Long-term Results of Pectoralis Major Muscle Transposition for Infected Sternotomy Wounds

PETER C. PAIROLERO, M.D.,* PHILLIP G. ARNOLD, M.D.,† and JOHN B. HARRIS, M.D.†

During an 11.5-year period, 100 consecutive patients (79 male, 21 female) underwent repair of an infected sternotomy wound. Sixty-five patients had failed attempts at wound closure by other physicians. Median age was 61.5 years (range, 5 to 85 years). Reconstruction included muscle in 79 patients, omentum in 4, and both in 15. A total of 175 muscles were transposed, including 169 pectoralis major, 3 rectus abdominis, 2 external oblique, and 1 latissimus dorsi. Median number of operations was four (range, 1 to 11). Mechanical ventilation was required in 30 patients. Two perioperative deaths occurred, one related to sepsis. Median follow-up was 4.2 years (range, 1.3 to 13.5 years). Twenty-six patients had recurrent infection. Median time from our closure to recurrence was 5.5 months (range, 0.3 to 27.6 months). Cause of recurrence was inadequate removal of cartilage in 16 patients, bone in 6, and retained foreign body in 4. Eighteen patients had the wound reopened with further resection; 10 had another muscle or omentum transposition. There were 30 late deaths, only one related to recurrent infection. At the time of death or last follow-up, 92 patients had a healed chest wall. Transposition of the pectoralis major muscle remains an excellent method of management for infected sternotomy wounds. Failure is directly related to persistent infection of cartilage, bone, or retained foreign bodies.

From the Sections of General Thoracic Surgery and Plastic and Reconstructive Surgery,† Mayo Clinic and Mayo Foundation, Rochester, Minnesota*

MEDIAN STERNOTOMY IS the preferred approach for most cardiac operations and for resection of many anterior mediastinal masses.[1] Although wound complications after sternotomy are rare, infection remains the most threatening because of the potential for mediastinal sepsis with extension to aortocoronary grafts, cardiotomy incisions, and intracardiac prostheses.[2] In the past, the consequences of such infections have been death or a prolonged convalescence.[3–6] In 1980 Jurkiewicz and associates[7] revolutionized management of this complication by reporting excellent results in patients treated by vigorous sternal debridement and obliteration of dead space with healthy, viable muscle. In 1984 we reported[8] similar excellent results; however our follow-up was short-term. The purpose of this report is to review our long-term experience with recalcitrant infected sternotomy wounds that were managed primarily with muscle transposition and to analyze factors affecting long-term successful closure.

Patients and Methods

Between June 1, 1977 and November 21, 1988, 100 consecutive patients with infected median sternotomy wounds were treated at the Mayo Clinic by the combined efforts of a single thoracic and plastic surgeon (PC Pairolero, PG Arnold). This combined approach, however, does not represent the entire experience with such wounds at our institution during that time. Seventy-nine patients were male and 21 were female. Ages ranged from 5 to 85 years (median, 61.5 years). Sternotomy was performed for cardiac disease in 92 patients and for mediastinal disease in eight (Table 1). Seventy-four patients had coronary artery bypass; the internal mammary artery was used in 36 patients (left in 32 and both in 4). Ninety patients had a single previous sternotomy; 10 had multiple sternotomies, all for cardiac disease. Six patients had 2 cardiac operations, two had 3, and two had 4. Ten of the 100 patients had their sternotomy wound reopened during the immediate postoperative period because of bleeding or dehiscence; two other patients had emergent vagotomy and antrectomy for bleeding duodenal ulcers. Eight patients had received previous mediastinal irradiation for previous malignant disease. Forty-four patients had their sternotomies performed at another institution.

Presented at the 102nd Annual Scientific Session of the Southern Surgical Association, Boca Raton, Florida, December 3–6, 1990.

Address reprint requests to Peter C. Pairolero, M.D., Mayo Clinic, 200 First St. Southwest, Rochester, MN 55905.

Accepted for publication February 1, 1991.

