswechsel einen tiefen Wundabstrich.

Alle 4 Patienten wiesen eine ausgesprochen komplexe Anamnese mit Langzeitimmunsuppression sowie weitere relevante Komorbidität für die Entstehung sekundärer Wundheilungsstörungen (n=1 Diabetes mellitus, n=1 chronische Dialysepflicht und n=2 passagere Dialysepflicht) auf.

Ergebnisse

Nach einer medianen Behandlungsdauer von 48 Tagen (41-57 Tage) konnten 3 Wunden operativ im Sinne einer sekundären Wundnaht verschlossen werden, die letzte heilte sekundär aus. In allen Fällen war eine komplikationslose Erstanlage des V.A.C.® mit Extubation im Operationssaal möglich. Die Infektwerte fielen in der Regel bereits innerhalb der ersten 2 Tage auf unter 50% der jeweiligen Ausgangswerte (Abbildung 1).

Fallberichte

Patientin 1 (49 Jahre)

Die Patientin stellte sich im März 2008 zur Resektion einer singulären pulmonalen Raumforderung rechtsseitig bei Z.n.



Abb. 2



Abb. 4

Mammakarzinom mit Mastektomie links vor. Bei terminaler Niereninsuffizienz mit Dialyse seit 2001 und schwerstem Typ I Diabetes war im Jahr 2004 eine kombinierte Pankreas- und Nierentransplantation erfolgt. Weiterhin war bei schwerer koronarer Herzerkrankung 1996 eine aortokoronarer Bypass-Operation (ACVB) durchgeführt worden. Bei initial unauffälligem Verlauf nach pulmonaler Metastasenentfernung kam es, nach Resektion einer zwischenzeitlich aufgetretenen zerebralen Raumforderung mit postoperativem Cortison-Stoß, am 33. Tag nach dem thorakalen und 14. Tag nach dem zerebralen Eingriff zur Ausbildung einer ausgeprägten Wundheilungsstörung der posterolateralen Thorakotomienarbe. Es zeigte sich eine vollständige Dehiszenz der Wundränder mit Aufbrauch der Interkostalmuskulatur und freiem Blick auf die Pleura (Abbildung 2). Zeichen der Wundheilung schienen völlig zu fehlen. Nachdem die ersten Abstriche lediglich S. aureus zeigten, begannen wir eine Therapie mit konventionellen Polyurethan-Schwämmen. Erst nach Sicherung eines MRSA und Umstellung auf GranuFoam Silver® kam es zu erkennbarer Entstehung von Granulationsgewebe. Bei sukzessiver Besserung der Wundverhältnisse sahen wir aufgrund des erheblich reduzierten Allgemeinzustandes der Patientin von einer sekundären Wundnaht ab. 41 Tage nach Einleitung der Wundtherapie war die Wunde nahezu vollständig verschlossen, eine pleurale Fistel konnte nicht mehr nachgewiesen werden (Abbildung 3).



Abb. 3



Abb. 5

Patient 2 (43 Jahre)

Aufgrund einer chronischen myelomonozytären Leukämie (CMML, Erstdiagnose 2005) sowie einer allogenen Stammzelltransplantation kam es zu einer schweren Graft versus Host Disease (GvHD) mit pulmonaler Beteiligung. Bei invasiver pulmonaler Mykose und Langzeitbeatmung, zuletzt mit zusätzlicher extrakorporaler Lungenunterstützung (extracorporeal lung assist, ECLA) führten wir im Juli 2008 eine minimalinvasive Doppellungen- und Pankreas-Nierentransplantation durch. Initial gestaltete sich der Verlauf am ehesten aufgrund eines Reperfusionssödems der transplantierten Organe protrahiert. Die Beatmungsunterstützung mit Stickstoffmonoxid konnte am 1., die extrakorporale Membranoxygenierung am 3. postoperativen Tag entfernt werden. Nach 9 Tagen wurde bei Verdacht auf Abstoßung eine 3-tägige Steroidstoßtherapie initiiert. Bei subjektivem Wohlbefinden erfolgte am 17. postoperativen Tag eine Verlegung auf Normalstation. Erst 42 Tage nach der Transplantation kam es zu einer ausgeprägten Wundheilungsstörung beider anterolateraler Minithorakotomien (Abbildung 4). Diese wurden bei Nachweis von Vancomycin resistenten Enterokokken (VRE) entsprechend unseres Algorithmus direkt mit GranuFoam Silver® versorgt. Nach 12 weiteren Verbandswechseln konnte am 48. Tag bei sterilen Wundverhältnissen ein sekundärer Verschluss erfolgen (Abbildung 5). Die Wunden waren bei Entlassung aus unserer Klinik reizlos.



Abb. 6

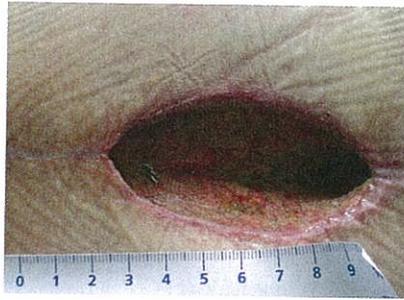


Abb. 7



Abb. 8



Abb. 9

Patientin 3 (44 Jahre):

Zwei Jahre nach der Diagnose einer idiopathischen pulmonalarteriellen Hypertonie (IPAH) erfolgte im Juni 2008 eine kombinierte Herz-Lungentransplantation via medianer Sternotomie. Postoperativ wurde am Folgetag ein Revisionseingriff bei Hämatothorax erforderlich und bei hohem postoperativem Katecholaminbedarf kam es sekundär zur Ausbildung eines anurischen Nierenversagens mit Hämodialysepflichtigkeit. Das Beatmungswearing gestaltete sich aus mehreren Gründen insgesamt protrahiert. Diesbezüglich sind insbesondere eine hämolytische Anämie bei G6PDH-Mangel sowie eine postoperative Pneumonie mit Legionella pneumophila zu erwähnen. Drei Monate nach der Transplantation kam es zu einer manifester prästernalen Wundinfektion (Abbildung 6). Die initiale Behandlung bestand in einem umfangreichen Wunddébridement mit partieller Cerclagenentfernung sowie direkter Anlage einer Silver®-Schwammes trotz fehlenden Nachweises eines Problemkeimes. Nach insgesamt 17 Verbandwechseln (Abbildung 7) und zwischenzeitlichem partiellen Wundverschluss konnte die Wunde nach 57 Tagen verschlossen werden.

Patient 4 (55 Jahre):

Nach Doppellungentransplantation im Mai 2005 aufgrund eines schweren Lungenemphysems (GOLD IV) mit cor pulmonale und respiratorischer Globalinsuffizienz basierend auf einem kongenitalen Alpha1-Antitrypsinmangel erfolgte die Wiederaufnahme in unsere Klinik zur Re-Transplantation bei chronischer Abstoßung. Bei Vorhandensein eines geeigneten Spenderorgans führten wir im Oktober 2008 eine erneute minimalinvasive Doppellungentransplantation durch. Postoperativ wurde der Verlauf durch Blutungen mit Hämatom und konsekutiver Rethorakotomie kompliziert. Hinzu kamen eine erneute Abstoßungsreaktion mit Urba-sonstherapie sowie eine Langzeitbeatmung aufgrund einer Pneumonie. Zwei Monate nach der Retransplantation kam es zur Ausbildung von Wundheilungsstörungen, wobei in den mikrobiologischen Abstrichen multiresistente Pseudomonaden nachweisbar waren. Unter Therapie mittels GranuFoam Silver® (Abbildung 8, 9) nach chirurgischem Débridement waren nach 19 Tagen die Wundabstriche steril und eine sekundäre Wundnaht möglich.

Diskussion

Aufgrund ihrer besonderen Situation sind organtransplantierte Patienten in besonderem Maße durch nosokomiale Infektionen gefährdet (6). Zum einen weisen sie häufig eine ausgeprägte Komorbidität (z.B. insulinpflichtiger Diabetes mellitus, chronische/passagere Hämodialyse) auf, zum anderen erleichtern die erforderliche Immunsuppression sowie lange postoperative Liegezeiten die Kontamination mit Problemkeimen.

Tiefe thorakale Wundinfektionen mit Übergreifen auf das Mediastinum, gerade bei Nachweis multiresistenter Keime, weisen eine Mortalität von bis zu 30% auf (7). Primäres Ziel der Therapie ist eine lokale Infektkontrolle mit Verhinderung eines Übergreifens auf benachbarte Strukturen durch einen primär möglichst wenig invasiven Eingriff (7), an den sich sekundär nach erfolgter mikrobieller Sanierung der Wundverschluss anschließt (3). Die genannten Kriterien werden durch die V.A.C.®-Therapie erfüllt. Neben kurzen und wenig belastenden Eingriffen ermöglicht diese Form der Behandlung eine Entfernung seröser Wundflüssigkeiten, die Reduktion von Gewebeödemen und fördert die Bildung von Granulationsgewebe (7). Zusätzlich kann der Einsatz neuartiger Silber-beschichteter PU-Schwämme zu einer deutlichen Reduktion der Keimlast im Wundgebiet beitragen (3).

Unser Therapiekonzept konnte erfolgreich auf die Behandlung immunsupprimierter Patienten mit nachgewiesenen Problemkeimen übertragen werden. In 3 von 4 Fällen war ein sekundärer Wundverschluss bei Vorliegen steriler Wundabstriche möglich, die letzte Wunde heilte spontan aus.

Zusammenfassend stellt die V.A.C.®-Therapie ein effizientes Verfahren zur Therapie komplexer thorakaler Wunden auch bei immunsupprimierten Patienten dar. Zusätzlich scheint der Einsatz Silber-beschichteter PU-Schwämme mit erwiesener antimikrobieller Wirkung die Wundheilung weiter zu begünstigen.

Literatur

1. Sjögren J, Malmjö M, Gustafsson R, Ingemansson R (2006) Poststernotomy mediastinitis: a review of conventional surgical treatments, vacuum-assisted closure therapy and presentation of the Lund University Hospital mediastinitis algorithm. *Eur J Cardiothorac Surg.* 30: 898-905.
2. Mokhtari A, Sjögren J, Nilsson J, Gustafsson R, Malmjö M, Ingemansson R (2008) The cost of vacuum-assisted closure therapy in treatment of deep sternal wound infection. *Scand Cardiovasc J.* 42: 85-9.

3. Kutschka I, Bisdas T, Fischer S, Hagl C, Kaladj N, Zardo P, Peters T, Haverich A (2008) Vacuum Assisted Closure (V.A.C.®) With GranuFoam Silver® Improves Healing of complicated sternal wounds. *Eur Surg.* 40: 78-81.
4. Castellano JJ, Shafii SM, Ko F, Donate G, Wright TE, Mannari RJ, Payne WG, Smith DJ, Robson MC (2007) Comparative evaluation of silver-containing antimicrobial dressings and drugs. *Int Wound J.* 4: 114-22.
5. Ulrich C, Hackethal M, Meyer T, Geusau A, Nindl I, Ulrich M, Forschner T, Sterry W, Stockfleth E (2008) Skin infections in organ transplant recipients. *J Dtsch Dermatol Ges.* 6: 98-105.
6. Fleck T (2005) Wundinfektion nach Lungentransplantation – Eine Übersicht über 4 Fälle. In: V.A.C.® Therapie. Willy, Kösel, Altusried-Krugzell (Hrsg.), 1. Auflage, Mai: 410.
7. Kutschka I, Dziadzka S, El Essawi A, Flory P, Harringer W (2006) Vacuum assisted closure for sternal wound infections – Initial therapy and bridging to reconstructive surgery. *Zentralbl Chir.* 131: 129-132.

Die Kombination dermalen Ersatzes (Matriderm®) und V.A.C.®

C. Ottomann, F. Sander, B. Hartmann
Unfallkrankenhaus Berlin, Zentrum für Schwerbrandverletzte mit
Plastischer Chirurgie, Berlin

Einleitung

Der komplette Verlust der dermalen Anteile der Haut bei tiefen zweit- und drittgradigen Verbrennungen führt während des Heilungsprozesses spalthauttransplantierte Wunden oft zur Kontraktion des Wundbettes und ausgedehnter Narbenbildung (1). Neben Wundhei-

lungsstörungen sind Narbenhypertrophien und mangelnde Elastizität der Haut Folge der fehlenden Dermis. Um funktionell und ästhetisch einwandfreie Ergebnisse zu erhalten, ist daher ein Dermisersatz notwendig (2). Eine optimale Rekonstruktion der dermalen Hautschicht wird durch eine Vollhaut-

transplantation erreicht. Der Vorteil liegt in der Transplantation autologen Ersatzes und des nicht notwendigen Ersatzes der Epidermis. Vollhauttransplantate stehen jedoch aufgrund des limitierten Spenderareals nur begrenzt zur Verfügung (3). In der modernen Verbrennungstherapie stehen dermale Ersatzstoffe zur Verfügung. Ein möglicher Ersatz besteht durch die Transplantation von Fremdhaut, die vorrangig als temporärer Hautersatz eingesetzt wird. Dermale Anteile der transplantierten Fremdhaut werden jedoch teilweise inkorporiert, so dass nach Entfernung der allogenen Epidermis ein dermales Gerüst verbleibt. Dieses Verfahren der Composite-Technik wurde erstmals von Cuono et al. 1987 beschrieben (4). Die Indikation besteht in der Notwendigkeit eines dermalen Untergrundes im Rahmen der CEA Transplantation, da sonst eine ausreichende Takerate der transplantierten Keratinozyten nicht erreicht werden kann.

Material und Methoden

Seit Ende der 80er Jahre stehen künstliche Dermisersatzstoffe zur Verfügung. Das erste kommerziell erhältliche Produkt stellte Integra® dar. Integra® artificial skin besteht aus bovinem Kollagen und dem Glykosaminoglykan Chondroitin-6-Sulfat. Als Schicht gegen Austrocknung und als Barriere gegen eine bakterielle Kolonisierung mit möglicher konsekutiver Infektion ist es zusätzlich von einer Silikonfolie bedeckt. Integra® wird nach radikalem Débridement bzw. Nekrektomie auf den vitalen und trockenen Wundgrund transplantiert. Das dreidimensionale Gerüst führt zu einer parallelen und geordneten Anordnung der Kollagenfibrillen, die durch die mit den Kapillaren eingewanderten Fibroblasten produziert werden und so die Bildung von Granulationsgewebe mit nachfolgender Narbenbildung verhin-



Abb. 1a-f: Die einzeitige Applikation des dermalen Ersatzes Matriderm® in Kombination mit einem V.A.C.® Schwamm in der rekonstruktiven Chirurgie nach Narbenexzision

V.A.C.[®] Therapy[™]

Clinical guidelines for deep sternal wound infections

A reference source for clinicians

CONTENTS

About the guidelines	1
1. Introduction	2
Mechanisms of action	2
2. V.A.C.[®] Therapy[™] in sternal wound infections	3
Management of deep sternal wound infections	3
Recognising sternal wound infections	3
Criteria for using V.A.C. [®] Therapy [™]	4
V.A.C. [®] Therapy [™] in special considerations	5
Discontinuing therapy	6
3. Dressing application guidelines	7
Dressing application technique	7
Dressing changes and repeat applications	10
Monitoring the patient and the wound	10
Optimising therapy	11
Index	12

Produced by
MEP Ltd on behalf of KCI Europe Holding BV
Medical Education Partnership Ltd
53 Hargrave Road, London N19 5SH
www.mepltd.co.uk



Copyright © KCI Licensing Inc

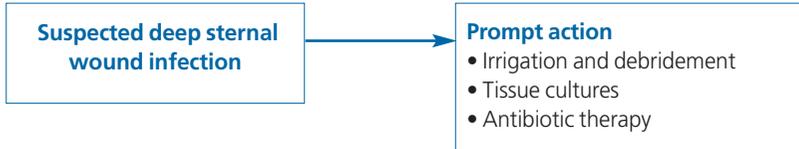
ISBN 90-78026-02-2
ISBN 978-90-78026 02-0

All rights reserved. No reproduction, copy or transmission of this publication may be made without written permission.

No paragraph of this publication may be reproduced, copied or transmitted save with written permission or in accordance with the provisions of the Copyright, Designs & Patents Act 1988 or under the terms of any licence permitting limited copying issued by the Copyright Licensing Agency, 90 Tottenham Court Road, London W1P 0LP.

