

Balloons, Beds, and Breakdown

Effects of Low-Air Loss Therapy on the Development of Pressure Ulcers in Cardiovascular Surgical Patients with Intra-aortic Balloon Pump Support

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Pressure ulcers are a potential problem confronting any patient who remains immobile for a prolonged period of time. More important than the financial costs of treatment, pressure ulcers are a source of dissatisfaction, anxiety, and pain for the patient and family.¹ Recent studies report the national incidence of hospital-acquired pressure ulcers to be less than 13% for all inpatients^{2,3} and greater than 50% for patients in intensive care settings.^{2,4} Kosiak's⁵ classical "inverse time-pressure" theory on the cause of pressure ulcers proposed that when externally applied pressures exceed capillary hydrostatic pressures, tissues are deprived of oxygen and nutrients. Consequently, normal cellular function is disrupted. Researchers have extended Kosiak's "time-

pressure" theory by identifying intrinsic factors that influence tissue tolerance to external forces.^{6,7} Conditions that have been identified as contributing to pressure ulcer development, such as restricted mobility, chronic disease, advanced age, impaired sensory perception, altered tissue perfusion associated with interstitial edema and anemia, and malnutrition, are extremely prevalent, if not the norm, in the ICU patient population.^{1,8}

Theoretically, pressure ulcers are preventable. Certain pathologic conditions and therapeutic interventions associated with extremely high acuity and prolonged immobility challenge this theory,⁹ however. Cardiovascular surgical (CVS) patients requiring intra-aortic balloon pump (IABP) support are prime candidates for developing pressure ulcers. Not only do they have risk factors similar to those in the ICU patient population, but they have many other risk factors as well. For

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example, comorbidities frequently associated with cardiovascular disease, including diabetes, hypertension, peripheral vascular disease (PVD), and renal insufficiency predispose the patient to ulcer formation by decreasing the tissue's tolerance to pressure.¹⁷ Furthermore, to maintain tissue perfusion while providing the surgeon with a workable surgical field, certain intraoperative interventions used during CVS, such as extracorporeal circulation,²⁷ hypothermia, hypovolemia, anemia,^{27,28,29} and vasopressor (vasoconstriction) therapy,²⁸ increase the chances of sustaining ischemic injury. Extracorporeal circulation was found to be a significant univariate predictor of pressure ulcer formation ($\chi^2 [1, n = 125] = 4.27, P = 0.039$) in a previous research study.¹⁷

Intraoperative variables responsible for pressure ulcer formation include prolonged duration on the operating room (OR) table²⁸ and altered sensory perception related to general anesthesia and deep sedation. During CVS, the patient is rendered motionless for extended periods of time and is unable to respond to ischemic pain. Kemp et al²⁷ identified 70% of pressure ulcers at the time patients were transferred from the OR table. Although several researchers have documented length of surgery as a significant predictor of pressure ulcer development, inconsistencies regarding the "critical" time required on the OR table to positively predict ulcer formation varies from greater than 4 hours²⁷ to greater than 8 hours.^{28, 29} Researchers have demonstrated that on a standard OR table, mean external pressures below bony prominences exceed (sometimes double) the mean capillary pressure and subsequently obstruct blood flow to the tissues.^{30, 31} Unfortunately, the ability to obtain repeated measurements of interface pressures to identify surfaces at risk in critically ill patients has been plagued by technical difficulties. Consequently, research in this area is minimal.¹⁷

Although externally applied pressure is considered to be the primary cause of pressure ulcer formation, altered perfusion significantly increases the tissues' susceptibility to external pressure.^{17, 29} Adaptation to acute ischemia may be less effective than adaptation to chronic ischemia due to a lack of compensatory mechanisms that develop over time. Accordingly, patients who require IABP support for acute ventricular dysfunction and the

inability to wean from cardiopulmonary bypass surgery may have an increased probability for pressure ulcer formation because of the acuteness and severity of their ischemic insult. In response to cardiogenic shock, blood is shunted away from "nonvital" organs (i.e., skin) to maintain perfusion to other organ systems such as the brain and heart. Although the incidence of pressure ulcers in CVS patients has been documented to be between 16% to 27%,^{21, 26, 32, 33} the occurrence of pressure ulcers in the CVS population with IABP support is not reported in the literature.