TABLE 1. *Indications for Median Sternotomy*

Indication	Number of Patients	Previous Mediastinal Radiation (No. of Patients)
Cardiac	92	3
CABG	70	
CABG + AVR	2	
CABG + MVR	2	
MVR	4	
AVR	6	
MVR + AVR	3	
Fontan procedure	3	
Tetralogy of Fallot	2	
Mediastinal	8	
Cancer	5	5
Pneumothorax	2	
Aortoinnominate bypass	1	
Total	100	8

CABG, coronary artery bypass graft; AVR, aortic valve replacement; MVR, mitral valve replacement.

Sternal drainage occurred 1 week to 4 years after sternotomy (median, 3 weeks). The median time interval between the onset of sternal drainage and sternal repair by us was 7.5 weeks (range, 2 days to 6 years). During this latter time period, 65 patients had previous attempts at wound closure by other physicians that failed, which included debridement in all patients, mediastinal irrigation and suction drainage in 41, omental transposition in 2, and muscle transposition in 1. The median number of sternal debridements in these 65 patients before evaluation by us was two (range, 1 to 10). All patients had purulent sternal drainage.

Our initial treatment included debridement of the manubrium and sternum in all patients. Early in our experience mediastinal debridement was limited. However now we think that mediastinal debridement should be done in all patients, and great effort was made in our later experience to remove all exposed foreign bodies, including felt pledgets, pacemaker wires, and sutures.[9] If heart or great vessel suture lines required reinforcement because of real or potential bleeding, fascia lata and transposed muscle were used. In addition to debridement, the manubrium and sternum were resected completely in 43 patients and partially in 33; all 76 had associated costochondral arches resected from the back to the ribs.

Tissue cultures, obtained in all patients, were positive in 95. One hundred eighty-two organisms were isolated (Table 2). A single organism was identified in 45 patients, two in 24, three in 20, four in 3, six in 1, and eight in 1. Eighty patients had only bacteria identified, two had only fungus, and 13 had both. No patient had multiple fungi isolated.

Muscle or omental flaps were used to obliterate the underlying mediastinal space in 98 patients. In the remaining two patients, the skin wound was left to heal secondarily after debridement only. Reconstruction was entirely with muscle transposition in 79 patients, omental transposition in 4, and both in 15. Transposition of the various muscles and omentum has been described previously.[8,10–16] A total of 175 muscles were transposed, including 169 pectoralis major (all based on the thoracoacromial leash), 3 rectus abdominis, 2 external oblique, and 1 latissimus dorsi. Seven patients had myocutaneous flaps (rectus abdominis in 2, pectoralis major in 2, external oblique in 2, and latissimus dorsi in 1). Bilateral pectoralis major muscle transpositions were performed in 79 patients. The nondominant humeral attachment of the pectoralis muscle was divided in 114 of the 169 transpositions (65.7%).

The median number of operations in these 100 patients was four (range, 1 to 11). The wound was closed at the time of the initial debridement in 11 patients. In the remaining 89 patients, the wound was dressed with moist gauze after initial debridement and/or resection and four times a day thereafter. All wounds were thoroughly irrigated with water at the time of each dressing change. Additional debridements to remove nonviable tissue were done as needed. Quantitative tissue cultures were not obtained to determine the timing of wound closure. Instead the wound was closed when healthy granulation tissue was present without gross exudate. Wound closure was performed a median of 14 days after the initial debridement (range, 2 to 192 days) in 88 patients.

Hospitalization ranged from 7 to 210 days with a median of 26.5 days. Fifty-nine complications occurred in 42 patients (Table 3). Eight patients had prolonged wound

TABLE 2. *Micro-organisms Identified in 95 Patients with Infected Sternotomy Wounds*

Micro-organisms	Number of Patients
Bacteria	
Staphylococcus aureus	65
Pseudomonas aeruginosa	19
Streptococcus viridins	16
Staphylococcus epidermidis	13
Enterobactor	10
Serratia marcescens	8
Staphylococcus pyogenes	7
Escherichia coli	6
Cornybacteria	6
Propionibacteria	5
Hemophylis influenza	3
Klebsiella pneumoniae	3
Proteus vulgaris	3
Actinobacillus	1
Salmonella	1
Fungus	
Candida albicans	13
Rhizopus arrhizus	1
Aspergillus fumigatus	1
Trichoderma	1

TABLE 3. *Complications*

Complication	Number
Wound infection	8
Wound hematoma	6
Myocardial infarct	4
Muscle necrosis	2
Omental necrosis	2
Renal failure	2
Acute femoral artery occlusion	1
Mycotic popliteal artery aneurysm	1
T_{4-5} disc space infection	1
Wrist space infection	1
Subphrenic abscess	1

infections (5 superficial, 3 deep); 7 were dismissed with a portion of the wound healing secondarily, and 1 was open at the time of perioperative death. All 6 patients with wound hematomas required operative evacuation. All patients with distant sites of infection (popliteal artery, T_{4-5} disc space, wrist space, and subphrenic space) also required surgical intervention.