OVERVIEW OF TREATMENT OPTIONS

WOUND ASSESSMENT AND INFECTION MANAGEMENT



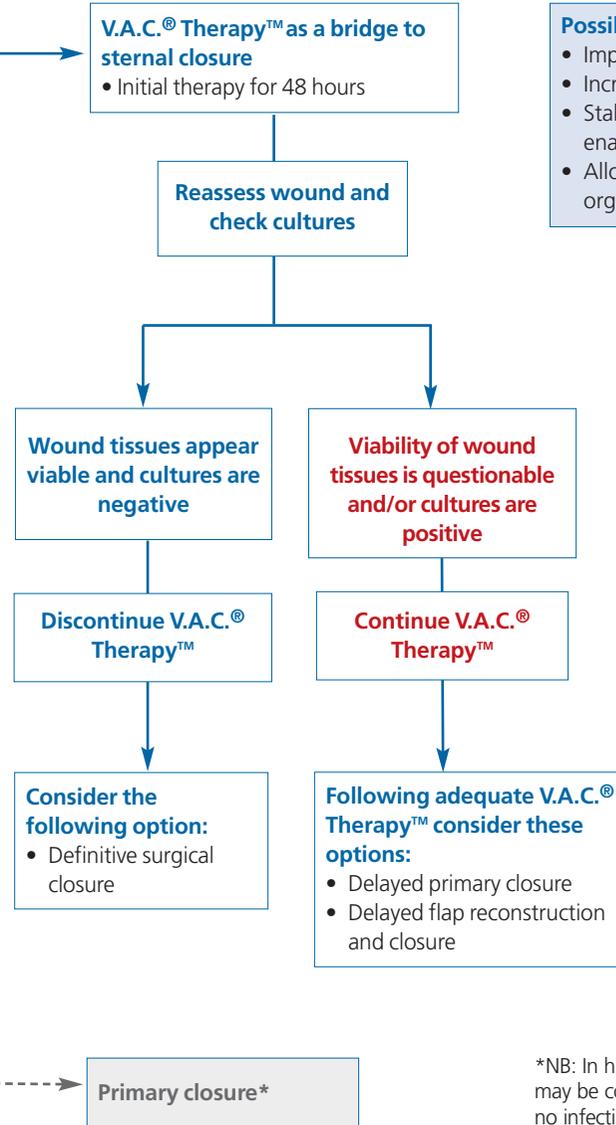
These guidelines are not intended as a guarantee of results, outcome or performance of the V.A.C.® Therapy™ system. They are recommendations to help clinicians establish treatment protocols for the use of V.A.C.® Therapy™ in the management of deep sternal wound infections.

As with any application, follow all appropriate manuals and reference guides as to product use and operation. The treatment of deep sternal wound infection using V.A.C.® Therapy™ does not preclude the use of other surgical techniques to manage infection, or the use of antimicrobial therapy.

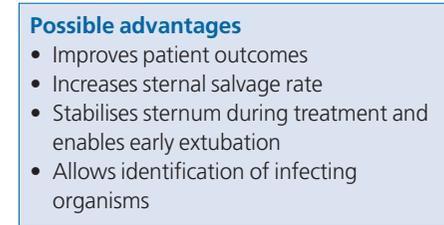
Always consult the relevant section of this booklet and any other product labelling and instructions prior to initiating V.A.C.® Therapy™. Contact your local KCI representative if you have any questions about operation or use. For further information visit www.kci-medical.com or www.kci1.com

Note: This document is only intended for distribution and use in operating geographies of KCI International, excluding the USA.

TREATMENT OPTIONS



OUTCOME



*NB: In highly selected cases, primary closure may be considered if the surgeon judges that no infection is present and tissues are viable.

This booklet provides a practical guide to the use of Vacuum Assisted Closure (V.A.C.[®] Therapy[™]), as an adjunctive therapy, in the management of postoperative deep sternal wound infections. The information presented was agreed by an international consensus group with representation from the US and Europe in the fields of cardiothoracic surgery, plastic and reconstructive surgery, vascular surgery, paediatric cardiovascular surgery and general surgery. The group convened in the Netherlands in May 2006 to develop the guideline content using group discussion and anonymous interactive voting. In addition, further input was received from specialists in the field unable to attend the consensus group meeting. The recommendations are based on current evidence or, where this is not available, the majority consensus opinion of the international group of experts.

INTERNATIONAL CONSENSUS GROUP

Tatjana Fleck, MD, Cardiothoracic Surgeon, Department of Cardiothoracic Surgery, Medical University of Vienna, Austria

Ronny Gustafsson, MD, PhD, Consultant in Cardiac Surgery, Department of Cardiothoracic Surgery, University Hospital, Lund, Sweden

Keith Harding, MB, ChB, MRCP, FRCS, Professor & Honorary Consultant in Rehabilitation (Wound Healing), Cardiff & Vale NHS Trust; Head of Wound Healing Research Unit, Cardiff University, Cardiff, UK

Richard Ingemansson, MD, PhD, Associate Professor, Department of Thoracic Surgery and Clinical Sciences, University Hospital, Lund, Sweden

Mitchell D Lirtzman, MD, FACS, FCCP, FACA, Cardiothoracic Surgeon, Cardiovascular Institute of the South/Lafayette, Lafayette, Louisiana, USA

Herbert L Meites, MD, FACS, MD, Medical Director, Paul Silverstein Burn Center and the Wound Center at INTEGRIS Baptist Medical Center, Oklahoma City, Oklahoma, USA

Reinhard Moidl, MD, Professor, Department of Cardiothoracic Surgery, Medical University of Vienna, Austria

Patricia Price, BA(Hons), PhD, CHPsychol, AFBPS, Professor of Health Sciences/Director of Education and Research, Wound Healing Research Unit, Cardiff University, Cardiff, UK

Andrew Ritchie, MD, PhD, FRCS, Consultant Cardiothoracic Surgeon, Papworth Hospital, Cambridge, UK

Jorge Salazar, MD, Director of Congenital Cardiac Surgery and Assistant Professor in the Division of Cardiothoracic Surgery, The University of Texas Health Center, San Antonio, Texas, USA

Johan Sjögren, MD, PhD, Department of Cardiothoracic Surgery, University Hospital, Lund, Sweden

David H Song, MD, FACS, Associate Professor of Surgery, Chief and Residency Program Director, Section of Plastic Surgery, University of Chicago Pritzker School of Medicine, Chicago, Illinois, USA

Bauer E Sumpio, MD, PhD, Professor and Head of Vascular Surgery, Yale University School of Medicine, New Haven, Connecticut, USA

Boulos Toursarkissian, MD, Associate Professor and Chief, Division of Vascular Surgery, UTHSCSA, Department of Surgery, San Antonio, Texas, USA

Ferdinand Waldenberger, MD, PhD, Associate Professor, Cardiovascular Surgical Centre, Hospital Wien-Hietzing, Vienna, Austria

Walter Wetzel-Roth, MD, Cardiothoracic Surgeon, Department of Vascular and Thoracic Surgery, Hospital Schwabmünchen, Germany

I. INTRODUCTION

V.A.C.[®] Therapy™ is the controlled application of continuous or intermittent topical negative pressure across the entire wound surface using a specialised wound dressing to help promote wound healing. V.A.C.[®] Therapy™ uses T.R.A.C.[®] (Therapeutic Regulated Accurate Care) technology to monitor and maintain the target pressure at the wound site, which helps to deliver consistent therapy.

MECHANISMS OF ACTION OF V.A.C.[®] THERAPY™

V.A.C.[®] Therapy™ helps promote wound healing via a number of mechanisms, some of which are characterised in laboratory studies and others, which are recognised, in part, in the clinical setting (Banwell PE, Musgrave M. Topical negative pressure therapy: mechanisms and indications. *Int Wound J* 2004; 1: 95-106).

The application of V.A.C.[®] Therapy™ may result in:

- **transmission of micromechanical forces to the underlying tissue.** These forces can induce micro-deformations of the tissue and may activate intracellular signalling pathways, resulting in increased cell proliferation and migration
- **stimulation of angiogenesis and promotion of wound contraction** via remodelling of collagen fibrils
- **increased local blood perfusion.** This can lead to a reduction in local oedema and removal of excess interstitial fluid
- **physical and biochemical changes in the wound environment,** which may contribute to the increased granulation tissue formation that has been observed during V.A.C.[®] Therapy™
- **reduction in inhibitory substances.** This may help to clear bacteria from the wound environment, and may remove toxic compounds such as inhibitory mediators and matrix metalloproteinases, thereby enhancing wound healing
- **a moist wound environment.** V.A.C.[®] Therapy™ provides a closed wound environment and makes use of an adhesive semi-occlusive drape that retains moisture and allows gas exchange. This drape also helps to prevent tissue dehydration and wound contamination.

Notice to users: To help reduce the potential risk of serious or fatal injury, always consult a physician, and read and follow all instructions for use, including V.A.C.[®] Therapy™ Safety Information, Dressing Application instructions, V.A.C.[®] Therapy™ Unit instructions and V.A.C.[®] Therapy™ Clinical Guidelines prior to each use. For additional information, please visit the website at www.kci-medical.com/www.kci1.com.

2. V.A.C.[®] THERAPY™ IN STERNAL WOUND INFECTIONS

MANAGEMENT OF DEEP STERNAL WOUND INFECTIONS

Deep sternal wound infection (mediastinitis) is associated with poor patient outcomes. This may result in considerable morbidity and mortality*, prolonged hospital stay, increased intensive care unit (ICU) use and the need for multiple surgical procedures. The cost associated with postoperative mediastinitis can be up to two to three times that for uncomplicated surgery (Loop FD, Lytle BW, Cosgrove DM, et al. Sternal wound complications after isolated coronary artery bypass grafting: early and late mortality, morbidity, and cost of care. *Ann Thorac Surg* 1990; 49(2):179-86).

*Mortality rates in deep sternal wound infections using conventional treatment

- Five percent mortality rate reported by Milano CA, Georgiade G, Muhlbaier LH et al. Comparison of omental and pectoralis flaps for post-sternotomy mediastinitis. *Ann Thorac Surg* 1999; 67: 377-80.
- Forty-six percent mortality rate (using open wound packing and debridement) reported by Sutherland RD, Martinez HE, Guynes WA et al. Postoperative chest wound infections in patients requiring coronary bypass: a controlled study evaluating prophylactic antibiotics. *J Thorac Cardiovasc Surg* 1977; 73: 947.

The involvement of a cardiothoracic surgeon with appropriate expertise is essential in the management of deep sternal wound infections. These are complex wounds, involve major organs and complications can be life threatening.

RECOGNISING STERNAL WOUND INFECTIONS

The diagnosis of deep sternal wound infection can be complex, and relies on a combination of a high index of suspicion, good clinical judgement, rapid and appropriate investigation including laboratory parameters and the presence of positive wound cultures.

Superficial infection of sternotomy wounds, involving subcutaneous tissue, but which does not penetrate down to the underlying steel wires, bone or cartilage, may progress to deep infection. Depending on the size/extent of wound dehiscence, V.A.C.[®] Therapy™ may be considered in superficial sternal infections as an adjunct to therapy.

Deep sternal wound infection can present with a stable or unstable sternum, and with or without the presence of osteomyelitis.

Terms including sternitis, mediastinitis, wound infection and wound complication have all been used in the past to describe deep sternal wound infections following surgery. Definitions and classifications for deep sternal wound infections are described elsewhere (Bruce J, Russell EM, Mollison J et al. The measurement and monitoring of surgical adverse events. *Health Technol Assess* 2001; 5(22):1-194. El Oakley R, Wright JE. Postoperative mediastinitis: classification and management. *Ann Thorac Surg* 1996; 61(3):1030-06).

Clinical features of mediastinitis may be masked by postoperative pain or concomitant infection. Mediastinitis should be suspected in the presence of any or all of the following:

- fever (i.e. above 38°C) and leucocytosis in the absence of local signs or symptoms
- wound discharge (found in 70-80% of cases)
- wound pain and tenderness
- sternal instability/dehiscence
- dyspnoea after respiratory causes have been ruled out
- wound cellulitis and wound fluctuance (evidence of fluid beneath incision)
- positive blood culture or wound culture in combination with one of the above.

The principles of treatment for deep sternal wound infections include early recognition and prompt treatment of infection (see *Overview of treatment options*, inside front flap). Thorough irrigation and appropriate debridement will be necessary, after which initiation of V.A.C.® Therapy™ may be considered.

CRITERIA FOR USING V.A.C.® THERAPY™

If the patient meets the standard criteria for using V.A.C.® Therapy™ (see V.A.C.® Therapy™ Clinical Guidelines), V.A.C.® Therapy™ should be used where the patient has a deep sternal wound infection with consideration of the following indications, contraindications and special circumstances.

V.A.C.® Therapy™ should be:

- considered in the management of deep sternal wound infections – i.e. standard treatment
- considered where deep infection is either suspected or confirmed (after appropriate debridement)
- initiated as early as possible, after surgical investigation, irrigation and debridement, together with targeted antibiotic therapy of the underlying infection and drainage.

The application of V.A.C.® Therapy™ in deep sternal wound infections may have the following benefits:

- re-establish sternal stability and permit sternal salvage
- promote formation of granulation tissue and wound closure
- enable patients to be extubated and mobilised early
- decrease long-term mortality compared to conventional treatment options (Sjögren J, Nilsson J, Gustafsson R et al. The impact of vacuum-assisted closure on long-term survival after poststernotomy mediastinitis. *Ann Thorac Surg* 2005; 80:1270-75).

V.A.C.® Therapy™ should NOT be used:

- without adequate surgical investigation, irrigation and debridement of the wound
- in the presence of undebried necrotic tissue or eschar
- where the patient has untreated/undebried osteomyelitis
- where there is excessive or uncontrolled bleeding
- where the patient has a chest or pulmonary malignancy, or malignancy in the wound
- when experience or knowledge of the method is inadequate.

Special care should be taken in any of the following circumstances:

- where there are exposed blood vessels, organs or nerves (adequate protection is required using overlying fascia, tissue or other protective barrier, e.g. one or more layers of a non-adherent vapour permeable interposed dressing)
- where the patient has vascular anastomoses, including coronary artery bypass grafts
- where the patient has weakened, irradiated or sutured blood vessels or organs
- where it is difficult to achieve haemostasis
- when the patient has a bleeding disorder, i.e. coagulopathy (manage by packing the wound and monitoring to assess appropriateness of V.A.C.® Therapy™)
- when the patient is receiving concomitant therapy with anticoagulant medication
- where the wound contains bone fragments or sharp edges that could puncture protective barriers, vessels or organs (manage by debriding the sharp edges and protecting with one or more layers of a non-adherent interposed dressing)

V.A.C.® THERAPY™ IN SPECIAL CONSIDERATIONS

In addition to the special circumstances listed above, the following have been identified as special considerations requiring particular expertise.

Paediatric patients and neonates

- the potential for haemodynamic effects is more significant. In the case of haemodynamic compromise, pressure settings may be reduced
- lower pressure settings (e.g. 50-75mmHg) may be recommended in neonates and infants
- to avoid the problems of skin retraction and heart compression, it is important not to overpack or oversize the foam dressing.

Post-cardiotomy syndrome

In post-cardiotomy syndrome the closure or even approximation of the sternal halves impairs cardiac filling and contraction. It is necessary to leave the sternum open for a few days, according to clinical judgement. In such cases V.A.C.® Therapy™ can be used as a temporary closure technique, usually after 24 hours, when bleeding is controlled and the patient is haemodynamically stable. Delayed closure using V.A.C.® Therapy™ provides a closed wound environment. It can also create a splinting effect of the chest, which may contribute to respiratory stability.

Fluid overload and systemic inflammatory response syndrome (SIRS)

Excessive fluid in the tissues and cavities (pleural/pericardial) causes the microcirculation to be significantly impaired. V.A.C.® Therapy™ may be helpful in decreasing tissue fluid overload and increasing the microcirculation.

DISCONTINUING THERAPY

V.A.C.® Therapy™ should be stopped immediately and the dressing removed in the following circumstances:

- the patient develops new onset cardiac arrhythmias/dysrhythmias, hypotension or low cardiac output state (where appropriate)
- the patient cannot tolerate the therapy because of pain (reducing pressure or repositioning the dressing may alleviate pain)
- the patient is persistently septic (requires exploration/re-evaluation)
- onset or sudden increase in bleeding.

If there is a sudden increase in bleeding for any reason, or if frank blood is seen in the tubing or in the canister, stop V.A.C.® Therapy™, take measures to stop the bleeding and seek attention from a surgeon with appropriate experience immediately.

DRESSING APPLICATION TECHNIQUE

In addition to general recommendations for dressing application technique, the following offers specific suggestions for the use of V.A.C.® Therapy™ in the management of deep sternal wound infections.

1. Wound assessment and infection management

The first application of V.A.C.® Therapy™ should take place in the operating room under general anaesthesia, following a comprehensive examination of the patient.

The decision to use V.A.C.® Therapy™ should be made by the lead clinician. Patients should be assessed on an individual basis.

- Remove all foreign bodies including free suture materials and wires, where appropriate.
- Take cultures from appropriate sites, as directed by the lead clinician, to identify the organism of the underlying infection. Tissue samples are better than swabs.
- The underlying infection should be subject to active management including surgical intervention and tailored antimicrobial therapy as deemed appropriate by the lead clinician.

Haemostasis needs to be assured, and clinically relevant coagulopathy identified and corrected. Bleeding should be investigated.

2. Debridement

- Thoroughly debride the wound of necrotic tissue, all eschar and hardened slough. Particular care is required to avoid injury to the heart, vascular anastomoses or other vital structures.
- Remove soft tissue, bone and costal cartilage that are clearly necrotic or infected as thoroughly as possible.
- Irrigate the debrided wound with normal saline or other solution as directed by the lead clinician.
- Assess the wound for any significant persistent bleeding.
- Check and eliminate all bone fragments and irregular or sharp sternal edges in contact with the heart (see Special care, p5). If necessary, and it is technically safe to do so, the heart and lungs should be freed from the sternal edges (this should only be performed by a surgeon with experience in this procedure).