According to the research facility's outcomes data, univariate logistic regression revealed that pressure ulceration ($\chi^2 [1, n = 157] = 48.99, P = <0.000001$) and IABP ($\chi^2 [1, n = 162] = 38.30, P = <0.00001$) were significant variables that extended length of stay more than 8 days following aortocoronary bypass surgery at this facility. Multivariate logistic regression analysis revealed that aortocoronary bypass patients ($n = 1050$) who developed skin breakdown ($n = 72$) between June of 1992 and December of 1994 were 4.5 times more likely to have an extended length of stay (more than 8 days, $P < 0.001$), and patients requiring IABP ($n = 75$) were 2.4 times more likely to have an increased length of stay ($P = 0.03$).

Prolonged restricted mobility during the postoperative period further complicates the situation. Because these patients are prone to repeated episodes of hypoperfusion and ischemia, they are usually unable to tolerate a regular turning schedule to relieve pressure. Postoperative factors that perpetuate immobility, such as mechanical ventilation, resection, hemodynamic instability, open sternum, multiple surgical procedures,^{34, 35} use of chemical or physical restraints, immobilizing devices, and the mere presence of the IAB catheter in the femoral artery can extend exposure to external mechanical forces (i.e., pressure, friction, and shear). Limb ischemia, the most common complication of IABP therapy that occurs in 7% to 49% of patients, reduces tissue tolerance and may contribute to pressure ulcer formation in the affected extremity.³⁶

Historically, prevention and treatment of pressure ulcers have primarily been a nursing responsibility.¹⁷ Early identification of high-risk populations using a validated, systematic assessment tool is essential to pressure ulcer

prevention.⁸ Strategies must be directed towards maintaining and improving skin tolerance to pressure in addition to providing protection against adverse external mechanical forces.¹ Preventive measures described by the Agency for Health Care Policy and Research (AHCPR) include periodic reassessment, scheduled repositioning, avoidance of moisture, friction, and shear, application of pressure-reducing or pressure-relieving surfaces, and nutritional support.¹ Despite the prevalence of pressure ulcers in critically ill patients, interventions to promote skin integrity and reduce pressure are usually not initiated until late in the patient's hospitalization or after skin breakdown has occurred.¹⁷

Pressure-reducing or pressure-relieving devices are prescribed frequently as part of a comprehensive skin care program. The rationale for using a support surface is based on the principle that pressure ulcer prevention requires the reduction or elimination of interface pressures to a level below that of the capillary closing pressure. A pressure-reducing device is a support surface that redistributes pressure away from the bony prominences but does not necessarily reduce interface pressures below the capillary closing pressure.² In comparison, a pressure-relieving device consistently reduces interface pressures below the capillary closing pressure.² Because the majority of healthcare reimbursement is based on fixed, negotiated rates, the clinical effectiveness of costly products marketed as pressure-relief devices is being questioned. Some clinicians believe that the costs of preventive measures outweigh the costs associated with treatment of a pressure ulcer.³¹ For example, low air-loss (LAL) beds designed to maintain low-interface tissue pressure (12 to 45 mm Hg), have an average daily rental price of \$80 to \$150.⁶ In comparison, the average daily cost in 1989 for inpatient treatment of pressure ulcers at one acute care facility was \$80.42.³ Although there are few studies that support the clinical effectiveness of LAL therapy as a preventive measure for pressure ulcers,⁶ nursing care standards recommend placing the patient with mechanical assistance or IABP support on LAL therapy. Despite the fact that pressure ulcer development in the CVS population with IABP has not been substantiated in the literature, aggressive and expensive preventive measures are advocated.

Because pressure-relief devices are used frequently to promote skin integrity, research is needed to determine the effectiveness of these products at preventing pressure ulcers in specific patient populations.

Statement of the Problem

Since 1990, standard beds at this facility have had pressure-reducing foam core mattresses. Although LAL beds were available as a preventive measure, usage was inconsistent. Consequently, hospital expenditures for specialty beds were astronomical in an era of decreasing reimbursement.