After each operative procedure, the patients generally were extubated on the evening of the operation. Mechanical ventilation beyond the second postoperative day, however, was required in 30 patients, 21 because of chest wall instability and 9 because of continuing sepsis. Two of the 21 patients with chest wall instability had tracheostomy; 1 was performed at another institution for unknown reasons, and 1 was performed by us for aspiration pneumonia. All wounds in these 21 patients were successfully closed, and extubation was possible in all by 14 days (median, 3.5 days). Six of the nine patients with sepsis had tracheostomy. Five of the wounds in these nine patients were closed and, again, extubation was possible in each. Extubation also was possible in one of the four patients in whom the wound could not be closed. The median length of mechanical ventilation in the five patients who were extubated was 45 days (range, 32 to 100 days).

Two perioperative deaths occurred. The first was in an 85-year-old man who underwent aortic valve replacement. Three weeks later the wound became infected. The patient had an uneventful course following debridement and closure with bilateral pectoralis major flaps until sudden death from a myocardial infarction 17 days later. The second death occurred in a 71-year-old man who had coronary artery bypass and who underwent emergency vagotomy and pyloroplasty on the 11th postoperative day for a bleeding duodenal ulcer. Bleeding continued and 4 days later he underwent antrectomy. This was followed by generalized sepsis, including mediastinitis and peritonitis. Although the mediastinum was debrided and bilateral pectoralis major flaps were transposed, the wound was never closed because of progressive respiratory and renal failure. Death occurred at 171 days.

One patient was lost to follow-up. This was a 61-year-old man who developed an infected sternotomy wound after coronary artery bypass. Although closed with bilateral pectoralis major flaps, sepsis continued and resulted in wound breakdown, respiratory failure, and tracheostomy. His hospital course also was complicated by acute common femoral artery occlusion that necessitated thrombectomy. However gradual improvement occurred and eventually he was transferred to his home hospital in Spain on the 88th postoperative day with both a tracheostomy and his sternum healing secondarily. Follow-up in the remaining 97 operative surviving patients ranged from 1.3 to 13.5 years (median, 4.2 years).

Seven patients were dismissed from the hospital with a portion of the sternal wound open. One patient was lost to follow-up and one died from sepsis at 3 months. The wounds in four of the patients healed spontaneously between 3 to 6 months, and one was closed with omentum and skin graft at 2 months.

Recurrent sternal infection occurred in 26 patients and was more frequent in the last half of our study period (in 21 of 66 patients) than in the first half (5 of 34 patients) ($\chi^2 = 2.584$, $p = 0.108$). Median time from our closure to recurrence was 5.5 months and ranged from 0.3 to 27.6 months. All recurrences presented as draining sinuses, most commonly associated with osteomyelitis and/or costochondritis. None had abscess collection in the mediastinum, pleura, or peritoneum, although several patients had draining sinus tracts to retained mediastinal foreign bodies. Drainage, erythema, and local tenderness were the most common findings. Other signs and symptoms were uncommon.

Factors possibly affecting recurrent sternal infection are shown in Table 4. Only inadequate removal of nonviable tissue at the time of definitive repair was significant. Those patients who had resection of the manubrium, sternum, and costochondral arches were significantly less likely to develop late recurrence (10 of 64 patients) than those who had debridement only (14 of 26 patients) ($p < 0.001$). The number of operations to close a wound successfully, the extent of resection (complete *versus* partial), a history of previous sternal irradiation, the number of previous sternotomies, the institution where the sternotomy was performed, the indication for sternotomy, and whether the internal mammary artery was used for cardiac revascularization did not affect recurrence ($p = NS$). Recurrence in our patients was attributed to inadequate removal of cartilage in 16 patients, bone in 6, and retained foreign bodies in 4.