The use of bone wax is not recommended.

3. Protection of the underlying structures

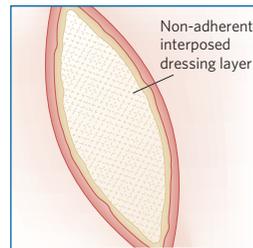
- Protect the heart and any exposed blood vessels/anastomoses, nerves or other organs using one or more layers of a non-adherent interposed dressing. The number of layers and type of non-adherent porous material used is a decision that should be made by the lead clinician. If possible, the interposed dressing should be placed under the sternal edges by at least 1-2cm, and especially under the left sternal portion.

Protection of the underlying structures is possibly the most important component of V.A.C.[®] Therapy™ application in this wound type.

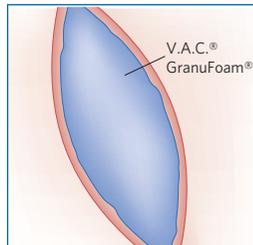
Structures may shift on application of V.A.C.[®] Therapy™, potentially causing contact between the heart and the sternal edges. The non-adherent interposed layer and positioning of the foam dressing is intended to protect the heart and prevents adhesions and injury (i.e. cardiac rupture) during therapy.

4. V.A.C.[®] foam dressing application

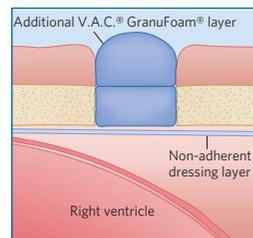
- Cut the foam (V.A.C.[®] GranuFoam[®]), trimming it to fit between the sternal edges, but 1.5 times broader than the sternal gap, to allow for volume reduction when the vacuum is applied. **Note: the width of GranuFoam[®] is usually sufficient to fit between the edges of an adult sternum; to fit, turn on its side and cut to depth required.**
- Place the foam strip deep into the wound to extend just below the lower edge of the sternum. Use sufficient foam so that when the foam contracts it is at the level of the posterior table of the sternum, and no higher. This prevents the heart from rising up above this level and from coming into contact with the posterior sternum.
- Apply an additional layer of GranuFoam[®] subcutaneously to cover the entire wound. This layer should be trimmed to the shape of the wound. Allow a 2cm margin over the surface of the skin, to allow for volume reduction during vacuum application.
- Use more than one piece of foam if the wound is larger than the largest dressing available. If required, ensure that adjoining pieces of foam are in direct contact with one another to provide even distribution of negative pressure.
- Count the pieces of foam and record this information in the patient's records.



A non-adherent, porous, interposed dressing layer is used to protect underlying structures



The first layer of V.A.C.[®] GranuFoam[®] is placed between the sternal bone edges to seal the gap.



A second layer of V.A.C.[®] GranuFoam[®] should be trimmed to the shape of the wound.

5. V.A.C.[®] Drape application

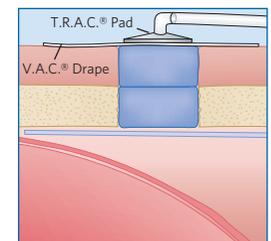
- Clean and dry the periwound area: if skin is moist as a result of perspiration, oil or body fluids, a degreasing agent may be required.
- For patients with fragile or excoriated periwound tissue, a protective, thin-layered dressing (liquid barrier film), a hydrocolloid dressing or a vapour-permeable adhesive film dressing may be applied.
- Size and trim the V.A.C.[®] Drape to cover the foam dressing, with an additional 2-5cm border to avoid skin irritation.
- Avoid covering chest tube exit sites with the drape.
- If necessary, use more than one piece of drape to cover the area.

6. T.R.A.C.[®] Pad and tubing application

- Create a T.R.A.C.[®] Pad opening in the drape. Lift the drape with your thumb and forefinger and cut a 1-2cm round hole.
- Apply the T.R.A.C.[®] Pad directly over the hole in the drape.
- Apply gentle pressure around the T.R.A.C.[®] Pad to ensure complete adhesion.
- If air leaks develop (e.g. around the exit site of the drainage tubes), additional drape can be applied to create a seal.
- If there is excess wound fluid or at the discretion of the lead clinician, one T.R.A.C.[®] Pad can be placed in the upper (cranial) part of the wound and a further T.R.A.C.[®] Pad placed in the lower (caudal) part of the wound. Connect the two tubes using a Y-connector.

7. Commencing V.A.C.[®] Therapy™

- If appropriate, use a trial pressure setting of 50mmHg to check for compression of the foam dressing into the wound.
- If the foam is not flat (i.e. at the level of the skin), manipulate or add additional foam and repeat.
- Repeat the trial procedure. If the foam is at the level of the skin, the negative pressure can then be increased to 125mmHg. In neonates and infants, a lower pressure setting (e.g. 50-75mmHg) may be recommended.



V.A.C.[®] dressing should be flat at the level of the skin when therapy is commenced

For patients with sternectomies or sternotomies, the continuous therapy setting is recommended throughout the treatment period to help stabilise the chest wall. The splinting effect may promote comfort for patients and may facilitate extubation.

Table 3.1: Recommended settings for infected sternal wounds*

Cycle	Target pressure V.A.C. [®] GranuFoam [®]	Dressing change interval
Continuous for duration of therapy	125mmHg	Every 48 hours (12-24 hours in presence of infection)

*Based on manufacturer's recommendations

DRESSING CHANGES AND REPEAT APPLICATIONS

The manufacturer currently recommends that dressing changes should be repeated every 48 hours or more often (i.e. every 12-24 hours) in the presence of infection.

- Change the V.A.C.[®] dressing as directed by the lead clinician.
- Depending upon the individual clinical circumstances, dressing changes may be carried out in the operating room under general anaesthesia, in the ICU using sedation as required, or at the bedside.
- At each dressing change, inspect all tissues, debride any necrotic and/or avascular areas, repeat cultures if appropriate and ensure adequate haemostasis. In addition, check there are no sharp edges and the posterior sternum is free from other tissues.
- Ensure all foam pieces have been removed.

Care must be taken when removing the dressing as there may be increased sternal instability.

MONITORING THE PATIENT AND THE WOUND

- Regularly monitor the patient and the wound and tailor therapy as appropriate.
- Ideally, the same person should carry out the evaluation throughout the treatment period. Photographic documentation of wound healing may be helpful.
- If no response/improvement in the wound is observed within one to two weeks, reconsider treatment including targeted antibiotic therapy and further debridement. Consider other treatment modalities (e.g. pectoralis flap to reset the sternum).
- Overall outcomes may be improved when KCI-trained wound care professionals are involved in supporting clinicians using V.A.C.[®] Therapy[™].

The progress of wound healing should be monitored regularly:

- The wound should begin to change colour and become a deeper red as perfusion increases and there is evidence of granulation tissue formation.
- As the wound forms new granulation tissue, new epithelial growth should be seen at the wound edges.
- The volume of wound exudate should decrease.
- Wound exudate may change in appearance from serosanguinous to serous or no secretion.

Note: Laboratory parameters may be checked daily to monitor the wound. In some centres, serum C-reactive protein (CRP) levels are used and the wound considered free of infection when the serum CRP level is 50-70mg/L and decreasing, without other tissue injury or infection elsewhere (Gustafsson R, Johnsson P, Algotsson L et al. Vacuum-assisted closure therapy guided by C-reactive protein levels in patients with deep sternal wound infections. *J Thorac Cardiovas Surg* 2002; 123: 895-900).

V.A.C.[®] Therapy[™] should be discontinued when the desired outcome has been achieved for the patient as defined by the lead clinician (i.e. resolution of infection and good granulation tissue in wound bed). A decision to restart V.A.C.[®] Therapy[™] if there is wound deterioration should be made at the discretion of the lead clinician.

OPTIMISING THERAPY

Obtaining maximum benefit from V.A.C.[®] Therapy[™] relies on the treatment of the underlying and associated wound aetiology, effective wound management strategies (e.g. nutritional support), together with appropriate and safe use of the technology. When V.A.C.[®] Therapy[™] is used, this should be in combination with antibiotics (according to local protocols) and be implemented as early as possible after appropriate debridement. The consensus group identified the following tips for optimising therapy:

- Exercise high index of suspicion. Decide early to open the sternum if there is any clinical concern.
- Avoid reopening spaces that have been closed when inspecting the wound at dressing changes. Exercise caution where new granulation tissue has formed.
- Ensure meticulous irrigation, drainage and debridement prior to initiating V.A.C.[®] Therapy[™].
- Use a non-adherent interposed dressing between the sternum and heart to protect underlying structures.
- Ensure sufficient foam to stabilise the sternum and protect underlying structures from bony edges.
- Ensure the width of the foam fits perfectly between the sternal edges.
- Use a negative pressure setting of 50mmHg at the start of the first application to ensure sufficient foam and check haemodynamic stability before increasing to target pressure levels.
- Use photographic documentation of the wound to accurately evaluate progress.
- Create a specialist multidisciplinary team, or seek advice from those who are experienced in the use of V.A.C.[®] Therapy[™] in deep sternal wound infections.

- Angiogenesis, stimulation of 2
 Anticoagulant medication 5
 Arrhythmias 6
 Assessment of wound 7
- Bleeding 5, 6, 7
 Blood vessels, exposed 5, 8
 Bone fragments 5, 7
 Bone wax 7
- C-reactive protein (CRP) levels 11
 Cellulitis 4
 Clinical features of deep sternal wound infection 4
 Coagulopathy 5
 Collagen fibril remodelling 2
 Colour changes in wound 10
 Commencing V.A.C.[®] Therapy™ 9
 Continuous therapy setting 9, 10
 Contraindications to use 5
 Coronary artery bypass grafts 5
 Culture samples 7
- Debridement 4, 5, 7, 11
 Delayed closure 5–6
 Diagnosis of deep sternal wound infection 3, 4
 Discontinuation of therapy 11
 indications for 6
 Drape application 9
 Dressing application technique 7–9
 Dressing changes 10, 11
 Dyspnoea, as sign of mediastinitis 4
 Dysrhythmias 6
- Evaluation of therapy 10–11
- Fever 4
 Fluctuance of wound 4
 Fluid overload 6
 Foam dressing application 8, 11
 in paediatric and neonatal therapy 5
- GranuFoam[®] application 8
 Granulation tissue formation 2, 4, 10
- Heart, protection 8
 Hypotension 6
- Indications for use 4
 Inhibitory mediators 2
 Interposed dressing layer 5, 8, 11
 Irrigation 4, 5, 7, 11
- Laboratory tests 11
 Low cardiac output 3
- Malignancy 5
 Matrix metalloproteinases 2
 Mechanisms of action, V.A.C.[®] Therapy™ 2
 Mediastinitis, diagnosis 3–4
 Micromechanical forces, actions 2
 Monitoring therapy 10–11
 Mortality rates, conventional therapy 3
 impact of V.A.C.[®] Therapy™ 4
- Neonatal use 5
 Nerves, exposed 5, 8
- Optimisation of therapy 11
 Organs, exposed 5, 8
 Osteomyelitis 5
- Paediatric use 5
 Pain
 resulting from V.A.C.[®] Therapy™ 6
 as sign of mediastinitis 4
 Periwound area preparation 9
 Photographic documentation 10, 11
 Post-cardiotomy syndrome 5
 Pressure settings 9, 10, 11
 in paediatric and neonatal therapy 5
- Sepsis, persistent 6
 SIRS (systemic inflammatory response syndrome) 6
 Special circumstances, V.A.C.[®] Therapy™ 5
 Sternal stability 4, 9
 Sternal wound infection, diagnosis 3–4
 Sternectomies/sternotomies 9
 Superficial infections 3
- T.R.A.C.[®] Pad and tubing application 9
- V.A.C.[®] Drape application 9
 V.A.C.[®] Therapy™
 commencement 9
 criteria for use 4–5
 mechanisms of action 2
 Vascular anastomoses 5
- Wound, features of mediastinitis 4
 Wound environment 2
 Wound exudate, volume and appearance 10
 Wound healing, monitoring 10–11

Asia
KCI Medical Asia Pte Ltd.
 50 Ubi Crescent #01-01
 Singapore 408568
 Tel +65 6742 6686
 Fax +65 6749 6686
 Toll Free 1 800 742 9929
 www.kci-medical.com

Australia
KCI Medical Australia Pty Ltd.
 Unit 2A-B
 6 Boundary Road
 Northmead NSW 2152
 Australia
 Tel +61 (0)2 9630 8877
 Fax +61 (0)2 9630 8855
 Toll Free 1 800 815 529
 Customer Service 1 300 136 546
 www.kci-medical.com

Austria
KCI Austria GmbH
 Franz-Heider-Gasse 3
 A-1230 Wien, Austria
 24h Cust. Service +43 1 86 330
 Fax +43 1 86 3306
 www.kci-medical.com

Belgium
KCI Medical Belgium B.V.B.A.
 Ambachtslaan 1031
 3990 Peer
 Limburg, Belgium
 Freephone 0800 524 63342
 Freefax 0800 825 99691
 Int. Tel +31 (0)30 635 58 85
 Int. Fax +31 (0)30 637 76 90
 www.kci-medical.com

Canada
KCI Medical Canada Inc.
 95 Topflight Drive
 Mississauga
 Ontario L5S 1Y1
 Canada
 Toll free 1 800 668 5403
 Tel 1 905 565 7187
 Fax 1 905 565 7270
 www.kci-medical.com

Denmark
KCI Medical ApS
 Nybrovej 83
 DK-2820 Gentofte
 Denmark
 Tel +45 3990 0180
 Fax +45 3990 1498
 www.kci-medical.com

France
KCI Médical Sarl
 Parc Technopolis
 17, Avenue du Parc
 91380 Chilly Mazarin
 France

Tel +33 (0)1 69 74 71 71
 Fax +33 (0)1 69 74 71 72 – Service
 Clients
 Fax +33 (0)1 69 74 71 73 –
 Administration
 www.kci-medical.com

Germany
KCI Medizinprodukte GmbH
 Hagenauer Strasse 47
 D-65203 Wiesbaden
 Germany
 24h Free Call Cust. Service
 +49 (0)800 783 3524
 Fax +49 (0)800 329 3524
 www.kci-medical.com

Ireland
KCI Medical Ltd.
 H17 Centrepont Business Park
 New Nangor Road
 Dublin 12
 Ireland
 24h Cust. Service 1 800 33 33 77
 Tel +353 (1) 465 9510
 Fax +353 (1) 465 9500
 www.kci-medical.com

Italy
KCI Medical Srl
 Via Meucci, 1
 20090 Assago (MI)
 Italy
 24h Cust. Service +39 02 457 174 218
 Tel +39 02 457 174 1
 Fax +39 02 457 174 210
 www.kci-medical.com

Latin America
KCI International Inc
 8023 Vantage Drive
 San Antonio, Texas 78230
 USA
 Tel +1 210 255 6460
 Fax +1 210 255 6991
 www.kci.com

South Africa
KCI Medical South Africa (Pty) Ltd.
 Block 6 · Thornhill Park
 94 Bekker Road · Midrand 1685
 South Africa
 24h Cust. Service +27 82 494 2984
 Tel +27 11 315 0445
 Fax +27 11 315 1757
 www.kci-medical.com

Spain
KCI Clinic Spain SL
 Calle Basauri 17 · Edificio A 2-F
 28023 Madrid
 Spain
 Tel +34 91 708 0835
 Fax +34 91 372 8648
 www.kci-medical.com

Sweden
KCI Medical AB
 Pyramidvägen 7
 SE-169 56 Solna
 Sweden
 Tel +46 8 544 996 90
 Fax +46 8 544 996 91
 www.kci-medical.com

Switzerland
KCI Medical GmbH
 Grindlenstrasse 5
 CH-8954 Geroldswil, Switzerland
 24h Cust. Service +41 0848 848 900
 Fax Cust. Service +41 0848 848 901
 Main +41 43 455 3000
 Fax +41 43 455 3020
 www.kci-medical.com

The Netherlands
**KCI International
 KCI Europe Holding B.V.**
 Parktoeren, 6th Floor
 Van Heuven Goedhartlaan 11
 PO Box 129, 1180 AC Amstelveen
 The Netherlands
 Tel +31 (0) 20 426 0000
 Fax +31 (0) 20 426 0099
 www.kci-medical.com

KCI Medical B.V.
 Duikboot 1
 3991 CK Houten
 The Netherlands
 24h Cust. Support +31 (0) 30 635 60 60
 Tel +31 (0) 30 635 58 85
 Fax +31 (0) 30 637 76 90
 www.kci-medical.com

United Kingdom
KCI Medical Ltd.
 KCI House
 Langford Business Park · Langford Locks
 Kidlington OX5 1GF, UK
 24h Cust. Service +44 (0) 800 980 8880
 Tel +44 (0)1865 840 600
 Fax +44 (0)1865 840 626
 www.kci-medical.com

KCI Medical Products (UK) Ltd.
 11 Nimrod Way
 Ferndown Industrial Estate
 Wimborne, Dorset BH21 7SH, UK
 Tel +44 (0)1202 654 100
 Fax +44 (0)1202 654 140
 www.kci-medical.com

KCI UK Holdings Ltd.
 1st Floor 3 Cedar Park
 Cobham Road
 Ferndown Industrial Estate
 Wimborne, Dorset BH21 7SB, UK
 Tel +44 (0) 1202 866 400
 Fax +44 (0) 1202 866 408
 www.kci-medical.com

Printed in the UK



Topical negative pressure effects on coronary blood flow in a sternal wound model

Sandra Lindstedt, Malin Malmström, Bodil Gesslein, Richard Ingemansson

Lindstedt S, Malmström M, Gesslein B, Ingemansson R. Topical negative pressure effects on coronary blood flow in a sternal wound model. *Int Wound J* 2008;5:503–509.