All CVS patients admitted in August of 1992 who spent more than 5 hours on the OR table or required vasoactive infusions and IABP ($n = 22$) in the postoperative period were evaluated by a certified telerostomal therapy nurse for pressure ulcer formation. Forty percent of these patients ($n = 9$) developed various stages of pressure ulcers according to the classification system in Table 1. After examining several case scenarios of IABP patients who had survived but developed Stage III or IV pressure ulcers, the postoperative management protocol for specialty bed usage was modified.



Stage I	Nonblanchable erythema of intact skin.
Stage II	Partial-thickness skin loss involving epidermis, dermis, or both; the ulcer is superficial and presents clinically as an abrasion, blister, or shallow crater.
Stage III	Full-thickness skin loss involving damage to or necrosis of subcutaneous tissue that may extend down to, but not through, underlying fascia; presents clinically as a deep crater with or without undermining of adjacent tissues.
Stage IV	Full-thickness skin loss with extensive destruction, tissue necrosis, or damage to muscle, bone, or supporting structures; undermining and sinus tracts also may be associated with this stage.

From Bergstrom N, Benness MA, Carlson CE, et al. Treatment of Pressure Ulcers. Clinical Practice Guideline, No. 15. Rockville, MD, US Department of Health and Human Services, Agency for Health Care Policy and Research. AHCPR Publication No. 95-0652, 1994.

Beginning in 1994, CVS patients with IABPs were routinely placed on an LAL bed for ulcer prophylaxis immediately following the operative procedure. After IABP removal, patients without pressure ulcers were transferred to a standard bed. From January 1994 through December 1995, almost 700 CVS patients required IABP support. Other practice changes implemented in 1994 included the addition of a full-time physician-intensivist in the Cardiovascular Recovery Room and the development of an interdisciplinary practice team that focused on "best-practice" issues, including early nutrition and metabolic support, prevention of hypoperfusion and organ insult, and aggressive treatment of interstitial edema to facilitate diffusion of nutrients and wastes.

From April through June 1995, the emergency therapy nurse, Cardiovascular Clinical Educator, and the Cardiovascular Clinical Nurse Specialist conducted a nonrandomized, correlational study. Twenty-one percent (6/29) of the CVS patients with IABP developed pressure ulcers, which represented a 50% reduction from the 1992 findings. Nevertheless, the 1995 prevalence of pressure ulcers for all ICU patients in this facility was 8.7%, which was considerably less by comparison. Fisher's exact test was used to examine the differences in pressure ulcer incidence in CVS patients with IABP who were placed on an LAL bed immediately following surgery and those placed on a standard bed. Although no direct inferences could be made because of the small sample size, there did not appear to be a difference in the proportion of patients developing pressure ulcers who received LAL therapy (19%, 4/21) and those who did not (25%, 2/8) ($P = 1.0$).

In 1995, hospital costs for specialty beds exceeded the \$480,000 budget by 25% and approximately \$100,000 of the total amount was spent on CVS patients with IABP for prevention of pressure ulcers. In response to these costs, immediate action was taken. The authors designed a randomized, prospective, interventional study to evaluate the clinical effectiveness of an LAL bed as a preventive device against pressure ulcer complications in CVS patients with IABP. Data collection began December 1, 1995 and continued until May 31, 1995. The purpose of this discussion is to present the results of the pilot study and the preliminary data.

Conceptual Framework

According to the AHCPR,¹ "a pressure ulcer is a localized area of tissue necrosis that develops when soft tissue is compressed between a bony prominence and an external surface for a prolonged period of time." Mechanical damage to tissues occurs most commonly at bone-tissue interfaces (i.e., occiput, sacrum, heel, and trochanter), where the highest pressures are generated in supine and lateral positions. Likewise, the smaller the surface area over which the external pressure is distributed, the greater the insult to the underlying tissue.⁴⁶ The National Pressure Ulcer Advisory Panel has developed a classification or "staging" system that describes the varying depths of tissue damage related to pressure (see Table 1).