Anterior chest wall pain was present in 37 patients. This pain was described as a stabbing sensation in 27 patients, tightness in 4, ache in 3, numbness in 1, and spasms in 1. The pain generally was always present, made worse by activity, and usually not relieved by analgesics or other pain clinic techniques. Symptoms were more common in

TABLE 4. *Factors Affecting Long-term Successful Closure*

Factors	Recurrence	No Recurrence	p Value*
Removal of tissue			
Debridement	14	10	
Resection	12	64	0.001
No. of operations			
Single	0	11	
Multiple	26	63	0.086
Extent of resection			
Complete	7	36	
Partial	5	28	0.854
Sternal radiation			
Yes	2	6	
No	24	68	0.724
No. of prior sternotomies			
Single	25	65	
Multiple	1	9	0.403
Institution			
Mayo Clinic	14	42	
Other	12	32	0.978
Indication for sternotomy			
Cardiac	23	67	
Mediastinum	3	7	0.939
Coronary artery bypass			
Mammary artery	8	28	
Vein only	14	24	0.262

* Chi square test.

patients who developed recurrent infection (19 of 26 patients) than in those who did not (18 of 74 patients) ($\chi^2 = 17.582$, $p < 0.001$). Five of nineteen patients with omental transpositions developed hernias through the tunnel through which the omentum was passed. All five patients were asymptomatic and none required surgical intervention. Progressive shortness of breath during follow-up was uncommon.

The wound healed spontaneously in 7 of the 26 patients with recurrent infection. One patient refused further surgical treatment and the wound currently continues to drain. The remaining 18 patients underwent additional operative procedures. All patients had further removal of nonviable tissue, 10 by debridement of residual manubrium, sternum, and cartilage, and eight by resection. Eight of these patients had debridement or resection only, seven also had another muscle transposition, and three had omental transposition. Retained foreign bodies also were removed from four patients. Following these procedures 13 patients had no further signs or symptoms of infection; however infection recurred for a second time in five patients, all of whom had had debridement rather than resection as management for the first recurrence. Three of these five patients underwent additional resection and a third muscle transposition, one had debridement only, and one refused further surgical intervention. At last follow-up the wounds in three of these five patients were healed, and two continue to drain (one patient who had debridement and one who had resection). In summary 22 of the 26 patients with recurrent sternotomy infections now have well-healed chest wounds without drainage.

There were 30 late deaths, only one related to infection. This patient was a 71-year-old man who had an uneventful course after wound closure with bilateral pectoralis major flaps. Although discharged from the hospital, infection persisted in this man. The wound was reopened and debrided, but adult respiratory distress syndrome developed and the patient died 2.5 months after initial closure. Cause of the death in the remaining 29 patients was cardiac in 20, cancer in 3, renal failure in 1, trauma in 1, radiation myelitis in 1, and unknown causes in 3. At the time of death, 27 patients had healed chest walls.

There were 98 operative survivors; one was lost to follow-up and one died after 2.5 months from continuing infection. Twenty-six patients developed recurrent sternal infection, 22 of whom were successfully treated. Sixty-seven patients are known to be alive. Median follow-up in these 67 patients was 4.7 years (range, 0.7 to 13.5 years). Sixty-five of these former patients have healed anterior chest walls. At the time of death or last follow-up, 92 patients had healed chest walls without drainage.

Discussion

Since 1957 median sternotomy has become the most commonly used incision in cardiac operations.[17] The rapidity and ease with which it can be performed and the excellent access it provides to the heart and great vessels are major factors contributing to its widespread acceptance. Although wound complications are few, infection remains the most ominous.[18,19] If left untreated these infections can extend to aortic and cardiac suture lines, prosthetic grafts, and intracardiac prostheses and can result in death.