ABSTRACT

Several studies have suggested that mediastinitis is a strong predictor for poor long-term survival after coronary artery bypass surgery (CABG). In those studies, several conventional wound-healing techniques were used. Previously, we have shown no difference in long-term survival between CABG patients with topical negative pressure (TNP)-treated mediastinitis and CABG patients without mediastinitis. The present study was designed to elucidate if TNP, applied over the myocardium, resulted in an increase of the total amount of coronary blood flow. Six pigs underwent median sternotomy. The coronary blood flow was measured, before and after the application of TNP (–50 mmHg), using coronary electromagnetic flow meter probes. Analyses were performed before left anterior descending artery (LAD) occlusion (normal myocardium) and after 20 minutes of LAD occlusion (ischaemic myocardium). Normal myocardium: 171.3 ± 14.5 ml/minute before to 206.3 ± 17.6 ml/minute after TNP application, $P < 0.05$. Ischaemic myocardium: 133.7 ± 18.4 ml/minute before to 183.2 ± 18.9 ml/minute after TNP application, $P < 0.05$. TNP of –50 mmHg applied over the LAD region induced a significant increase in the total coronary blood flow in both normal and ischaemic myocardium.

Key words: Coronary blood flow • Poststernotomy mediastinitis • Topical negative pressure (TNP)

INTRODUCTION

Topical negative pressure (TNP) has been shown to facilitate the healing of chronic and problematic wounds (1–10). The physiological and molecular biological mechanisms by which TNP promotes wound healing are still largely unknown. However, TNP is known to stimulate blood flow in tissues such as subcutaneous tissue and skeletal muscle (11–13). TNP produces mechanical stress and a pressure gradient across the tissue, which cause a surge of blood to the area. Mechanical forces and increased blood flow are known to stimulate granulation tissue

formation, that is endothelial proliferation, capillary budding and angiogenesis (14–16).

Several studies have suggested that mediastinitis is a strong predictor for poor long-term survival after coronary artery bypass surgery (CABG) (17–22). Braxton and coworkers showed in a large study including 36 078 patients after isolated CABG that actuarial survival after 10 years was 39% in patients with mediastinitis and 70% in patients without mediastinitis (20). Milano *et al.* have suggested that mediastinitis may cause negative long-term effects on several organs such as the heart and kidneys (19). Theoretically, a massive immunological response during a prolonged period of infection may cause adverse effects on bypass grafts. In those studies, reporting poor long-term survival after mediastinitis, several conventional wound-healing techniques were used (closed irrigation, delayed wound closure or reconstructing with omentum or pectoral flaps).

Previously, we have showed no difference in long-term survival between CABG patients

Key Points

- TNP increases the total amount of coronary blood flow
- mechanical forces and increased blood flow are known to stimulate granulation tissue formation, that is endothelial proliferation, capillary budding and angiogenesis

Authors: S Lindstedt, MD, Department of Cardiothoracic Surgery, Heart and Lung Center, Lund University Hospital, Lund, Sweden; M Malmström, MD, PhD, Department of Medicine, Lund University Hospital, Lund, Sweden; B Gesslein, MSc, Department of Medicine, Lund University Hospital, Lund, Sweden; R Ingemansson, MD, PhD, Department of Cardiothoracic Surgery, Lund University Hospital, Lund, Sweden

Address for correspondence: S Lindstedt, MD, Department of Cardiothoracic Surgery, Heart and Lung Center, Lund University Hospital, SE-221 85 Lund, Sweden
E-mail: sandra.lindstedt@skane.se

Key Points

- the present study was designed to elucidate if the increase in microvascular blood flow is a result of an increase of the total coronary blood flow or a redistribution of blood from other areas of the heart
- no such study has to our knowledge been performed before
- a porcine model was used for the present study

with TNP-treated mediastinitis and CABG patients without mediastinitis (23). This indicates that these patients might have developed increased coronary collateral blood vessels during TNP and may therefore be better prepared when bypass grafts fail to work. It may be that the TNP stimulation of blood flow and development of collateral blood vessels in part account for the reduced long-term mortality in patients treated with TNP for poststernotomy mediastinitis after CABG.

We have earlier shown that TNP of -50 mmHg significantly increases microvascular blood flow in the underlying myocardium (24). The present study was designed to elucidate if the increase in microvascular blood flow is a result of an increase of the total coronary blood flow or a redistribution of blood from other areas of the heart. An unchanged or decreased amount of the total coronary blood flow to the heart might theoretically cause ischaemia in parts of the myocardium not exposed to TNP. However, an increase in total coronary blood flow will stimulate granulation tissue formation, that is endothelial proliferation, capillary budding and angiogenesis. No such study has to our knowledge been performed before.

MATERIALS AND METHODS**Experimental animals**

A porcine model was used for the present study. Six domestic landrace pigs of both genders, with a mean body weight of 70 kg, were fasted overnight with free access to water. The study was approved by the Ethics Committee for Animal Research, Lund University, Sweden. The investigation complied with the 'Guide for the Care and Use of Laboratory Animals' as recommended by the U.S. National Institutes of Health and published by the National Academies Press (1996).

Anaesthesia

All animals were premedicated intramuscularly with ketamine (30 mg/kg) before they were brought into the laboratory. Before commencing surgery, sodium thiopental (5 mg/kg), atropine (0.02 mg/kg) and pancuronium (0.5 mg/kg) were given intravenously. Tracheotomy was performed with a Portex endotracheal tube (7.5-mm internal diameter, Medcompare™, San Francisco, CA, USA). A servo-ventilator

(Siemens Elema 300A, Stockholm, Sweden) was used for mechanical ventilation throughout the experiment. The ventilator settings used were minute volume = 100 ml/kg, $FiO_2 = 0.5$, breathing frequency = 16 breaths/minute and positive end-expiratory pressure = 5 cm H_2O .

Anaesthesia and muscular paralysis were maintained with a continuous intravenous infusion of 8–10 mg/kg/hour propofol (Diprivan®; AstraZeneca, Sodertalje, Sweden), 0.15 mg/kg/hour fentanyl (Leptanal®; Lilly, Solna, Sweden) and 0.6 mg/kg/hour pancuronium (Pavulon®; Organon Teknika, Boxtel, the Netherlands).

Data acquisition

Mean arterial pressure, central venous pressure, heart frequency and ventilatory parameters were recorded throughout the experiments.

Surgical procedure

Surgery was performed through median sternotomy. After heparinisation (400 IU/kg), a cardiopulmonary bypass (CPB) was installed with an arterial cannula [22 French, DLP® Elongated One-Piece Arterial Cannula (EOPA™); Medtronic Inc., Minneapolis, MO] in the distal ascending aorta and a venous cannula (32 French, MC2® Two-Stage Venous Cannula, also from Medtronic Inc.) inserted through the right atrium. Before cannulation of the heart, the cannulae were inserted through the thoracic wall to prevent air leakage during TNP application. CPB was conducted in normothermia. Ventricular fibrillation was subsequently induced in the heart. No aortic cross-clamping was performed and no cardioplegia was used. The mean arterial pressure was maintained between 60 and 80 mmHg. A left ventricular vent (DLP® Vent, also from Medtronic Inc.) was used to protect the left chamber from overloading. Pulmonary ventilation was applied at a rate of 4 l/minute during the experiments. Coronary pulmonary bypass prevents the risk of circulatory failure during left anterior descending artery (LAD) occlusion, thereby facilitating experimental analysis in the case of the ischaemic myocardium.

Coronary electromagnetic flow meter probes

The coronary electromagnetic flow meter probes (model BL 613; Biotronex Laboratory Inc., Chester, MD) were positioned around the proximal part of the three coronary vessels: LAD,

circumflex coronary artery (CCX) and right coronary artery (RCA), respectively. The coronary blood flow was measured continuously throughout the experiments. Calibration of each probe was checked in vitro at the end of each experiment.

A round hole with a diameter of 5 cm was made in the middle of a phrenic nerve pad (Phrenic Nerve Pad[®]; Medtronic Inc.) and placed on top of the heart. The pad was stabilised to the surrounding myocardium with eight to ten sutures (Prolene 5-0; Ethicon Inc., Somerville, NJ, USA) and to the posterior sternal edges with sutures (Dermalon 2-0; Davis and Geck, St Louis, MO). A retractor was used throughout the experiments to keep the sternal edges apart. A polyurethane foam dressing, with an open pore structure of 400–600 μm (KCI, Copenhagen, Denmark), was placed between the sternal edges. The foam was continuously sutured to the surrounding skin (Dermalon 2-0; Davis and Geck). The wound was sealed with a transparent adhesive drape. A track pad (KCI) was inserted through the drape and was connected to a vacuum pump, (V.A.C. pump unit, KCI). When the negative pressure is applied, the heart will be drawn up towards the phrenic nerve pad and the foam without interfering with the sternal edges. This procedure causes the application of negative pressure to affect only the myocardium exposed by the 5-cm-diameter hole.

Experimental protocol

The coronary blood flow was measured continuously by electromagnetic flow meter probes. Recordings were made in normal myocardium before and while a negative pressure of -50 mmHg was applied.

The LAD was then occluded for 20 minutes with an elastic vessel loop. Coronary blood flow was then measured before and after 20 minutes of occlusion. A negative pressure of -50 mmHg was then applied to the myocardium, and coronary blood flow changes were recorded. The negative pressure was then removed.

Statistics

Calculations and statistical analysis were performed using GraphPad 4.0 software. Statistical analysis was performed using Student's paired *t*-test. Significance was defined as $P < 0.05$ and non significant (n.s.) at $P > 0.05$. Values are

presented as means \pm standard error of the mean.

RESULTS

Normal myocardium

A TNP of -50 mmHg induced an immediate, significant increase in total coronary blood flow in normal myocardium (171.3 ± 14.5 ml/minute before to 206.3 ± 17.6 ml/minute after TNP application, $P < 0.05$) (Figure 1). Divided between the three coronary arteries, -50 mmHg induced an immediate increase in local coronary blood flow in the CCX (49.2 ± 6.1 ml/minute before to 53.7 ± 5.0 ml/minute after TNP application, n.s. $P > 0.05$), the LAD (60.5 ± 11.1 ml/minute before to 77.8 ± 12.2 ml/minute after TNP application, $P < 0.05$) and the RCA (61.7 ± 11.6 ml/minute before to 74.8 ± 13.1 ml/minute after TNP application, $P < 0.05$) (Figure 2).

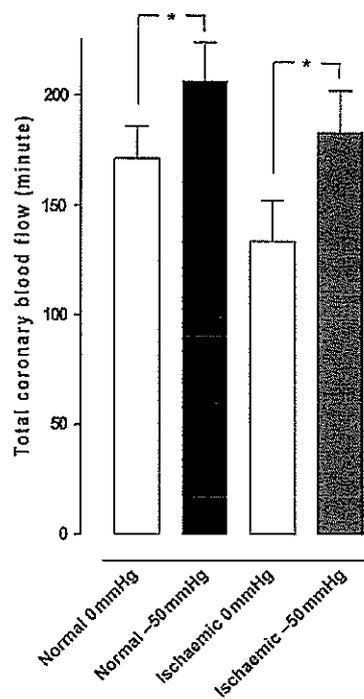


Figure 1. The total blood flow changes using coronary electromagnetic flow meter probes in normal and ischaemic myocardium before and during application of -50 mmHg. The measurements were performed at the proximal part of the right coronary artery, the left anterior descending artery and the circumflex coronary artery in six pigs. The change in total coronary artery blood flow is shown as mean values \pm standard error of the mean. Statistical analysis was performed using Student's paired *t*-test, and significance was defined as $*P < 0.05$ and not significant (n.s.) at $P > 0.05$.

Key Points

- a TNP of -50 mmHg induced an immediate, significant increase in total coronary blood flow in normal myocardium

Key Points

- TNP has become the therapy of choice for mediastinitis because of the exceptional clinical outcome
- TNP acts by a subatmospheric pressure application over the wound by controlled suction through a porous dressing
- the fundamental scientific mechanism of TNP is only partially understood
- one of the known effects of TNP is enhanced blood flow to the wound edge and granulation tissue formation; TNP increases blood flow velocity and opens up the capillary beds

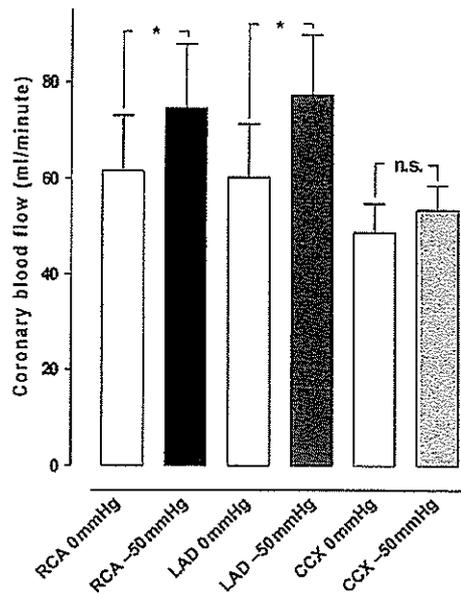


Figure 2. The blood flow measured using coronary electromagnetic flow meter probes in normal myocardium. The measurements were performed at the proximal part of the right coronary artery (RCA), left anterior descending artery (LAD) and circumflex coronary artery (CCX) in the myocardium in six pigs, with a topical negative pressure of -50 mmHg. The results are shown as mean values \pm standard error of the mean. Statistical analysis was performed using Student's paired t-test, and significance was defined as * $P < 0.05$ and not significant (n.s.) at $P > 0.05$. occl., occlusion.

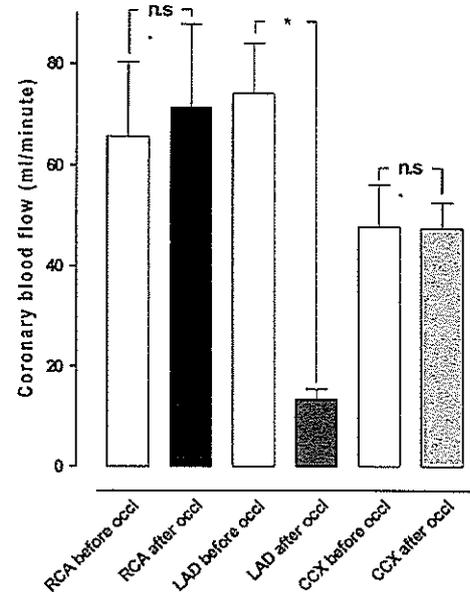


Figure 3. The blood flow measured using coronary electromagnetic flow meter probes before and after 20 minutes of left anterior descending artery (LAD) occlusion. The measurements were performed at the proximal part of the right coronary artery (RCA), LAD and circumflex coronary artery (CCX) in six pigs. Results are shown as mean values \pm standard error of the mean. Statistical analysis was performed using Student's paired t-test, and significance was defined as * $P < 0.05$ and not significant (n.s.) at $P > 0.05$.

LAD occlusion

Ischaemia was induced by occlusion of the LAD for 20 minutes. The coronary blood flow in the CCX increased (from 47.8 ± 8.4 ml/minute before to 56.3 ± 5.6 ml/minute after TNP application, n.s. $P > 0.05$). The coronary blood flow in the LAD decreased significantly (from 74.3 ± 9.8 ml/minute before to 13.3 ± 2.0 ml/minute, $P < 0.05$) after 20 minutes of LAD occlusion. The coronary blood flow in the RCA increased (from 65.8 ± 14.6 ml/minute before to 67.5 ± 14.7 ml/minute after TNP application, n.s. $P > 0.05$; Figure 3).

Ischaemic myocardium

A TNP of -50 mmHg induced an immediate, significant increase in total coronary artery blood flow in ischaemic myocardium (133.7 ± 18.4 ml/minute before to 183.2 ± 18.9 ml/minute after TNP application, $P < 0.05$; Figure 1). Application of -50 mmHg induced an immediate increase in local coronary blood flow in the CCX (52.2 ± 4.2 ml/minute before to 69.2 ± 7.0 ml/minute after TNP application,

n.s. $P > 0.05$). The coronary blood flow in the LAD significantly increased (from 15.0 ± 2.4 ml/minute before to 21.0 ± 7.9 ml/minute after TNP application, $P < 0.05$), and the coronary blood flow in RCA also significantly increased (from 66.2 ± 17.9 ml/minute before to 93.0 ± 15.7 ml/minute after TNP application, $P < 0.05$; Figure 4).