The pathogenesis of a pressure ulcer involves the progression of pressure, ischemia, and necrosis.⁴⁶ As illustrated in Kosiak's inverse "time-pressure" theory, if external pressure, exerted by either direct compression or shearing force, exceeds capillary closing pressure (12 to 33 mm Hg), capillary occlusion diminishes or obstructs blood flow to the tissues.^{28, 44} Ischemia, defined by West,³⁵ is the reversible cellular injury that occurs when tissue demand for oxygen exceeds the supply. Oxygen supply and demand imbalance results in anaerobic metabolism, decreased production of adenosine triphosphate and protein synthesis, accumulation of free fatty acids, increased production of lactate, sodium-potassium pump dysfunction, and intracellular edema.^{34, 36, 40, 42, 43} A simplified model developed by Goode and Allman⁴⁵ that illustrates the pathogenesis of pressure ulcers is presented in Figure 1.

Pathophysiologic events that occur during ischemia range from potentially reversible injury to the irreversible destruction and necrosis of all cellular components in a tissue area.³² Cellular damage is proportional to the intensity and duration of applied pressure, the rate of onset, the presence of collateral circulation, and the metabolic demands of a particular tissue.^{40, 42} Duration of pressure rather than intensity is more significant to the development of a pressure ulcer.⁴⁰ Kosiak found that pressures of 60 to 70 mm Hg continuously applied for 1 to 2 hours produced irreversible

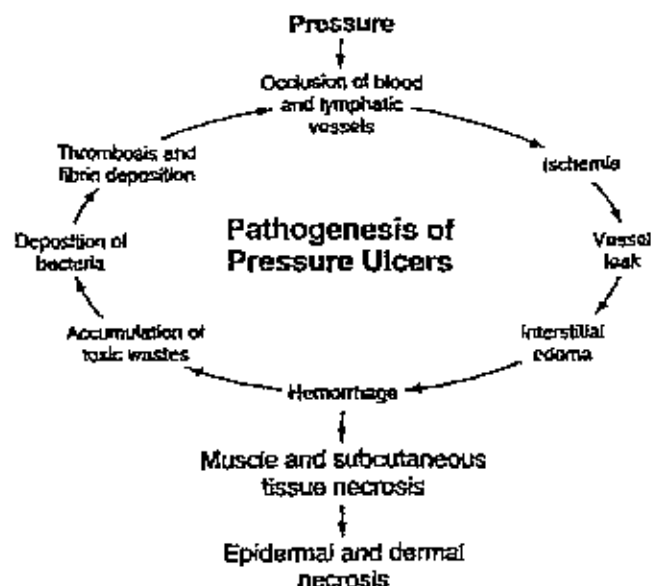


Figure 1. Pathogenesis of pressure ulcers. (From Goode P, Allman R: The prevention and management of pressure ulcers. *Med Clin North Am* 73:6, 1989.)

cellular changes while tissues that experienced pressures three times higher for up to 3 hours with intermittent pressure relief had consistently less or no cellular damage.²⁰

According to Braden and Bergstrom,²² decreased sensory perception and immobility increase risks of pressure ulcer development. In addition to duration and intensity of pressure, the researchers identified tissue tolerance as a critical component in the etiology of pressure ulcers. Tissue tolerance refers to the ability of the skin and its supporting structures to endure the effects of pressure over time without adverse outcomes and is determined by intrinsic and extrinsic factors. Intrinsic factors are those that influence the integrity of the tissue's supporting structures and are composed of cells, vascular and lymphatic systems, interstitial fluid, collagen, and elastin. In comparison, advanced age, malnutrition, and hypoperfusion are intrinsic factors that adversely affect the tissue's support structures.

Extrinsic factors that decrease tissue tolerance to pressure include exposure to moisture, friction, and shear. Friction between the patient's skin and the bed surface is most com-

monly experienced by populations that cannot lift (or be lifted) sufficiently, such as elderly patients, paraplegics, and orthopedic patients. Superficial skin abrasions and damage to the vasculature and lymphatics caused by friction and shearing forces can be minimized by using a draw sheet and total assistance to reposition or transfer patients.