The earliest treatment of infected sternotomy wounds consisted of debridement and open sternal drainage, which had an attendant mortality rate of more than 50%.[20] Shumacker and Mandelbaum[3] and Bryant et al.[4] subsequently described the technique of closed mediastinal irrigation and suction drainage, which reduced the mortality rate to approximately 20%. In 1976 Lee, et al.,[21] offered an alternative method of management by transposing the omentum to eliminate mediastinal dead space. Jurkiewicz and associates expanded this concept by using pectoralis major muscle flaps and reduced the mortality rate to zero.[7] Since then these and other authors reported equally excellent results in large numbers of patients, often using multiple stages.[8,22-28]

Nearly all authors agree that the pectoralis major muscle is ideal to manage patients with complicated infected median sternotomy wounds. The muscle has a dominant blood supply based on the thoracoacromial artery. Mo-

bilization of this muscle allows an arc of rotation that reaches all of the anterior chest wall except the lower sternum. Nonetheless treatment of infected sternotomy wounds continues to be controversial. Recently good results were reported with less extensive and single-stage procedures, suggesting that extensive debridement and multiple operations may not be necessary.[29,30] However most reports fail to distinguish between the different types of sternal wound infections and none report factors that influence successful long-term closure.

Clearly not all infected median sternotomy wounds require muscle or omental transposition. Superficial infections may be treated successfully by adequate drainage, specific parenterally administered antibiotics, local wound care, and simple secondary closure. Deeper infections that involve the sternum and the anterior mediastinum, however, require exploration and thorough debridement.

Deep infected median sternotomy wounds present in one of three ways (Table 5).[31] The earliest are type I infections. These patients present with serosanguineous wound drainage within a few days of sternotomy. Few patients have skin cellulitis; none have mediastinal suppuration, osteomyelitis, or costochondritis. Wound cultures often demonstrate no growth; when positive, however, a staphylococcal species is encountered in nearly 50% of cases. Early recognition of type I wound infection requires a high index of suspicion and they are best managed in the operating room under general anesthesia, where both the subcutaneous and mediastinal spaces can be inspected. Because the mediastinum is always soft and pliable, residual space is obliterated easily by suction drainage, thereby allowing the wound eventually to heal in most patients. A few, however, will progress and will require more extensive debridement with obliteration of dead space by viable tissue. Our experience with type I wound infection is minimal because most are managed by the operating cardiac surgeon. Nonetheless 11 of our patients had this early type of infection; all were managed with a single operative procedure and none developed recurrent infection. Equally important is the fact that 41 of our patients had mediastinal irrigation and suction drainage with immediate closure by other surgeons, yet infection progressed in all. When we evaluated these 41 patients, all had type II infection, as described below, and all required more extensive operations. We could not determine historically what type of infection was present at the time of previous treatment at other institutions.

TABLE 5. *Classification of Infected Sternotomy Wounds*

Classification	Number of Patients
Type I	11
Occurs within first few days	
Serosanguineous drainage	
Cellulitis of skin absent	
Mediastinum soft and pliable	
Osteomyelitis and costochondritis absent	
Cultures usually negative	
Type II	84
Occurs within first few weeks	
Purulent drainage	
Cellulitis of skin present	
Mediastinal suppuration	
Osteomyelitis frequent, costochondritis rare	
Cultures positive	
Type III	5
Occurs months to years later	
Chronic draining sinus tract	
Cellulitis of skin localized	
Mediastinitis rare	
Osteomyelitis, costochondritis, or retained foreign body always present	
Cultures positive	
Total	100

Type II infections present as fulminant mediastinitis usually within the first few weeks after operation. These patients often have purulent wound drainage, skin cellulitis, and obvious communication between the sternum and mediastinum. Many have acute osteomyelitis but only a few have associated costochondritis. Cultures usually are positive for staphylococcal species. Like type I infections, our practice is to inspect type II wounds in the operating room under general anesthesia, where all recesses of the mediastinum must be explored thoroughly. Necrotic tissue such as bone, cartilage, or soft tissue should be excised. Any exposed healthy cartilage should be resected back to normal bone.[32] The cut surface of cartilage is at greater risk of recurrent infection than the cut surface of bone. All foreign material, including sutures, pledgets, and sternal wire, are removed. Every effort should be made to avoid entering the pleural space if there is no clinical or roentgenographic evidence of empyema.