DISCUSSION

Poststernotomy mediastinitis is a rare but potentially lethal complication following cardiac surgery (17,19,25). Late TNP has become the therapy of choice for mediastinitis because of the exceptional clinical outcome (6,7,10,23). TNP acts by a subatmospheric pressure application over the wound by controlled suction through a porous dressing. However, the fundamental scientific mechanism of TNP is only partially understood. One of the known effects of TNP is enhanced blood flow to the wound edge and granulation tissue formation (1,2,15). TNP increases blood flow velocity and opens up the capillary beds. Mechanical forces

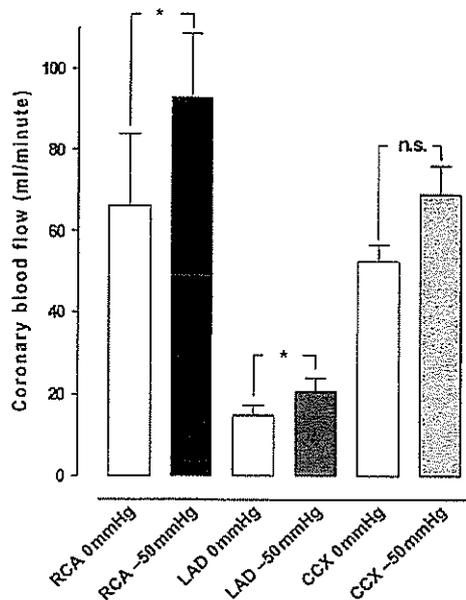


Figure 4. The blood flow measured using coronary electromagnetic flow meter probes in ischaemic myocardium. The measurements were performed at the proximal part of the right coronary artery (RCA), the left anterior descending artery (LAD) and the circumflex coronary artery (CCX) in the myocardium in six pigs, with a topical negative pressure of -50 mmHg. The results are shown as mean values \pm standard error of the mean. Statistical analysis was performed using Student's paired *t*-test, and significance was defined as $*P < 0.05$ and not significant (n.s.) at $P > 0.05$.

exerted by TNP and increased blood flow affect the cytoskeleton in the vascular cells, stimulating endothelial proliferation, capillary budding and angiogenesis, that is granulation tissue formation (14,16,26,27).

In patients with poststernotomy mediastinitis treated with TNP, the TNP is in direct contact with the heart, which is exposed through the diastase of the sternotomy. We have previously observed that these patients develop a richly vascularised granulation tissue over the heart within 6–7 days on the exposed surface of the heart. In the present study, we hypothesised that the application of TNP to the surface of the heart would stimulate neovascularisation and microvascular blood flow in the myocardium, as seen in skeletal muscle during TNP (13,28–31). It is commonly known among cardiothoracic surgeons that mediastinitis is a strong predictor of poor long-term survival after CABG, using conventional wound-healing techniques (such as closed irrigation, delayed wound closure or reconstructing with omentum or pectoral flaps).

It has been suggested that mediastinitis may cause negative long-term effects on several organs, such as the heart and kidneys (18,20,32). Previously, we have showed no difference in long-term survival between CABG patients with TNP-treated mediastinitis and CABG patients without mediastinitis (23). This indicates that these patients might have developed increased coronary collateral blood vessels during TNP and may therefore be better prepared when bypass grafts fail to work. It may be that the TNP stimulation of blood flow and development of collateral blood vessels in part account for the reduced long-term mortality in patients treated with TNP for poststernotomy mediastinitis after CABG.

We have earlier shown that TNP of -50 mmHg significantly increases microvascular blood flow in the underlying myocardial tissue (24). In the present study, we show that application of a TNP of -50 mmHg directly on the myocardium (over the LAD region) results in an increase of the total coronary blood flow not only in normal but also in ischaemic myocardium (Figure 4). An unchanged or decreased amount of the total coronary blood flow might theoretically cause ischaemia in parts of the myocardium not exposed to TNP. Because the present study shows a significant increase in the total amount of coronary blood flow, ischaemic areas are not likely to occur.

Therapeutic angiogenesis, wherein exogenous growth factors are administered to ischaemic tissue to enhance the reperfusion of these tissues, has been investigated as a potential therapy for patients with advanced coronary artery disease as an alternative to conventional treatment such as percutaneous coronary intervention (PCI) and CABG. It includes both protein and gene therapy, and they have both successfully induced angiogenic responses in animal studies, but optimal delivery of the angiogenic factor is difficult (33,34). However, vascular endothelial growth factor (VEGF) proteins have been shown to play a key role in the modulation of angiogenesis and vascular growth (35,36). Moreover, TNP produces a mechanical shear stress that is known to activate VEGF (37–41).

In the present study, we show that topically applied negative pressure of -50 mmHg over the LAD region results in a significant increase of the total amount of coronary blood flow to the heart muscle. This might in part explain the improved long-term outcome using TNP treatment

Key Points

- in patients with poststernotomy mediastinitis treated with TNP, the TNP is in direct contact with the heart, which is exposed through the diastase of the sternotomy
- we have previously observed that these patients develop a richly vascularised granulation tissue over the heart within 6–7 days on the exposed surface of the heart
- it may be that the TNP stimulation of blood flow and development of collateral blood vessels in part account for the reduced long-term mortality in patients treated with TNP for poststernotomy mediastinitis after CABG
- because the present study shows a significant increase in the total amount of coronary blood flow, ischaemic areas are not likely to occur

on patients with poststernotomy mediastinitis compared with conventional therapy presented in an earlier report (23).

ACKNOWLEDGEMENTS

We would like to thank Johan Ingemansson (Statistical Solutions IP) for his expert contribution to the statistic analyses. This study was supported by Anders Otto Swärd's Foundation/Ulrika Eklund's Foundation, Anna Lisa and Sven Eric Lundgren's Foundation for medical research, the Åke Wiberg Foundation, the M. Bergvall Foundation, the Swedish Medical Association, the Royal Physiographic Society in Lund, the Swedish Medical Research Council, the Crafoord Foundation, the Swedish Heart-Lung Foundation, the Swedish Government Grant for Clinical Research and the Swedish Hypertension Society.

REFERENCES

- 1 Morykwas MJ, Argenta LC, Shelton-Brown EJ, 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.
- 2 Argenta LC, Morykwas MJ. Vacuum-assisted closure: a new method for wound control and treatment: clinical experience. *Ann Plast Surg* 1997;38:563-76; discussion 77.
- 3 Armstrong DG, Lavery LA. Negative pressure wound therapy after partial diabetic foot amputation: a multicentre, randomised controlled trial. *Lancet* 2005;366:1704-10.
- 4 O'Connor J, Kells A, Henry S, Scalea T. Vacuum-assisted closure for the treatment of complex chest wounds. *Ann Thorac Surg* 2005;79:1196-200.
- 5 Brown KM, Harper FV, Aston WJ, O'Keefe PA, Cameron CR. Vacuum-assisted closure in the treatment of a 9-year-old child with severe and multiple dog bite injuries of the thorax. *Ann Thorac Surg* 2001;72:1409-10.
- 6 Gustafsson RI, Sjogren J, Ingemansson R. Deep sternal wound infection: a sternal-sparing technique with vacuum-assisted closure therapy. *Ann Thorac Surg* 2003;76:2048-53; discussion 53.
- 7 Sjogren J, Gustafsson R, Nilsson J, Malmso M, Ingemansson R. Clinical outcome after poststernotomy mediastinitis: vacuum-assisted closure versus conventional treatment. *Ann Thorac Surg* 2005;79:2049-55.
- 8 Gustafsson R, Johnsson P, Algotsson L, Blomquist S, Ingemansson R. Vacuum-assisted closure therapy guided by C-reactive protein level in patients with deep sternal wound infection. *J Thorac Cardiovasc Surg* 2002;123:895-900.
- 9 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.

- 10 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; discussion 600.
- 11 Wackenfors A, Sjogren J, Gustafsson R, Algotsson L, Ingemansson R, Malmso M. Effects of vacuum-assisted closure therapy on inguinal wound edge microvascular blood flow. *Wound Repair Regen* 2004;12:600-6.
- 12 Chen SZ, Li J, Li XY, Xu LS. Effects of vacuum-assisted closure on wound microcirculation: an experimental study. *Asian J Surg* 2005;28:211-7.
- 13 Wackenfors A, Gustafsson R, Sjogren J, Algotsson L, Ingemansson R, Malmso M. Blood flow responses in the peristernal thoracic wall during vacuum-assisted closure therapy. *Ann Thorac Surg* 2005;79: 1724-30; discussion 30-1.
- 14 Vandenberg HH. Mechanical forces and their second messengers in stimulating cell growth in vitro. *Am J Physiol* 1992;262:R350-5.
- 15 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; discussion 97-8.
- 16 Greene AK, Puder M, Roy R, Arsenault D, Kwei S, Moses MA, Orgill DP. Microdeformational wound therapy: effects on angiogenesis and matrix metalloproteinases in chronic wounds of 3 debilitated patients. *Ann Plast Surg* 2006;56:418-22.
- 17 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.
- 18 Loop FD, Lytle BW, Cosgrove DM, Mahfood S, McHenry MC, Goormastic M, Stewart RW, Golding LA, Taylor PC. J. 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; discussion 86-7.
- 19 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.
- 20 Braxton JH, Marrin CA, McGrath PD, Ross CS, Morton JR, Norotsky M, Charlesworth DC, Lahey SJ, Clough RA, O'Connor GT; Northern New England Cardiovascular Disease Study Group. Mediastinitis and long-term survival after coronary artery bypass graft surgery. *Ann Thorac Surg* 2000;70:2004-7.
- 21 Toumpoulis IK, Anagnostopoulos CE, Derose JJ Jr, Swistel DG. The impact of deep sternal wound infection on long-term survival after coronary artery bypass grafting. *Chest* 2005;127: 464-71.
- 22 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.

- 23 Sjogren J, Nilsson J, Gustafsson R, Malmso M, Ingemansson R. The impact of vacuum-assisted closure on long-term survival after post-sternotomy mediastinitis. *Ann Thorac Surg* 2005;80:1270-5.
- 24 Lindstedt S, Malmso M, Ingemansson R. Blood flow changes in normal and ischemic myocardium during topically applied negative pressure. *Ann Thorac Surg* 2007;84:568-73.
- 25 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.
- 26 Korff T, Augustin HG. Tensional forces in fibrillar extracellular matrices control directional capillary sprouting. *J Cell Sci* 1999;112(Pt 19):3249-58.
- 27 Chen CS, Mrksich M, Huang S, Whitesides GM, Ingber DE. Micropatterned surfaces for control of cell shape, position, and function. *Biotechnol Prog* 1998;14:356-63.
- 28 Petzina R, Gustafsson L, Mokhtari A, Ingemansson R, Malmso M. Effect of vacuum-assisted closure on blood flow in the peristernal thoracic wall after internal mammary artery harvesting. *Eur J Cardiothorac Surg* 2006;30:85-9.
- 29 Chen SZ, Cao DY, Li JQ, Tang SY. [Effect of vacuum-assisted closure on the expression of proto-oncogenes and its significance during wound healing]. *Zhonghua Zheng Xing Wai Ke Za Zhi* 2005;21:197-200.
- 30 Wackenfors A, Sjogren J, Algotsson L, Gustafsson R, Ingemansson R, Malmso M. The effect of vacuum-assisted closure therapy on the pig femoral artery vasomotor responses. *Wound Repair Regen* 2004;12:244-51.
- 31 Koller A, Kaley G. Endothelial regulation of wall shear stress and blood flow in skeletal muscle microcirculation. *Am J Physiol* 1991;260:H862-8.
- 32 De Feo M, Renzulli A, Ismeno G, Gregorio R, Della Corte A, Utili R, Cotrufo M. Variables predicting adverse outcome in patients with deep sternal wound infection. *Ann Thorac Surg* 2001;71:324-31.
- 33 Yau TM, Kim C, Li G, Zhang Y, Fazel S, Spiegelstein D, Weisel RD, Li RK. Enhanced angiogenesis with multimodal cell-based gene therapy. *Ann Thorac Surg* 2007;83:1110-9.
- 34 Kim C, Li RK, Li G, Zhang Y, Weisel RD, Yau TM. Effects of cell-based angiogenic gene therapy at 6 months: persistent angiogenesis and absence of oncogenicity. *Ann Thorac Surg* 2007;83:640-6.
- 35 Rosengart TK, Lee LY, Patel SR, Kligfield PD, Okin PM, Hackett NR, Isom OW, Crystal RG. Six-month assessment of a phase I trial of angiogenic gene therapy for the treatment of coronary artery disease using direct intramyocardial administration of an adenovirus vector expressing the VEGF121 cDNA. *Ann Surg* 1999;230:466-70; discussion 70-2.
- 36 Rosengart TK, Lee LY, Patel SR, Sanborn TA, Parikh M, Bergman GW, Hachamovitch R, Szulc M, Kligfield PD, Okin PM, Hahn RT, Devereux RB, Post MR, Hackett NR, Foster T, Grasso TM, Lesser ML, Isom OW, Crystal RG. Angiogenesis gene therapy: phase I assessment of direct intramyocardial administration of an adenovirus vector expressing VEGF121 cDNA to individuals with clinically significant severe coronary artery disease. *Circulation* 1999;100:468-74.
- 37 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.
- 38 Detmar M, Brown LF, Berse B, Jackman RW, Elicker BM, Dvorak HF, Claffey KP. Hypoxia regulates the expression of vascular permeability factor/vascular endothelial growth factor (VPF/VEGF) and its receptors in human skin. *J Invest Dermatol* 1997;108:263-8.
- 39 Shyu KG, Chang ML, Wang BW, Kuan P, Chang H. Cyclical mechanical stretching increases the expression of vascular endothelial growth factor in rat vascular smooth muscle cells. *J Formos Med Assoc* 2001;100:741-7.
- 40 Milkiewicz M, Mohammadzadeh F, Ispanovic E, Gee E, Haas TL. Static strain stimulates expression of matrix metalloproteinase-2 and VEGF in microvascular endothelium via JNK- and ERK-dependent pathways. *J Cell Biochem* 2007;100:750-61.
- 41 Seko Y, Seko Y, Takahashi N, Shibuya M, Yazaki Y. Pulsatile stretch stimulates vascular endothelial growth factor (VEGF) secretion by cultured rat cardiac myocytes. *Biochem Biophys Res Commun* 1999;254:462-5.

Pressure transduction to the thoracic cavity during topical negative pressure therapy of a sternotomy wound

Christian Torbrand, Richard Ingemansson, Lotta Gustafsson, Per Paulsson, Malin Malmjö

Torbrand C, Ingemansson R, Gustafsson L, Paulsson P, Malmjö M. Pressure transduction to the thoracic cavity during topical negative pressure therapy of a sternotomy wound. *Int Wound J* 2008;5:579–584.

ABSTRACT

The present study was performed to examine pressure transduction to the thoracic cavity during topical negative pressure (TNP) therapy of a sternotomy wound. Seven pigs underwent median sternotomy. Pressure transduction catheters were placed on the anterior surface of the heart (under the foam), in the pericardium (under the heart), in the left pleura and in the oesophagus at the level of the heart. The wound was sealed as for TNP therapy. The vacuum source was set to deliver negative pressures between -50 and -200 mmHg. The pressure on the anterior surface of the heart changed in a linear relationship with the applied negative pressure and was slightly lower than the applied negative pressure (-102 ± 9 mmHg at delivered -125 mmHg). Further down in the thoracic cavity, in the pericardium (under the heart), in the left pleura and in the oesophagus, the wound pressure was only slightly affected by TNP therapy. In conclusion during TNP therapy, negative pressure is effectively transmitted to anterior portions of the heart. This may explain our recent findings that TNP increases microvascular blood flow in the myocardium. The pressure difference between the anterior and the posterior portions of the heart causes the right ventricle to be sucked up towards the posterior parts of the sternum, where it might be exposed to the sharp edges of the sternal bone, which may result in heart injury.

Key words: Experimental surgery • Mediastinal infection • Wound healing

INTRODUCTION

Cardiac surgery is complicated by poststernotomy mediastinitis in 1–5% of all procedures (1), and this is a potentially life-threatening complication (2). The reported early mortality in poststernotomy mediastinitis following coro-

nary bypass surgery grafting is between 8% and 25% (3,4). Established treatment of poststernotomy mediastinitis includes surgical debridement, drainage, irrigation and reconstruction using pectoral muscle flap or omentum transposition. In 1999, Obdeijn *et al.* described a new mode of treatment for poststernotomy mediastinitis using a vacuum-assisted closure technique (5), which is based on the principle of applying subatmospheric pressure by controlled suction to a sealed, airtight wound through a porous dressing. The technique, also known as topical negative pressure (TNP) therapy, has resulted in excellent clinical outcome (1–5). Scientific evidence regarding the mechanisms by which TNP promotes wound healing has started to emerge. The suction force

Key Points

- scientific evidence regarding the mechanisms by which TNP promotes wound healing has started to emerge

Authors: C Torbrand, MD, Department of Medicine, Lund University Hospital, Lund, Sweden; R Ingemansson, MD, PhD, Department of Cardiothoracic Surgery, Lund University Hospital, Lund, Sweden; L Gustafsson, MSc, PhD, Department of Medicine, Lund University Hospital, Lund, Sweden; P Paulsson, MD, Department of Cardiothoracic Surgery, Lund University Hospital, Lund, Sweden; M Malmjö, MD, PhD, Department of Medicine, Lund University Hospital, Lund, Sweden

Address for correspondence: M Malmjö, MD, PhD, Vascular Research, Lund University, BMC A13, SE-221 84 Lund, Sweden

E-mail: malin.malmjo@med.lu.se

Key Points

- the present study was designed to examine to which extent negative pressure is transduced to the anterior portions of the heart and also to deeper parts of the thoracic cavity
- a porcine sternotomy wound model was used

created by TNP therapy enables the drainage of excessive fluid and debris, which leads to the removal of wound oedema, reduction in bacterial count and enhanced granulation tissue formation (6–9). Knowledge of the effects of TNP in a sternotomy wound is limited (10).