Additional factors that have been hypothesized to diminish tissue tolerance include emotional stress, alterations in body temperature, increased interstitial fluid volume, and smoking.²⁴ Hypothermia, used during CVS to decrease metabolic demand for organ protection, is associated with peripheral vasoconstriction and decreased tissue perfusion. Conversely, hyperthermia during the postoperative period increases metabolic demand and may perpetuate hypoperfusion as a result of vasodilatation in the postoperative period. Regardless of the direction, temperature extremes may decrease tissue tolerance to external pressure and increase risks for pressure ulcer development. Extravascular shifting of fluids following cardiopulmonary bypass surgery results in an increased amount of interstitial edema that extends the distance between

the capillary and cells. Consequently, diffusion of cellular nutrients and metabolic wastes is compromised and the tissues become more susceptible to ischemic injury.²⁷

CVS patients with IABP support have multiple risk factors contributing to pressure ulcer formation. Prolonged hypoperfusion as a result of severe ventricular dysfunction in combination with the other risk factors previously described impairs the tissue's intrinsic mechanisms to tolerate an extended period of immobility. Interventions to prevent pressure ulcer formation should be directed towards supplementing the intrinsic factors that promote skin integrity (i.e., nutrition, hemoglobin, oxygen, and cardiac output) and eliminating harmful extrinsic factors (i.e., external pressure, friction, and shearing).

Review of the Literature

Stotts and Paul²⁸ performed a secondary analysis of pressure ulcer predictors in 117 cardiovascular and neurosurgical patients to determine which critical variables would be predictive of pressure ulcer development. Three studies were conducted comparing operative, risk, nutritional status, and general status in patients who developed pressure ulcers and those who did not. Data were collected until discharge or for the first 3 weeks of hospitalization, whichever occurred first. Lymphocyte counts ($U = 1.0$, $P < 0.05$) and modified Norton Scale scores ($U = 225.0$, $P < 0.01$) were significantly different between the groups. Patients who developed ulcers had much higher lymphocyte levels (19% versus 1.9%) and lower pressure ulcer risk scores using the Norton Scale ($x = 15.9$, $SD = 3.57$ versus $x = 18.2$, $SD = 2.57$) than those whose skin remained intact. Although surgical patients who developed ulcers had a larger estimated blood loss (892 mL versus 315 mL; $t = 58.5$, $P < 0.01$), hemoglobin or hematocrit values were not significantly different. Nutritional risks (i.e., albumin and total protein), health status (i.e., age, weight, and temperature), and operative risks (i.e., type, length, and number of procedures performed) were not significant predictors of pressure ulcers. These findings should be interpreted with caution due to the small sample size.

Olson et al²⁹ prospectively studied the incidence of pressure ulcers and factors related

to their development in 149 medical-surgical patients admitted to an acute care setting. Patients who acquired pressure ulcers ($n = 20$, 13.4%) were found to have lower hemoglobin levels (12.6 versus 14.0 g/dL, $t = 2.17$, $P = 0.03$), spent more time in bed (22.6 versus 20.3 h, $t = 2.17$, $P = 0.0001$) and less time sitting in a chair (1.5 versus 3.1 h, $t = 3.2$, $P = 0.002$) when compared with patients who did not develop ulcers. Backward stepwise logistic regression analysis revealed that lower hemoglobin and hours spent in bed were predictors of pressure ulcer development ($X^2 = 9.306$, $df = 2$, $P = 0.0095$). Although the subjects who developed ulcers were older, smoked more, had a lower admission weight and lymphocyte count, these factors were not statistically different. Interestingly, cardiovascular medical-surgical patients and general surgical patients had the highest incidence of pressure ulcers (26.2 and 21.6, respectively). More than any other group, cardiovascular patients developed the largest number of Stage II ulcers.

Kemp et al²⁷ conducted a prospective, descriptive study to identify preoperative and intraoperative predictors of pressure ulcers. Fifteen of 125 (12%) surgical patients developed a total of 23 pressure ulcers most commonly located on the heels, elbows, and sacrum. Seventy percent of the pressure ulcers were identified at the end of the surgical procedure when patients were being transferred off the OR table. By discharge or postoperative day ten, 61% of the ulcers had resolved. Using discriminant function, age, time on OR table, and extracorporeal circulation were determined to be significant predictors of pressure ulcer formation ($X^2 [3, n = 125] = 16.12$, $P = 0.001$). The authors reported the multivariate model to be 80% sensitive and 76% specific for predicting pressure ulcers. Elderly patients (aged 75 years or older) who remained on the OR table for at least 8 hours and had a procedure requiring cardiopulmonary bypass (i.e., CVS) were most likely to develop pressure ulcers. Serum albumin, total protein, 20 postoperative Braden scores were not significantly different between patients who developed pressure ulcers and those who did not. Although the results were not statistically significant by univariate analysis, patients who developed pressure ulcers tended to have lower preoperative Braden scores (20)

versus 21.51, $P = 0.186$) and a preoperative albumin less than 3g/dL ($P = 0.039$). Because the majority of pressure ulcers were detected immediately following the surgical procedure, the researchers did not consider postoperative Braden scores to be useful in the surgical population.