The key to deciding whether type II wounds can be closed at the time of initial debridement is the condition of the mediastinum. If soft and pliable, suction drainage usually will obliterate mediastinal space after the sternum is closed following debridement. Obliteration of dead space below the sternum combined with muscle transposition on the external surface usually will prevent reinfection and many wounds will heal with this technique. However there is a group of patients with advanced type II infections in which the suppuration has caused the mediastinum to become indurated and thickened. As a result pliability decreases and mediastinal dead space cannot be obliterated by suction drainage alone. These wounds mandate obliteration of this space by other techniques. Late type II infections were the most common wound we treated. It was also the wound most likely to develop recurrent infection. These latter findings probably reflect the tertiary nature of our practice and explain why recurrent infection was more common in the last half of

our experience (31.8%) than in the first half (14.7%), although this difference is not statistically significant.

Type II wounds that are not closed immediately are dressed with gauze moistened with saline solution following thorough resection. The dressing is changed in the patient's room every 4 to 6 hours and the wound irrigated with water. If possible the patient showers because drainage is considerably better in the upright position. If bedridden irrigation is done in a special treatment facility with a hand-held shower or Water Pik (Teledyne, Inc., Fort Collins, CO).[33] If at any time there is evidence of new or persistent necrotic tissue, the patient is returned to the operating room for further debridement.

Closure of the wound is performed when there is no evidence of drainage, when nonviable periosteum, perichondrium, bone, or cartilage has all been removed, and when the edges of the debrided sternum or ribs are clean. Our first choice to obliterate the mediastinal space is the pectoralis major muscle transposed on the dominant proximal thoracoacromial neurovascular leash. Frequently both pectoralis major muscles are mobilized. The nondominant humeral attachment is divided as needed to permit the degree of rotation and advancement required; sometimes both humeral attachments must be divided to close the wound. The overlying skin and subcutaneous tissue then are closed with direct suture. We also prefer to close the lower portion of the wound with skin and subcutaneous tissue rather than the rectus abdominis muscle because recurrences, in our experience, were not related to this portion of the wound. Myocutaneous flaps are used on rare occasions when they seem to be the best geometric solution to a wound of a particular size, shape, and location. If the epicardium is exposed, the omentum may be transposed to obliterate any pericardial space and to cover the heart, if necessary. The more superficial layers of the chest wall then are closed by skin graft, muscle flaps, or direct closure of skin and subcutaneous tissues.

Sometimes the pectoralis major muscles are absent or insufficient to close the wound. In these patients consideration must be given to other types of transposition; we prefer omental or rectus abdominis muscle transposition. Both of these flaps should be considered second-line treatment choices. Omental transposition has the distinct disadvantage of both entering a sterile visceral cavity and producing a ventral hernia. One of our 19 patients who had omental transposition developed a subphrenic abscess because of this procedure and five developed hernias. The rectus abdominis muscle flap may not be as dependable if the internal mammary artery has been used in a coronary artery bypass, thrombosed from infection, or ligated with sternal debridement.

Type III infections occur months to years later. These patients present with a chronic draining sinus tract from the manubrium, sternum, costochondral arches, or retained foreign bodies. All have osteomyelitis and/or costochondritis but mediastinitis is rare. Most patients also have localized skin cellulitis around the sinus tract site. Cultures often are positive. When the sinus tract is explored, all infected bone and necrotic tissue must be resected back to healthy bone. If the sinus tract extends into the mediastinum, usually there is retained foreign material and this also must be removed. If the sinus tract enters cartilage, the entire cartilage must be resected back to normal bone. After debridement we prefer to dress this wound open with frequent dressing changes and repeat debridement as indicated. Once the wound is clean, closure is as described above with preference given again to pectoralis major muscle transposition.

Twenty-six per cent of our patients developed recurrent infection. Interestingly the recurrence rate was nearly identical for both the second (28%) and third (25%) surgical attempts to heal the wounds. When factors affecting recurrent infection were analyzed, only incomplete removal of nonviable tissue was significant. Those patients who had debridement only had a significantly greater chance of developing late recurrence as compared to those who had resection; this observation also was true after the second and third attempts at surgical wound healing. Although not statistically significant, no recurrence occurred in patients in whom we were able to close the wound in one procedure. We think, however, that this is so only because of the small number of patients involved.