It is known that blood flow to the muscle tissue of the wound edge is increased by TNP therapy (7–9). We recently show that TNP of a sternotomy wound stimulates blood flow to the myocardium (10). The present study was designed to examine to which extent negative pressure is transduced to the anterior portions of the heart and also to deeper parts of the thoracic cavity. A porcine sternotomy wound model was used, and pressure was recorded on the anterior portions of the heart, in the pericardium (behind the heart), in the left pleura and in the oesophagus during the application of TNP therapy at pressures between -50 and -200 mmHg.

MATERIALS AND METHODS

Animals

A porcine sternotomy wound model was used. Seven domestic landrace pigs with a mean weight of 70 kg were fasted overnight with free access to water. The study was approved by the Ethics Committee for Animal Research, Lund University, Sweden. The investigation complied with the 'Guide for the Care and Use of Laboratory Animals' as recommended by the US National Institutes of Health and published by the National Academies Press (1996). The anaesthesia was induced, and the surgical procedure was performed as previously described (11).

Anaesthesia and surgical preparation

An intramuscular injection of ketamine (Ketaminol vetTM 100 mg/ml; Farmaceutici Gellini S.p.A, Aprilia, Italy) 15 mg/kg body weight, in combination with midazolam (Dormicum 1 mg/ml; Roche, Stockholm, Sweden) and xylazine (Rompun vetTM 20 mg/ml; Bayer AG, Leverkusen, Germany) 2 mg/kg, was used for premedication. Anaesthesia was induced by continuous intravenous infusion of propofol (DiprivanTM 20 mg/ml; AstraZeneca, Södertälje, Sweden) at a dosage of 0.1–0.2 mg/kg/minute in combination with intermittent fentanyl (LeptanalTM; Lilly, France) and atracurium besylate (TracriumTM; Glaxo, Täby, Sweden) at doses of 0.02 µg/kg and 0.2–0.5 mg/kg, respectively. Before surgery, a trache-

otomy (Portex endotracheal tube 7.5 mm internal diameter) was performed.

A ventilator (Servo-Ventilator 900; Elema-Schönander, Stockholm, Sweden) was used for mechanical ventilation. The same settings were used for all animals: volume-controlled, pressure-regulated ventilation, 8.5 l/minute, 15 breaths/minute and an inhaled oxygen fraction of 35%. A lower midline abdominal incision was made over the urinary bladder. The urinary bladder was exposed, and a urinary catheter (Silicone Foley Catheter; Tyco Healthcare, Tullamore, Ireland) was inserted, sutured and connected to a urinary bag (Unomedical a/s, Haarlev, Denmark). The abdominal incision was continuously sutured with Dermalon 2.0 (Davis-Geck, Hampshire, UK).

A midline sternotomy was performed. The tip of a saline-filled pressure catheter was placed in the left pleura, in the pericardium (under the heart) and on the surface of the heart (under foam) through the sternotomy wound. A fourth pressure transduction catheter was inserted into the oesophagus, through the mouth, so that the tip was positioned at the level of the heart. The pressure catheters were connected to a calibrated custom-built pressure gauge. For probe positions, see Figure 1. A layer of polyurethane foam (KCI, Copenhagen, Denmark) was placed between the sternal edges. A second layer of foam was placed over the first layer and secured to the surrounding skin. The wound was sealed with a transparent adhesive drape (KCI), and the

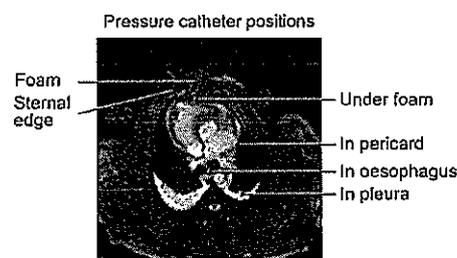


Figure 1. Magnetic resonance image illustrating a cross-section of the pig thoracic cavity with an open sternotomy. First, the pressure transduction catheters were placed on the anterior surface of the heart (under the foam), in the pericardium (under the heart) and in the left pleura. A polyurethane foam dressing was placed between the sternal edges, and non collapsible drainage tubes were connected to the foam. The open wound was sealed with transparent adhesive drape, and drainage tubes are connected to a purpose-built vacuum source. A fourth pressure transduction catheter was placed in the oesophagus at the level of the heart.

two evacuation tubes connected the foam with the vacuum source (V.A.C.[®] pump unit; KCI).

Experimental protocol

Wound pressures were recorded on the anterior surface of the heart (under the foam), in the pericardium (under the heart), in the left pleura and in the oesophagus. Wound pressures were measured both with the respirator on and with the respirator turned off at the end of inspiration. In order to eliminate systemic errors, both the sequence of applying negative pressure (-50, -75, -100, -125, -150, -175 and -200 mmHg) and the order in which the experiments were performed with the respirator turned on and off were varied between the animals in a randomised design.

Calculations and statistics

The experiments were performed on seven pigs. Calculations and statistical analysis were performed using GraphPad Prism[®] 4.0 software

(GraphPad Software Inc, San Diego, CA). Statistical analysis was performed using Kruskal-Wallis test with Dunn's test for multiple comparisons. Significance was defined as $P < 0.05$. Values are presented as means \pm SEM.

RESULTS

TNP was applied to the sternotomy wound at negative pressures between -50 and -200 mmHg. The pressure over the anterior surface of the heart (under the foam) changed in a linear relationship with the applied negative pressure and was slightly lower than the applied negative pressure (-102 \pm 9 mmHg in the wound at delivered -125 mmHg; Figures 2 and 3). The pressure difference between the wound space and the vacuum source was larger at higher negative pressures (-39 \pm 3 mmHg in the wound at delivered -50 mmHg and -163 \pm 12 mmHg at delivered -40 mmHg).

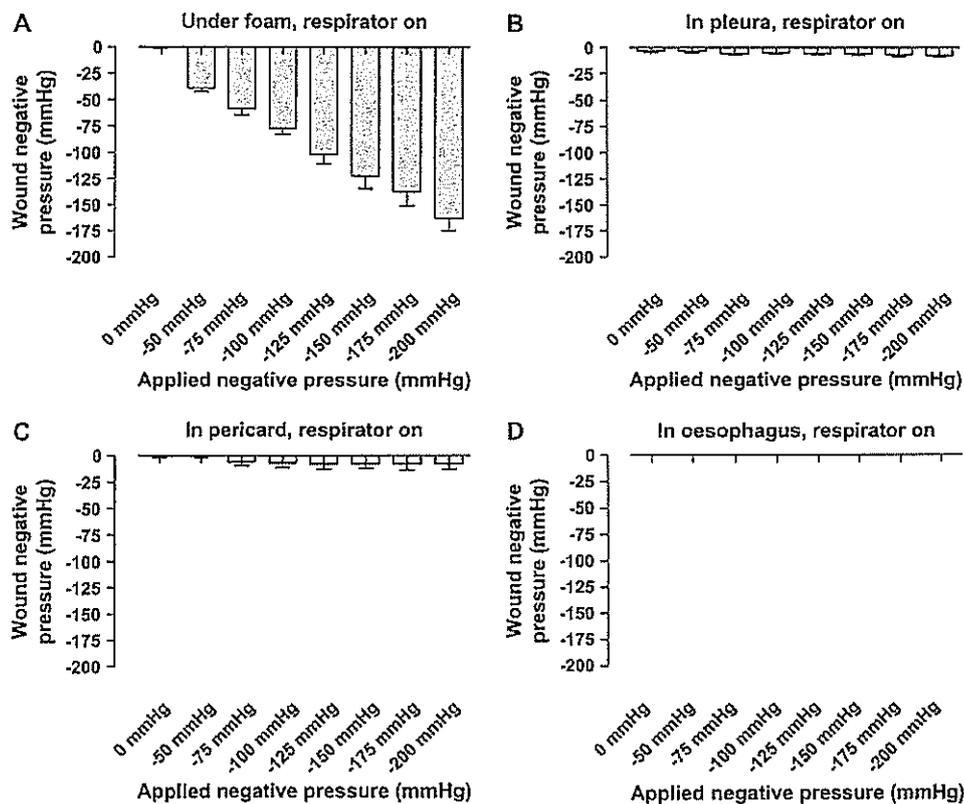


Figure 2. Wound pressures measured with the respirator on. The vacuum source was set to deliver negative pressures between -50 and -200 mmHg. Wound pressures were recorded using pressure transduction catheters on (A) the anterior surface of the heart (under the foam), (B) in the pericardium (under the heart), (C) in the left pleura and (D) in the oesophagus. Values are presented as means \pm SEM from seven experiments. Note how the pressure under the foam changed in a linear relationship with the applied negative pressure, while the pressure further down in the thoracic cavity was not altered.

Key Points

- TNP is a recently introduced technique that promotes the healing of difficult wounds, including poststernotomy mediastinitis
- we show that the pressure during TNP therapy is effectively delivered to the anterior portions of the heart

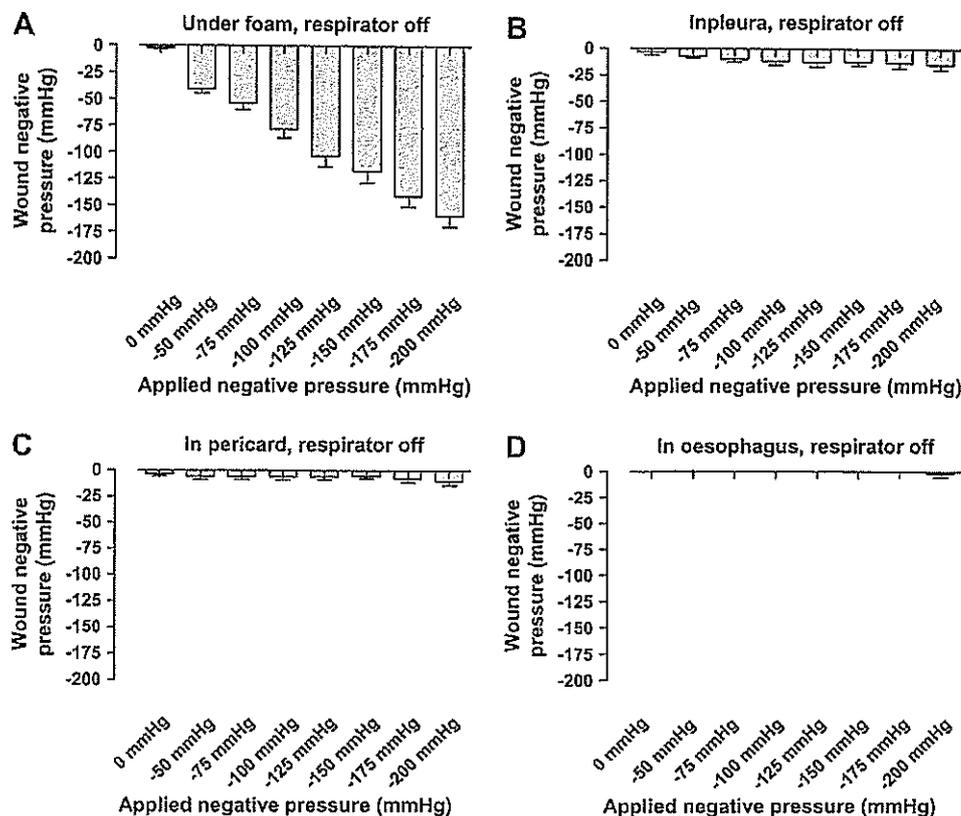


Figure 3. Wound pressures measured after the respirator had been turned off at the end of inspiration. The vacuum source was set to deliver negative pressures between -50 and -200 mmHg. Wound pressures were recorded using pressure transduction catheters on (A) the anterior surface of the heart (under the foam), (B) in the pericardium (under the heart), (C) in the left pleura and (D) in the oesophagus. Values are presented as means \pm SEM from seven experiments. Note that the pressure recordings are similar to those with the respirator on.

Further down into the thoracic cavity, in the pericardium (under the heart), in the left pleura and in the oesophagus, the wound pressure was not significantly affected by TNP therapy (Figures 2 and 3). The pressure recordings were similar with the respirator turned on as with the respirator turned off at the end of inspiration (Figures 2 and 3).

DISCUSSION

Mediastinitis is a devastating complication in open heart surgery. TNP is a recently introduced technique that promotes the healing of difficult wounds, including poststernotomy mediastinitis (4,5,8). The technique entails application of a negative pressure to a sealed, airtight wound. The suction force created by TNP therapy is known to stimulate blood flow to the wound edge and facilitate the drainage of excessive fluid and debris, which leads to the removal of

wound oedema, reduction in bacterial count and enhanced granulation tissue formation (6–9). Scientific evidence regarding the effects in a sternotomy wound is still limited (10).

The present study was performed to explore to which extent negative pressure is transduced to the anterior portions of the heart and also to deeper locations in the sternotomy wound. We show that the pressure during TNP therapy is effectively delivered to the anterior portions of the heart. This may explain our recent findings that TNP at -50 mmHg increases microvascular blood flow in the myocardium (10). The blood flow effects by TNP are believed to be through mechanical stress and a pressure gradient across the tissue, which causes a surge of blood to the area (8,9,12–14). TNP is known to stimulate blood flow to the wound edge that is exposed to negative pressure, while tissue further from the vacuum source remains unaffected (8,9). This is in accordance with the

current findings that pressure is not transduced far beyond the locations of the open-pore structure dressings of TNP therapy, for example to the pericardium (behind the heart), the pleura or the oesophagus.

The present results show negative pressure on the anterior portions of the heart, at similar levels as set on the vacuum source, while there is no negative pressure beneath the heart. This pressure difference may explain the finding that, upon the delivery of negative pressure, the anterior portion of the right ventricle is sucked up towards the anterior thoracic wall. The pressure causes the right ventricle to bulge into the space between the sternal edges, which deforms the anterior portion of the heart (15). We believe that this may have two negative effects.

First, pressure on the right ventricle may mechanically hinder venous return and cardiac pumping. Indeed, cardiac output and end diastolic volume are known to be slightly decreased upon application of negative pressure (15). Interestingly, interposition of four layers of paraffin gauze dressing over the heart during TNP therapy resulted in a lesser decrease in cardiac output (15). It is known that the interface dressings prevent the delivery of TNP (16).

Second, the pressure difference between the anterior and the posterior portions of the heart causes the right ventricle to be sucked up towards the posterior parts of the sternum where it might be exposed to the sharp edges of the sternal bone. This may result in right ventricle rupture, which is an uncommon but feared complication of TNP therapy in post-sternotomy mediastinitis (17). Development of TNP therapy for sternotomy wounds by facilitating pressure transduction to the bottom of the wound may be beneficial in hindering the strong suction force on the heart. It is known that motion between the sternal edges in combination with adherent heart structures to the thoracic wall are factors that predispose for heart rupture. It is important to use surgical techniques to minimise these risk factors (18). In summary, adherences below the sternal edges must be released, and three or four layers of interface dressing should be placed over the anterior portions of the heart in order to cover and protect visible parts of the right ventricle from the sternal edges. The interface dressing reduces the formation of adherences between the sternum and the right

ventricle, and the paraffin content facilitates movement.

In conclusion, the pressure over the anterior surface of the heart (under the foam) changed in a linear relationship with the applied negative pressure. These results may explain the positive effects on myocardial blood flow by TNP (10). Further down into the thoracic cavity, in the pericardium (under the heart), in the left pleura and in the oesophagus, the wound pressure was not affected by TNP therapy. The effect of negative pressure can therefore only be anticipated to be effective in superficial parts of the wound, for example on the sternotomy wound edges and on the anterior surface of the heart and not in deeper parts of the wound. The pressure difference between the anterior and the posterior portions of the heart during TNP therapy causes the right ventricle to be sucked up towards the posterior parts of the sternum where it might be exposed to the sharp edges of the sternal bone, which may result in heart injury.

ACKNOWLEDGEMENTS

We thank Martin Ugander, MD, PhD, for valuable contribution to the manuscript. This study was supported by the Anders Otto Swärds Foundation/Ulrika Eklunds Foundation, Anna Lisa and Sven Eric Lundgrens Foundation for Medical Research, Åke Wiberg Foundation, the M. Bergvall Foundation, the Swedish Medical Association, the Royal Physiographic Society in Lund, the Swedish Medical Research Council, the Crafoord Foundation, the Swedish Heart-Lung Foundation, the Swedish Government Grant for Clinical Research and the Swedish Hypertension Society.