Papantonio et al²⁸ examined the incidence and risk factors related to the development of sacral pressure ulcers following elective cardiac surgery. The subjects ($n = 136$) were assessed eight times during a 6-day period starting on the day of surgery. Sacral ulcers developed on 27% ($n = 37$) of the subjects, of which 43% ($n = 16$) remained in Stage I and 57% ($n = 21$) progressed to Stages II or III. Ulcers of greater severity tended to appear sooner; 43% of ulcers classified as Stage II or III developed before 72 hours. Patients who developed sacral ulcers were older (over 60 years), had a higher incidence of diabetes and respiratory disease, were transferred from another facility, and had lower hematocrit (less than 38%) and serum albumin levels (less than 3.7 g/dL).

Although cardiopulmonary bypass time was not a significant variable, patients who developed pressure ulcers spent a longer amount of time on the OR table. Interestingly, patients who had combination or repeated surgical procedures were twice as likely to develop ulcers. Intraoperative variables, including patient position on OR table, and esophageal and rectal temperatures were not found to be significant predictors of ulcer formation. Except for nitroprusside, which provided protection against pressure ulcer formation, the use of vasoactive medications intraoperatively was not significant. Nevertheless, patients who received vasoconstrictive medications were automatically placed on static air mattresses. Despite a 27% incidence of sacral pressure ulcers, only 13% of the sample never received any pressure-reduction therapy during the postoperative period. Except for the use of artificial sheepskin, which was not significant, data comparing the incidence of pressure ulcers and the type of bed surface used were not reported. Although patients who developed ulcers typically were receiving pressure-relief products, the authors speculated that the products were initiated too late in the postoperative period to be effective. As a result of the study findings,

practice changes were implemented to provide all CVS patients having extended immobility with either an air-static mattress or an LAL bed. Researchers recommended adoption of a risk assessment tool for planning ulcer prevention and the use of pressure-reducing surfaces on OR tables.

Statement of Purpose

Specific aims of the pilot study were (1) to determine the incidence of pressure ulcer development in CVS patients with IABP, (2) identify preoperative, intraoperative, and postoperative factors that can be used in the main study to predict pressure ulcer formation, (3) determine the estimated magnitude of effect of LAL beds and calculate the sample size needed for the main study, (4) establish inter-rater reliability between investigators for Acute Physiology and Chronic Health Evaluation (APACHE) II and Braden Scale scores, and (5) establish correlation between APACHE II and Patient Identification for Rotational Therapy (PIRT) scores.⁸

Methods

Patient or family written consent was deemed unnecessary by the Human Subjects Review Committee because the study protocol did not require deviations from the normal standard of care. A two-group, randomized, quasiexperimental design¹⁴ was used as the methodologic framework for the pilot study to determine the incidence of pressure ulcer development in CVS patients with IABP support when placed on a standard or LAL bed. All CVS patients who received an IABP in OR for failure to wean from cardiopulmonary bypass were eligible to be entered into the study. Patients who received an IABP in OR on an even day were placed on an LAL bed (experimental) while those who received an IABP in OR on an odd day were placed on a standard bed (control).

The pilot study was conducted in the cardiovascular recovery room of a 956-bed, private, not-for-profit, teaching hospital located in a large metropolitan area in the south central United States. Approximately 200 to 300 adult patients are admitted to CVRR monthly who have undergone various cardiovascular

or cardiothoracic surgical procedures. Of these patients, roughly 10% require IABP support in the postoperative period. Skin assessments and data collection were performed daily by either the enterostomal therapy nurse, Cardiovascular Clinical Educator, or Cardiovascular Clinical Nurse Specialist beginning on the day of surgery and continuing until 24 hours after IABP removal. Settings for the individualized sac pressures on the LAL beds were monitored daily for appropriateness. Patients were initially assessed for breakdown within 16 hours of admission into CVRR. Pressure ulcers were staged by the enterostomal therapy nurse. Acceptable inter-rater reliability (ranged from $r = 0.95$ to 0.99) for Braden, PIRT, and APACHE II scores was established between the three investigators was established with the first five subjects and every fifth subject thereafter. For the pilot phase of the study, data were collected December 1, 1995 through May 31, 1996. The decision to collect preoperative, intraoperative, and postoperative variables was based on an extensive review of the literature, which has been presented (Tables 2 to 6).