Recurrent infections often were managed with another muscle transposition in addition to debridement or resection. Most transpositions involved the same pectoralis major muscle in that the previously transposed muscle was remobilized to allow debridement or resection and then retransposed. None of our patients had loss of muscle with this technique.

Long-term respiratory failure after successful wound healing was unlikely and was not related to whether mechanical ventilation was required during the time of sternal wound management. Instead progressive shortness of breath was related more commonly to late cardiac failure. This observation correlates with our previous experience with resection of the anterior chest wall for neoplasms.[34]

We conclude that there is a spectrum of infected sternotomy wounds and that all follow the same time-honored principles of wound healing elsewhere: the wound must be drained adequately; all necrotic tissue, devascularized tissue, and foreign material must be removed; and all residual space must be obliterated. If these principles are achieved, the entire spectrum of sternotomy wound infections can be healed successfully. For patients with late, recalcitrant median sternotomy wound infections, these principles are best achieved by sternal resection combined with pectoralis major muscle transposition.

Acknowledgments

The authors thank our colleagues in Thoracic and Cardiovascular Surgery and Plastic and Reconstructive Surgery for their cooperation and support of this effort.

References

1. Barnhorst DA. Extracardiac thoracic complications of cardiac surgery. Surg Clin North Am 1973; 53:937–944.
2. Sanfelippo PM, Danielson GK. Complications associated with median sternotomy. J Thorac Cardiovasc Surg 1972; 63:419–423.
3. Shumacker HB Jr, Mandelbaum I. Continuous antibiotic irrigation in the treatment of infection. Arch Surg 1963; 86:384–387.
4. Bryant LR, Spencer FC, Trinkle JK. Treatment of median sternotomy infection by mediastinal irrigation with an antibiotic solution. Ann Surg 1969; 169:914–920.
5. Culliford AT, Cunningham JN Jr, Zeff RH, et al. Sternal and costochondral infections following open-heart surgery: a review of 2,594 cases. J Thorac Cardiovasc Surg 1976; 72:714–726.
6. Serry C, Bleck PC, Javid H, et al. Sternal wound complications: management and results. J Thorac Cardiovasc Surg 1980; 80: 861–866.
7. Jurkiewicz MJ, Bostwick J III, Hester TR, et al. Infected median sternotomy wounds: successful treatment by muscle flaps. Ann Surg 1980; 191:738–743.
8. Pairolero PC, Arnold PG. Management of recalcitrant median sternotomy wounds. J Thorac Cardiovasc Surg 1984; 88:357–364.
9. Arnold PG, Pairolero PC. Intrathoracic muscle flaps in the surgical management of life-threatening hemorrhage from the heart and great vessels. Plast Reconstr Surg 1988; 81:831–835.
10. McCraw JB, Arnold PC. McCraw and Arnold's Atlas of Muscle and Musculocutaneous Flaps. Norfolk: Hampton Press Publishing, 1986.
11. Arnold PG, Pairolero PC. Use of pectoralis major muscle flaps to repair defects of anterior chest wall. Plast Reconstr Surg 1979; 63:205–213.
12. Pairolero PC, Arnold PG. Bronchopleural fistula: treatment by transposition of pectoralis major muscle. J Thorac Cardiovasc Surg 1980; 79:142–145.
13. Arnold PG, Pairolero PC. Chest wall reconstruction: experience with 100 consecutive patients. Ann Surg 1984; 100:725–731.
14. Arnold PG, Pairolero PC, Waldorf JC. The serratus anterior muscle: intrathoracic and extrathoracic utilization. Plast Reconstr Surg 1984; 73:240–246.
15. Arnold PG, Pairolero PC. Surgical management of the radiated chest wall. Plast Reconstr Surg 1986; 77:605–612.
16. Jurkiewicz MJ, Arnold PG. The omentum: an account of its use in the reconstruction of the chest wall. Ann Surg 1977; 185:548–554.
17. Julian OC, Lopez-Belio M, Dye WS, et al. The median sternal incision in intracardiac surgery with extracorporeal circulation: a general evaluation of its use in heart surgery. Surgery 1957; 42: 753–761.
18. Engelman RM, Williams CD, Gouge TH, et al. Mediastinitis following open-heart surgery: review of two years' experience. Arch Surg 1973; 107:772–778.
19. Thurer RJ, Bognolo D, Vargas A, et al. The management of mediastinal infection following cardiac surgery: an experience utilizing continuous irrigation with povidone-iodine. J Thorac Cardiovasc Surg 1974; 68:962–968.
20. Sarr MG, Gott VL, Townsend TR. Mediastinal infection after cardiac surgery. Ann Thorac Surg 1984; 38:415–423.
21. Lee AB Jr, Schimert G, Shatkin S. Total excision of the sternum and thoracic pedicle transposition of the greater omentum. Surgery 1976; 80:433–436.
22. Nahai F, Rand RP, Hester TR, et al. Primary treatment of the infected sternotomy wound with muscle flaps: a review of 211 consecutive cases. Plast Reconstr Surg 1989; 84:434–441.
23. 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.
24. Hazelrigg SR, Wellons HA Jr, Schneider JA, Kolm P. Wound complications after median sternotomy. J Thorac Cardiovasc Surg 1989; 98:1096–1099.
25. Johnson JA, Gall WE, Gundersen AE, Cogbill TH. Delayed primary closure after sternal wound infection. Ann Thorac Surg 1989; 47:270–273.
26. Stiegel RM, Beasley ME, Sinke JD, et al. Management of postoperative mediastinitis in infants and children by muscle flap rotation. Ann Thorac Surg 1988; 46:45–46.
27. Demmy TL, Park SB, Liebler GA, et al. Recent experience with major sternal wound complications. Ann Thorac Surg 1990; 49: 458–462.
28. Majure JA, Albin RE, O'Donnell RS, Arganese TJ. Reconstruction of the infected median sternotomy wound. Ann Thorac Surg 1986; 42:9–12.
29. Grossi EA, Culliford AT, Krieger KH, et al. A surgery of 77 major infectious complications of median sternotomy: a review of 7,949 consecutive operative procedures. Ann Thorac Surg 1985; 40: 214–223.
30. Jeevanandam V, Smith CR, Rose EA, et al. Single-stage management of sternal wound infections. J Thorac Cardiovasc Surg 1990; 99: 256–263.
31. Pairolero PC, Arnold PG. Management of infected median sternotomy wounds (editorial). Ann Thorac Surg 1986; 42:1–2.
32. Williams CD, Cunningham JN, Falk EA, et al. Chronic infection of the costal cartilage after thoracic surgical procedures. J Thorac Cardiovasc Surg 1973; 66:592–598.
33. Trelstad A, Osmundson D. Water Piks: wound cleansing alternative. Plastic Surg Nursing 1990; 9:117–119.
34. Meadows JA III, Staats BA, Pairolero PC, et al. Effect of resection of sternum and manubrium in conjunction with muscle transposition on pulmonary function. Mayo Clin Proc 1985; 60:604–609.