REFERENCES

- 1 Raudat CW, Pagel J, Woodhall D, Wojtanowski M, Van Bergen R. Early intervention and aggressive management of infected median sternotomy incision: a review of 2242 open-heart procedures. *Am Surg* 1997;63:238-41; discussion 241-2.
- 2 El Oakley RM, Wright JE. Postoperative mediastinitis: classification and management. *Ann Thorac Surg* 1996;61:1030-6.
- 3 Crabtree TD, Codd JE, Fraser VJ, Bailey MS, Olsen MA, Damiano RJ Jr. 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.

Key Points

- the effect of negative pressure can therefore only be anticipated to be effective in superficial parts of the wound, for example on the sternotomy wound edges and on the anterior surface of the heart and not in deeper parts of the wound
- the pressure difference between the anterior and the posterior portions of the heart during TNP therapy causes the right ventricle to be sucked up towards the posterior parts of the sternum where it might be exposed to the sharp edges of the sternal bone, which may result in heart rupture

- 4 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.
- 5 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.
- 6 Sjogren J, Gustafsson R, Nilsson J, Malmso M, Ingemansson R. Clinical outcome after poststernotomy mediastinitis: vacuum-assisted closure versus conventional treatment. *Ann Thorac Surg* 2005;79:2049-55.
- 7 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.
- 8 Wackenfors A, Gustafsson R, Sjogren J, Algotsson L, Ingemansson R, Malmso M. Blood flow responses in the peristernal thoracic wall during vacuum-assisted closure therapy. *Ann Thorac Surg* 2005;79:1724-30; discussion 1730-1.
- 9 Wackenfors A, Sjogren J, Gustafsson R, Algotsson L, Ingemansson R, Malmso M. Effects of vacuum-assisted closure therapy on inguinal wound edge microvascular blood flow. *Wound Repair Regen* 2004;12:600-6.
- 10 Lindstedt S, Malmso M, Ingemansson R. Blood flow changes in normal and ischemic myocardium during topically applied negative pressure. *Ann Thor Surg* 2007;84:568-573.
- 11 Petzina R, Gustafsson L, Mokhtari A, Ingemansson R, Malmso M. Effect of vacuum-assisted closure on blood flow in the peristernal thoracic wall after internal mammary artery harvesting. *Eur J Cardiothorac Surg* 2006;30:85-9.
- 12 Chen SZ, Li J, Li XY, Xu LS. Effects of vacuum-assisted closure on wound microcirculation: an experimental study. *Asian J Surg* 2005;28:211-7.
- 13 Fabian TS, Kaufman HJ, Lett ED, Thomas JB, Rawl DK, Lewis PL, Summitt JB, Merryman JI, Schaeffer TD, Sargent LA, Burns RP. The evaluation of subatmospheric pressure and hyperbaric oxygen in ischemic full-thickness wound healing. *Am Surg* 2000;66:1136-43.
- 14 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; discussion 1600.
- 15 Petzina R, Ugander M, Gustafsson L, Engblom H, Sjogren J, Hetzer R, Ingemansson R, Arheden H, Malmso M. Hemodynamic effects of vacuum-assisted closure therapy in cardiac surgery, assessment using magnetic resonance imaging. *J Thorac Cardiothorac Surg* 2007;133:1154-1162.
- 16 Jones SM, Banwell PE, Shakespeare PG. Interface dressings influence the delivery of topical negative-pressure therapy. *Plast Reconstr Surg* 2005;116:1023-8.
- 17 Abu-Omar Y, Naik MJ, Catarino PA, Ratnatunga C. Right ventricular rupture during use of high-pressure suction drainage in the management of poststernotomy mediastinitis. *Ann Thorac Surg* 2003;76:974; author reply 974-5.
- 18 Gustafsson RI, Sjogren J, Ingemansson R. Deep sternal wound infection: a sternal-sparing technique with vacuum-assisted closure therapy. *Ann Thorac Surg* 2003;76:2048-53; discussion 2053.

Primary or Delayed Closure for the Treatment of Poststernotomy Wound Infections?

Tatjana M. Fleck, MD,* Rupert Koller, MD,† Pietro Giovanoli, MD,† Reinhard Moidl, MD,* Martin Czerny, MD,* Michael Fleck, CM,* Ernst Wolner, MD,* and Martin Grabenwoger, MD*

Objective: The methods of primary versus delayed wound closure for the treatment of sternal wound infections after cardiac surgery were retrospectively compared.

Methods: From January 2001 to March 2003, 132 patients (median age 66 years, male to female ratio 88:44) with sternal wound infection after cardiac surgery were treated at our department. After thorough debridement, 35 patients received preconditioning of the wound before implementation of definitive therapy; the remainder (97 patients) were treated with immediate closure.

Results: From the 35 patients with preconditioning, 19 patients proceeded to delayed primary closure, whereas the remaining 14 patients were referred to plastic reconstruction with a pectoralis muscle flap. Primary success rate in this group was 100%.

In the immediate primary closure group, 33 patients experienced 1 or more therapy failures, resulting in a recurrence rate of 39%. Fifteen patients received a pectoralis muscle flap as definite treatment modality.

Conclusions: Immediate primary closure is associated with a high rate of local infection recurrence. Surgical debridement and conditioning of the wound until resolution of infections with delayed primary closure or plastic reconstruction is suggested as the more appropriate treatment modality, with promising results.

(*Ann Plast Surg* 2004;52: 310–314)

Surgical site infections are the most common nosocomial infections among surgical patients. Deep sternal wound infections, although not frequent, are encountered with an incidence between 0.5% and 5% and a median mortality rate

of 17%. However, the impact of these infections on patient morbidity and hospital costs are tremendous so that every effort has to be undertaken to reduce this devastating complication after cardiac surgery.^{1–3} Although many authors as Mandelbaum and Schumaker in 1963⁴ with closed mediastinal irrigation and Jurkiewicz et al⁵ in 1980 with the use of muscle flaps for reconstruction have investigated several treatment modalities, none has been able to substantially reduce the mortality and the associated increase of costs and prolonged hospital stay of sternal wound infections.^{6–10}

However, despite the presence of these various treatment modalities, no consensus exists so far regarding the optimum management for sternal wound infections.

Herein, we describe our experience with preconditioning of the wound compared with conventional debridement and immediate primary closure.

MATERIALS AND METHODS

Patient Collective

One hundred thirty-two patients with a male to female ratio of 88 to 44 and a median age of 66 years (ranging from 33 to 82 years) sustained poststernotomy wound infection during the time period of January 2001 to March 2003 at our department.

Operation preceding sternal wound infection was coronary artery bypass grafting in 73%, valve replacement or reconstruction in 31%, replacement of the ascending thoracic aorta in 8%, and heart transplantation in 3%. In 21% of the patients, more than 1 procedure was concomitantly performed.

Pre-existing comorbidities were similar in both groups and were comparable with a standard Caucasian population requiring adult heart surgery (median Euroscore of 6 points, ranging from 0 to 15 points).

Routine antibiotic prophylaxis consisted of a cephalosporin (Cefazoline) 30 minutes before skin incision and 4 hours and 8 hours thereafter.

Intensive care unit (ICU) stay after the primary cardiac procedure was median 2 days (ranging from 1 to 14 days).

Received June 27, 2003 and accepted for publication, after revision, August 4, 2003.

From the *Department of Cardiothoracic Surgery, University of Vienna, AKH Vienna, 1090 Vienna, Austria; and †Department of Plastic and Reconstructive Surgery, University of Vienna, AKH Vienna, 1090 Vienna, Austria.

Reprints: Tatjana Fleck MD, Department of Cardiothoracic Surgery, University of Vienna, AKH Vienna, Leitstelle 20A, Währinger Gürtel 18–20, 1090 Vienna, Austria. E-mail: t9204604@hotmail.com

Copyright © 2004 by Lippincott Williams & Wilkins

ISSN: 0148-7043/04/5203-0310

DOI: 10.1097/01.sap.0000105524.75597.c0

Median onset of infection was on average after 14 days (ranging from 5 to 72 days). Interestingly we observed 2 patterns of infection presentation so that the median start after 14 days has to be further specified into an early presentation group with a median infection start between postoperative day 5 and 9 and a late presentation group with a median infection start between postoperative day 14 and 72.

A sternal infection was defined when 1 or more of the following signs were present and other origins of infection were excluded:

- serous or purulent drainage from the incision site with or without partial disruption of the incision

- sternal pain

- sternal instability (positive bimanual sternal compression test)

- elevated or elevating infection parameters (acute phase protein, leukocytes)

- fever, chills, lethargy

- erythematous skin surrounding the incision

Antibiotic therapy was first directed towards *Staphylococcus epidermidis* as the most offending bacterium. After obtaining the results from the cultures, antibiotics were adapted when appropriate.

Wound Classification

Wound classification was defined according to the suggestions of El Oakley and Wright.¹⁹ Oakley classification type 2A (superficial infection involving only the subcutis) was found in 27 patients (24%), whereas Oakley type 2B (deep infection with sternal instability through involvement of the sternal bone and mediastinitis) was present in the remaining 85 patients (95%).

Compared with our patient collective, we identified 73 patients (55%) with an infection start within 14 days after surgery; in 24 patients (18%), mediastinitis occurred after 1 or more failed therapeutic trial.

Treatment Protocols

Whenever a patient was in suspicion of having a sternal wound infection, treatment was straightforward, with opening of the wound in the operating theater. Therefore, all patients underwent opening and inspection of the sternal wound under aseptic conditions in the operating theater. When the infection was clinically confirmed by the attending surgeon, the viability of the sternal bone was checked. If an involvement of the sternal bone or sternal instability was evident, wire removal was indicated. After careful evaluation of the mediastinum, bacterial cultures, as well as bone biopsies, were routinely taken. Subsequently aggressive debridement with resection of all nonviable tissue and irrigation with Povidone-iodine solution and H₂O₂ was performed.

In the debridement only group, rewiring and primary wound closure with single suture layers was performed after

insertion of a 32 French mediastinal drain. Subsequent dressing changes were done on a daily base. The drain was removed when culture results taken from the drainage fluid were negative.

In the preconditioning group, the vacuum-assisted closure (VAC) system was used with continuous therapy mode and 125 mm Hg of suction. Every 48 to 72 hours, the VAC system was changed under aseptic conditions in the operating theater. Details of our VAC implantation technique were previously described in detail elsewhere.¹⁸ Requirements for VAC removal and employment of definitive surgery were as follows: decline of serological inflammation parameters, less than 100,000 CFU per gram of tissue in bacteriological cultures, and resolution of local infection signs in the wound.

RESULTS

A significant difference between the 2 treatment modality preconditioning versus primary closure could be observed. Whereas all 35 patients with pretreatment had a primary success rate of 100%, 35 patients (36%) out of the 97 patients with debridement, rewiring, and closed drainage sustained 1 or more therapy failures.

Furthermore, after several failed attempts (median attempts 2, ranging from 1 to 4) to primary closure 15 patients proceeded to plastic reconstruction with a pectoralis muscle flap.

In the preconditioning group, the decision between muscle flap or direct closure mainly depended on an involvement of the sternal bone, as well as on the amount of viable tissue to reach a tension-free wound closure to secure a regular healing process. In case of irregular healing or necrotic sternal bone after VAC treatment, muscle flap reconstruction was mandatory to achieve long-term sternal stability. Primary closure was feasible in 19 cases (54%), whereas the remainder (14 patients, 40%) mandated muscle flap reconstruction. Results are depicted in Table 1.

Removal of the VAC system was done after a mean of 10 days after implementation (ranging from 4 to 15 days), and on average 3 dressing changes (ranging from 1 to 13) were necessary until the wound was amenable for definitive therapy.

Patients of both groups received a prolonged course of culture-dependent intravenous antibiotics (mainly Trimethoprim and Teicoplanin) for an average of 10 days (ranging from 7 to 21 days). Antibiotics were discontinued on average 10 days after the patient received definitive treatment in the VAC group.

In-hospital mortality for the entire cohort was 7% and 30-day survival was 93%. Median hospital stay ranged from 7 to 45 days (median 19 days) in the preconditioning group and from 5 to 72 days (median 24 days) in the immediate closure group.

TABLE 1. Patient Treatment Modality and Outcome

Wound infection type		
Superficial (no bone involved)	34 pat	26%
Deep (sternal instability)	74 pat	74%
Surgical therapy		
Surgical debridement	97 pat	73%
Delayed closure	35 pat	26%
Pectoralis muscle flap without pretreatment	15 pat	15%
Pectoralis muscle flap with pretreatment	14 pat	40%
Secondary closure after preconditioning	19 pat	60%
Outcome		
Therapy failure after primary closure	35 pat	36%
Therapy failure after VAC	0 pat	0%
Culture results		
<i>Staphylococcus epidermidis</i>	83 pat	64%
<i>Staphylococcus aureus</i>	9 pat	7%
<i>Enterococcus fecalis</i>	16 pat	12%
Methicillin resistant <i>Staphylococcus aureus</i>	16 pat	12%
More than 1 bacterium present	26 pat	20%

Bacterial cultures isolated *Staphylococcus epidermidis* in 83 patients (63%), *Staphylococcus aureus* in 9 patients (7%), *Enterobacter fecalis* in 16 patients (12%), and Methicillin-resistant *Staphylococcus aureus* in 16 patients (12%). More than 1 specimen of bacteria was present in 26 patients (20%).

Follow-up period ranged from 1 to 30 months. Therapy failure occurred in 35 patients (36%) with surgical debridement and primary closure, whereas no recurrence of infection was noted in the 35 patients with pretreated wounds.

DISCUSSION

Surgical management of sternal wound infections depends mainly on the severity of the infection and entails traditionally debridement, rewiring, and closed drainage with or without antibiotic irrigation. However, the results of this traditional approach are unsatisfactory, with recurrence of local infection as high as 50%. Table 2 gives an overview over the current literature. But despite these well-known complication rates and the compelling evidence from alternative treatment modalities as delayed primary closure, pretreatment with the VAC system, and immediate or delayed plastic reconstruction, the surgical community is reluctant to adapt to this novel concept, even though the fact that sternal wound infections can be cost intensive and time consuming particularly when the primary therapy fails.²²

To diminish these high recurrence rates associated with immediate closure, various alternatives were developed during the last decades as plastic reconstruction with muscle flaps or the use of the greater omentum. (Table 3) Furthermore, refinements

TABLE 2. Literature Overview for Immediate Primary Closure

1. Tegnell et al [27]
Debridement and closed drainage, recurrence rate 33%
2. Francel et al [6]
Debridement and primary closure, recurrence rate 35%
3. Rand et al [7]
Closed irrigation, recurrence rate 88.2%
4. De Feo et al. [2]
Debridement and closed irrigation, recurrence rate 16%
5. Catarino et al [25]
Primary closure and irrigation, recurrence rate 50%
6. Berg et al [28]
Closed continuous or suction drainage, recurrence rate 5%

TABLE 3. Literature Overview for Delayed Closure

1. Zacharias et al [26]
Wound debridement, open packing with Povidone iodine until resolution of infection and then secondary closure, recurrence rate 2.4%
2. Lindsey [23]
Debridement and open packing until resolution and then muscle flap, recurrence rate 12%
3. Brandt et al [22]
Same as 2, recurrence rate 0%
4. Fleck et al [18]
Debridement and VAC system until resolution and then primary closure or muscle flap, recurrence rate 0%
5. Gustafsson et al [17]
Debridement and VAC system until resolution and then primary closure, recurrence rate 0%
6. Francel et al [6]
Debridement and secondary closure or muscle flap, recurrence rate 2%
7. Rand et al [7]
Immediate muscle flap or delayed muscle flap, recurrence rate 0%
8. Jones et al [9]
Primary muscle flap closure, recurrence rate 3.8%
9. El Gamel et al [10]
Primary muscle flap closure, recurrence rate 1%
10. Catarino et al [25]
Debridement and VAC system, and then delayed primary closure, recurrence rate 0%
11. Gustafsson et al [17]
VAC system followed by delayed primary closure, recurrence rate 0%

of traditional approaches were developed as delayed primary closure and high-pressure suction drainage.^{5,6,23-25}

In summary, compelling evidence exists, supported by various recent publications, to delay definitive therapy, regardless if primary closure or plastic reconstruction will be

attempted, until the wound situation has been optimized.^{18,23} We observed a significantly shorter ICU stay after muscle flap closure for patients having been treated with the VAC system (median stay 1 day, ranging from 1 to 4 days) as compared with patients without VAC system pretreatment (median stay 9.5 days, ranging from 4 to 26 days).¹⁸

The reason for this might be associated with the better overall condition and the improved wound situation with a consecutive reduction in bacterial colonization of patients pretreated with the VAC system before definitive surgery. Therefore, the risk of sepsis due to swept bacteria into the circulation is markedly diminished.