Instruments

Instruments used in the pilot study included a standard bed, an LAL bed, the Braden Scale, and APACHE II. Standard beds in CVRR have a pressure-reducing foam mattress replacement product designed to reduce interface pressures, although not necessarily below the capillary occlusion pressure. The mattress replacement is a polyurethane foam product designed to promote equal distribution of weight away from the bony prominences and is covered by a waterproof fabric to reduce the potential of liquid accumulation inside the mattress. Manufacturers report that sacral interface pressures range from 14 to 26 mm Hg on a new mattress. However, researchers evaluating the effectiveness of "in-use" mattresses produced by the same manufacturer documented sacral-coccygeal interface pressures ranging from 33 to 35 mm Hg and heel interface pressures ranging from 34 to 38 mm Hg. New mattresses were distributed throughout the hospital during July of 1995 and have a limited 5-year warranty. Replacement mattresses are intended to be used for several years and costs generally range from

\$50 to \$60. Since their distribution, the products' pressure-reducing capabilities over time have not been monitored.

LAL beds used in the study had 16 compartmentalized air sacs that were separately controlled to reduce the interface pressure below the capillary occlusion pressure (i.e., pressure-relieving device). Manufacturers of this LAL therapy state that interface pressures of sacral and heel areas range from 8 to 10 mm Hg.⁴⁶ The nylon quilted fabric cover prevents little air passage and decreases friction. Daily rental for the LAL bed is \$80 plus an initial electrical safety inspection charge of \$16. Additional features of the specialty bed, including pulsation or lateral rotation, were not used for the duration of the study.

To identify patients at risk for developing pressure ulcers, the Braden scale was selected for the pilot study.⁹ The instrument is composed of six subscales reflecting sensory perception, skin moisture, physical activity, nutritional intake, friction and shear, and patient ability to change or control body position. Each subscale has a rating and a description of criteria for assigning that rating. Potential scores range from 6 to 23; lower scores indicate a greater risk of pressure ulcer formation. Predictive validity and reliability of the instrument have been determined in the adult ICU population. Inter-rater reliability between registered nurses was $r = 0.99$ ($P < 0.0001$). A Braden score of 16 has been documented to be 100% sensitive and 90% specific for predicting pressure ulcer formation. Construct validity of the scale and conceptual schema are still under evaluation. Overall, the Braden Scale has undergone extensive psychometric evaluation compared with other risk assessment tools for predicting pressure ulcers.

The APACHE II scoring system¹⁸ was used in the pilot study to measure the severity of illness in subjects. Twelve physiologic variables that are routinely evaluated in critically ill patients are categorized according to their value. Values are assigned scores that range from 0 (normal) to 4 (most abnormal). The total score of all the variables, including age and previous health status, is used as an acuity indicator for severity of disease; the higher the score, the greater the acuity and potential for death. APACHE II has been extensively validated as a measurement of patient acuity in many ICU populations within the United

Variable	Control (n = 20)		Treatment (n = 16)		P value
	X or f (%)	SD	X or f (%)	SD	
Age (years)	69	2.31	67	5.51	0.17+
Gender					
Male	17 (85%)		8 (56%)		0.07†
Female	3 (15%)		7 (44%)		
Ethnic group					0.55‡
Caucasian	16 (80%)		13 (81%)		
Hispanic	3 (15%)		2 (13%)		
Black	0		1 (6%)		
East Indian	1 (5%)		0		
Preoperative albumin (g/dL)	3.97 (n = 18)	0.65	3.08 (n = 13)	0.59	0.001§
Ejection fraction (%)	36 (n = 16)	12.82	42 (n = 14)	13.70	0.214§
Preoperative/intraoperative CPE	1 (5%)		2 (12.5%)		0.57†
APACHE POD 1	24 (n = 18)	8.09	27 (n = 11)	8.53	0.34§
PIRT POD 1	30 (n = 17)	6.43	32 (n = 11)	6.70	0.84§
Bradycardia POD 1	9.45	1.09	9.88	1.49	0.58§
Comorbid conditions	4.10	1.37	4.75	2.11	0.29§
Preoperative LOS	1.5	2.16	2.0	2.85	0.554§
Skin breakdown (acute phase)	3 (15%)		3 (18.8%)		1.00†
Skin breakdown (late)	1 (5%)		2 (12.5%)		0.57†

*n = 36.