DISCUSSIONS

DR. FRANCIS ROBICSEK (Charlotte, North Carolina): I agree with Dr. Arnold and his coworkers that the pectoralis muscle is perhaps the surgeon's best friend, and in our practice we use it not only to treat sternum infection but also to prevent some of the complications before they occur.

We use it in especially two types of patients. In very thin women who are liable to develop sternal complication, we mobilize the pectoralis muscle back to about 1.5 or 2 inches, more on the side where the mammary is intact and unite it in the front of the sternum. This gives an excellent soft padding. And in our experience, the wound healing is much better.

The second type of patients in whom we use this method are heavyset, robust individuals, especially those with obstructive pulmonary-arterial disease.

I believe that the pectoralis muscle, besides being a friend, is also a culprit that causes or contributes to some of the disruptions by pulling the sternum apart. If you reverse this process by uniting it in the front of the sternum, then the pectoralis muscle is working for you instead of against you.

We also use this method in sternal disruptions, even before the infection occurs. In such cases we do the sternal weaving around the wire down on the sides of the sternum then unite the pectoral muscle in the front of the sternum. In practically all of the cases, you can obtain a very nice healing.

Use of muscle flaps in thoracic infection is probably the most important development in the history of thoracic surgery in the last decade. And for this Dr. Arnold and his colleagues who have pioneered this procedure deserve the gratitude of the profession.