Initial successful reports using the vacuum assisted closure system for the management of sternal wound encouraged the authors to use the VAC System as first-line therapy for the treatment of poststernotomy wound infections since November 2001.¹¹⁻¹⁸

Two distinct patterns of initiation of infection were mainly observed in our study population, which is in accordance with a recent publication by Francel and Kouchoukos.⁶ The first infection type presents early within 5 to 9 days after surgery, with often serous discharge from the incision wound by an otherwise well-progressing patient. Once the infection becomes established, the patient develops fever, chills, elevated infection parameters, and sternal pain.

The second type presents late 10 to 30 days after operation. Usually the patient is already discharged and returns with partial disruption of the incision, with purulent discharge and sternal instability. These patients usually need plastic reconstruction due to involvement of the sternal bone. Many of the patients with chronic osteomyelitis of the sternum could have been avoided and might be due to not having surgically revised the sternum at an early stage of infection, as after retrospective evaluation of hospital and outpatient records it was found that discrete symptoms have been present for some time before definite diagnosis.

Known risk factors as insulin-dependent diabetes, obesity, operation duration, re-exploration for bleeding, use of blood products, and usage of (bilateral) mammary artery grafts² have not been associated with an increased risk to acquire sternal wound infection in our study cohort. Furthermore, we were not able to predict any certain predisposition for postoperative infection. However, we observed a direct relation between inadequate hygienic practice of the surgical team and an increased risk of developing a sternal wound infection.

Additionally, we assume that the incidence of wound infections could be more effectively decreased by modification of human behavior and elevating hygienic performance standards between surgical team members, which seems a still underestimated factor in the prevention of sternal wound infections as outlined by Borer and associates²⁰ in a recent publication.

We are aware of the potential limitations associated with the retrospective study design with the main limitation of the nonrandomized patient groups.

In conclusion, the high mortality and morbidity rate of poststernotomy wound infections necessitates rapid and aggressive treatment of this potentially fatal complication after cardiac surgery. As the conventional and established immediate primary closure therapy is associated with a high rate of infection recurrence, wound closure should be delayed until the wound condition has been optimized to prevent continuing sternal infection.

REFERENCES

1. Loop FD, Lytle BW, Cosgrove DM, et al. Sternal wound complications after isolated coronary artery bypass grafting: early and late mortality, morbidity and cost of care. *Ann Thorac Surg.* 1990;49:179-187.
2. De Feo M, Renzulli A, Ismeno G, et al. Variables predicting adverse outcome in patients with deep sternal wound infection. *Ann Thorac Surg.* 2001;71:324-331.
3. Hollenbeak CS, Murphy DM, Koenig S, et al. The clinical and economic impact of deep chest surgical site infections following coronary artery bypass graft surgery. *Chest.* 2000;118:397-400.
4. Shumacker HB, Mandelbaum I. Continuous antibiotic irrigation in the treatment of infection. *Arch Surg.* 1963;86:384-387.
5. Jurkiewicz MJ, Bostwick J, Hester TR, et al. Infected median sternotomy wound: successful treatment by muscle flaps. *Ann Surg.* 1980;191:738-744.
6. Francel TJ, Kouchoukos NT. A rational approach to wound difficulties after sternotomy: the problem. *Ann Thorac Surg.* 2001;72:1411-1418.
7. Rand RP, Cochran RP, Aziz S, et al. Prospective trial of catheter irrigation and muscle flaps for sternal wound infection. *Ann Thorac Surg.* 1998;65:1046-1049.
8. Yasuura K, Okamoto H, Morita S, et al. Results of omental flap transposition for deep sternal wound infection after cardiovascular surgery. *Ann Surg.* 1998;227:455-459.
9. Jones G, Jurkiewicz MJ, Bostwick J, et al. Management of the infected sternotomy wound with muscle flaps. *Ann Surg.* 1997;225:766-768.
10. El Gammal M, Yonan NA, Hassan R, et al. Treatment of mediastinitis: early modified Robicsek closure and pectoralis major advancement flaps. *Ann Thorac Surg.* 1998;65:41-47.
11. Morykwas MJ, Argenta LC, Shelton-Brown EI, et al. Vacuum assisted closure: a new method for wound control and treatment: animal studies and basic foundation. *Ann Plast Surg.* 1997;38:553-562.
12. Argenta LC, Morykwas MJ. Vacuum assisted closure: a new method for wound control and treatment: clinical experience. *Ann Plast Surg.* 1997;38:563-577.
13. Hersh RE, Jack JM, Dahrman MI, et al. The vacuum assisted closure device as a bridge to sternal wound closure. *Ann Plast Surg.* 2001;46:250-254.
14. Obdeijn MC, de Lange MY, Lichtendahl DHE, et al. Vacuum assisted closure in the treatment of poststernotomy mediastinitis. *Ann Thorac Surg.* 1999;68:2358-2360.
15. Tang ATM, Ohri SK, Haw MP. Novel application of vacuum assisted closure technique to the treatment of sternotomy wound infection. *Eur J Cardiothorac Surg.* 2001;17:482-484.
16. Harlan JW. Treatment of open sternal wounds with the vacuum assisted closure system: a safe, reliable method. *Plast Reconstruct Surg.* 2002;109:710-712.
17. Gustafsson R, Johnsson P, Algotsson L, et al. Vacuum assisted closure therapy guided by C reactive protein level in patients with deep sternal wound infection. *J Thorac Cardiovasc Surg.* 2002;123:895-900.
18. Fleck TM, Moidl R, Fleck M, et al. The vacuum assisted closure system for the treatment of deep sternal wound infections after cardiac surgery. *Ann Thorac Surg.* 2002;74:1596-1600.
19. El Oakley RM, Wright JE. Postoperative mediastinitis: classification and management. *Ann Thorac Surg.* 1996;61:1030-1036.

20. Borer A, Gilad J, Meydan N, et al. Impact of active monitoring of infection control practices on deep sternal infection after open heart surgery. *Ann Thorac Surg.* 2001;72:515-520.
21. Milano CA, Kesler K, Archibald N, et al. Mediastinitis after coronary bypass surgery: risk factors and long term survival. *Circulation.* 1995;92:2245-2251.
22. Brandt C, Alvarez JM. First line treatment of deep sternal infection by a plastic surgical approach: superior results compared with conventional cardiac surgery orthodoxy. *Plast Reconstr Surg.* 2002;109:2231-2237.
23. Lindsey JT. A retrospective analysis of 48 infected sternal wound closures: delayed closure decreases wound complications. *Plast Reconstr Surg.* 2002;109:1882-1885.
24. Mathisen DJ, Grillo HC, Vlahakas GJ, et al. The omentum in the management of complicated cardiothoracic problems. *J Thorac Cardiovasc Surg.* 1988;95:677-684.
25. Catarino PA, Chamberlain MH, Wright NC, et al. High pressure suction drainage via a polyurethane foam in the management of post sternotomy mediastinitis. *Ann Thorac Surg.* 2000;70:1891-1895.
26. Zacharias A, Habib R. Factors predisposing to median sternotomy complications: deep versus superficial infection. *Chest.* 1996;110:1173-1178.
27. Tegnell A, Aren C, Öhman L. Coagulase negative staphylococci and sternal infections after cardiac operation. *Ann Thorac Surg.* 2000;69:1104-1109.
28. Berg HF, Brands WGB, van Geldorp TR, et al. Comparison between closed drainage techniques for the treatment of postoperative mediastinitis. *Ann Thorac Surg.* 2000;70:924-929.

Das sternale V.A.C.[®]-System: Erfahrungen aus Wien

The vienna experience with the sternal V.A.C.[®]-system

T. Fleck

AKH Wien, Universitätsklinik für Chirurgie, Abteilung für Herz- und Thoraxchirurgie, Wien

Hintergrund

Die Behandlung sternaler Wundinfektionen, hat sich seit Einführung des V.A.C.[®]-Systems in den letzten Jahren grundlegend verändert. Wir verwenden seit Ende 2001 das V.A.C.[®] für die Behandlung postoperativer Wundinfektionen nach herzchirurgischen Eingriffen und konnten so reichlich Erfahrung sammeln, die wir hier präsentieren möchten.

Material und Methoden

Seit November 2001 bis Dezember 2008 haben 280 Patienten (Alter 10 Tage bis 86 Jahre) ein sternales V.A.C.[®]-System erhalten. Indikation war entweder eine sternale Wundinfektion in 250 Patienten oder als Überbrückung bis zum definitiven Sternumverschluss bei hämodynamisch instabilen Patienten (n=30). Zum Zeitpunkt der Diagnosestellung waren die Patienten im Schnitt am 13,4 (4-71) postoperative Tag.

Ergebnisse

Die mittlere V.A.C.[®]-Therapiedauer betrug 12,1 Tage (3-71) und es wurden im Schnitt 3 V.A.C.[®]-Wechsel durchgeführt. In 65% der Fälle war die Infektion tief (Mediastinitis), 35% hatten nur eine oberflächliche Infektion, die auf die Subkutis beschränkt war. Ein sekundärer Sternumverschluss nach Ende der

V.A.C.[®]-Therapie war in 76% möglich. Der Rest (24%) musste plastisch chirurgisch, mittels Pektoralis-Lappen-Plastik versorgt werden. Ein Wiederauftreten der Infektion nach erfolgtem Wundverschluss kam in 7,6% (19/250) Patienten vor.

Diskussion

Die Voraussetzung für eine erfolgreiche Behandlung der sternalen Wundinfektion ist eine möglichst frühzeitige Diagnose, damit ein Fortschreiten der Infektion auf den Sternumknochen vermieden werden kann und damit die Voraussetzung geschaffen wird, diesen auch wieder zu verschließen, was in einer Kostenersparnis resultiert.

Durch die große Patientenzahl, die wir zu versorgen haben, ist es sehr wichtig, dass bestimmte Guidelines und Standards eingehalten werden, sowie regelmäßig Erfolgskontrollen durchgeführt werden, um den hohen Qualitätsstandard zu halten und um etwaige Komplikationen rechtzeitig zu erkennen.

Background

There has been a notable change in the treatment of sternal wound infection during the last years since the introduction of the V.A.C.[®]-system. As we have gained an extensive experience in using the V.A.C.[®]-system in cardiac surgery patients over the last 8 years, it was the

aim of this study to evaluate the success rate, complications and lessons learned.

Methods

Since November 2001 to December 2008 280 patients (age range from 10 days to 86 years) received a vacuum assisted closure system for the treatment of sternal wound infection (n=250) or as a bridge to sternal closure in n=30. The diagnosis of sternal infection was made a mean on postoperative day 13,4 (from 4 to 71days). In 65% there was a deep infection with Mediastinitis, whereas 35% had a superficial infection.

Results

Mean V.A.C.[®]-duration was 12,1 days (ranging from 3 to 71days). Secondary sternal closure after V.A.C.[®]-therapy was possible in 76% of the patients, whereas 24% proceeded to plastic surgical reconstruction with muscle flaps, due to destruction of the sternal bone. Recurrence rate was 7,6% (19/250) which was due to not opening of the entire wound or too early closure.

Discussion

The key to successful management of sternal wound infection is early recognition, which enables preservation of the sternal bone and facilitates secondary sternal closure which reduces hospital stay and costs. However, especially in a high volume center as ours, certain guidelines in treating patients with wound infection have to be determined and followed, in order to ensure the best outcome.

Literatur

1. Fleck TM, Moidl R, Fleck M, Koller R, Giovanoli P, Wolner E, Grabenwoger M (2002) The vacuum assisted closure system for the treatment of deep sternal wound infections after cardiac surgery. *Ann Thorac Surg* 74: 1596-600.
2. Fleck T, Moidl R, Koller R, Giovanoli P, Wolner E, Grabenwoger M (2004) Delayed or primary closure for the treatment of post sternotomy wound infection? *Ann Plast Surg*. 52(3): 310-314.
3. Fleck T, Gustafsson R, Ingemansson R, Song DH, Harding K, Lirtzman MD, Meites H, Price P, Moidl R, Waldenberger, Salazar J, Sumpio BE (2006) The management of deep sternal wound infection using topical negative pressure therapy. *Int wound J* 3: 273-280.
4. Fleck T, Moidl R, Grimm M, Wolner E, Zuckermann A (2007) Vacuum assisted closure therapy for the treatment of sternal wound infections after heart transplantation: Preliminary results *Zentralblatt f Chirurgie* 2: 138-142.
5. Fleck T, Simon P, Burda B, Wolner E, Wollenek G (2006) Vacuum assisted closure therapy for the treatment of sternal wound infections in neonates and small infants. *ICVTS* 5: 285-288.
6. Fleck T, Moidl R, Giovanoli P, Aszmann O, Bartunek A, Blacky A, Grabenwoger M, Wolner E (2006) A conclusion from the first 125 patients treated with the vacuum assisted closure system for postoperative sternal wound infection. *ICVTS* 5: 145-8.
7. Fleck T, Kicking B, Moidl R, Waldenberger F, Wolner E, Grabenwoger M, Wisser W (2008) Management of open chest and delayed sternal closure with the vacuum assisted closure system: preliminary experience. *ICVTS* 7: 801-804.

The Impact of Vacuum-Assisted Closure™ on Long-Term Survival After Post-Sternotomy Mediastinitis

AUTHOR: Johan Sjögren, Johan Nilsson, Ronny Gustafsson, Malin Malmsjö & Richard Ingemansson

JOURNAL: Annals of Thoracic Surgery 2005;80:1270-1275

BACKGROUND

- Post-sternotomy mediastinitis after coronary artery bypass grafting is reported to be a strong predictor for poor late survival when using conventional wound-healing therapies¹.
- The risk of late death is reported to be two to three times higher in patients suffering from mediastinitis after cardiac surgery compared with patients without mediastinitis

PURPOSE

The goal was to compare the long-term survival after mediastinitis treated with V.A.C.® Therapy™ with that of patients without mediastinitis. A second goal was to identify risk factors for developing mediastinitis.

METHODS

- 4827 patients underwent isolated coronary bypass grafting in the Lund University Hospital between January 1999 and September 2004, of which 46 developed mediastinitis.
- The 46 patients developing mediastinitis were treated with V.A.C.® Therapy™, but without additional tissue flaps
- Actuarial survival was compared with the log-rank test.
- Univariate and multivariate analysis were used to identify risk factors for developing mediastinitis

Inclusion criteria

- Positive culture
- Clinical signs of post sternotomy mediastinitis
- Reoperation with removal of the sternal wires is required

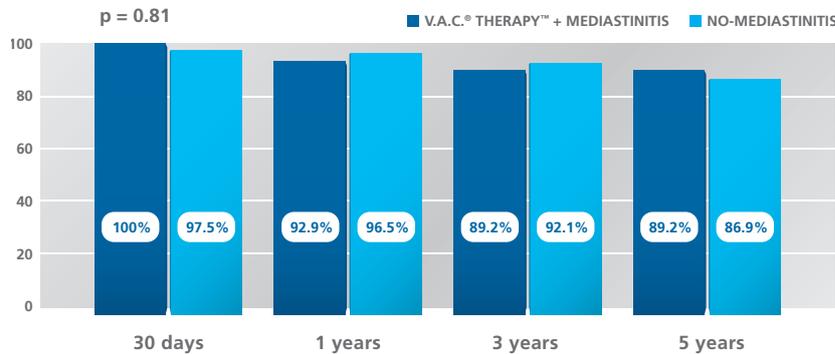
Exclusion criteria

- Negative substernal tissue cultures at the first revision
- Superficial sternal wound infection



Results and Conclusions

Survival in patients with mediastinitis treated with V.A.C.® Therapy™ compared to patients without mediastinitis



No difference in survival between patients with mediastinitis treated with V.A.C.® Therapy™ and patients that did not develop mediastinitis

Risk factors for mediastinitis (percentage of patients)

Risk factor	Mediastinitis + V.A.C.® Therapy™	Control (no-mediastinitis)	p Value
LVEF <30%	21.7	7.9	0.003
Diabetes mellitus	23.9	6.6	<0.001
Obesity (BMI >30)	47.8	19.1	<0.001v
Three-vessel disease	93.5	64.8	<0.001
Renal failure ^a	13	1.9	<0.001
Properative dialysis	4.3	0.7	0.05
Heart Failure	30.4	14.3	0.004
NYHA class III/IV	60.9	43.7	0.02
Procedure time	216 ±47 (minutes)	197 ±50 (minutes)	0.005

^aSerum creatinine > 200µmol/L (2.27 mg/dL)

BMI = body mass index; CI = confidence interval; LVEF = left ventricular ejection fraction; OR = odds ratio

CONCLUSIONS

- The present study suggests similar long-term survival between patients with V.A.C.® Therapy™-treated mediastinitis and patients without mediastinitis after isolated CABG.
- Risk factors for developing mediastinitis were found. The independently identified risk factors were similar to those found in previous studies.
- Despite the fact that risk factors for developing mediastinitis were significantly higher in the V.A.C.® Therapy™ group, the mortality was not increased.
- V.A.C.® Therapy™ minimizes the effect of mediastinitis on late survival after coronary artery bypass grafting.

¹Braxton, J. H. et al. Mediastinitis and long-term survival after coronary artery bypass graft surgery. Ann. Thorac. Surg. 70, 2004-2007 (2000).