†Fisher's exact test.

‡Chi-square.

§T-test independent samples.

Late = POD 6 until discharge; acute phase = POD 1-4; LOS = length of stay; X = mean; f = frequency.

Variables	Control (n = 20)		Treatment (n = 16)		P value
	f (%)	f (%)	f (%)	f (%)	
Comorbidities					
CAD	20 (100)		15 (94)		0.44†
HTN	12 (60)		11 (69)		0.58‡
PVD	3 (15)		4 (25)		0.67†
CVA	3 (15)		4 (25)		0.67†
MI	11 (55)		10 (63)		0.65‡
Renal	3 (15)		2 (13)		1.00†
COPD	5 (25)		2 (13)		0.42†
DM	9 (25)		0		0.00†
CV surgical procedure					0.40†
ACB	13 (65)		14 (88)		
Valve repair/replacement	1 (5)		0		
ACB + valve	5 (25)		2 (13)		
Aneurysm repair	1 (5)		0		
Emergency surgery	2 (10)		5 (31)		0.20†
Reoperation	10 (50)		5 (31)		0.25†
Return to OR	5 (25)		6 (38)		0.48†
Open sternum	4 (20)		3 (19)		1.00†

*n = 36.

†Fisher's exact test.

‡Chi-square test.

CHF = congestive heart failure; CAD = coronary artery disease; AMI = acute myocardial infarction; HTN = hypertension; PVD = peripheral vascular disease; CVA = cerebral vascular accident; MI = myocardial infarction; COPD = chronic obstructive pulmonary disease; DM = diabetes mellitus; CV = cardiovascular; ACB = aortic coronary bypass; OR = operating room; f = frequency.

Variable	Pressure Ulcers (n = 6)		No Pressure Ulcers (n = 30)		P value
	X or f (%)	SD	X or f (%)	SD	
Age (years)	68	4.05	62	11.36	0.04*
Gender					
Male	6 (83%)		21 (70%)		
Female	1 (17%)		9 (30%)		0.65*
Ethnic Group					
Caucasian	4 (67%)		25 (83%)		
Hispanic	1 (17%)		4 (13%)		
Black	1 (17%)		0		
East Indian	0		1 (3%)		0.14†
Preoperative albumin (g/dL)	3.7	0.91	3.5 (n = 25)	0.74	0.68*
Ejection fraction (%)	35	11.84	40 (n = 25)	13.68	0.41*
Preoperative/intraoperative CPR	0		3 (10%)		1.00‡
APACHE POD 1	32 (n = 5)	9.88	24 (n = 25)	7.38	0.04*
PIRT POD 1	37 (n = 5)	4.08	30 (n = 23)	6.13	0.01*
Braden POD 1	9	1.22	10	1.19	0.02*
IABP days	6.56	2.58	5.03	2.67	0.17*
No. of comorbidities	5.33	1.50	4.20	1.75	0.14*
Skin intact—preoperatively§	6 (100%)		30 (100%)		1.00*
Low air-loss therapy	3 (50%)		3 (50%)		1.00*
Total LOS	17.0	10.77	21.4 (n = 29)	16.27	0.53*
Preoperative LOS	3.0	3.22	1.46	2.27	0.16*

Comparison of descriptive data between subjects who developed pressure ulcers and those whose skin remained intact (n = 36).

* Test independent samples.

† Chi-square.

‡ Fisher's exact test.

§ Information obtained from preinduction intraoperative nursing assessment form.

X = mean; SD = standard deviation; f = frequency; LOS = length of stay; CPR = cardiopulmonary resuscitation; IABP = intra-aortic balloon pump.

States and abroad.²⁸ The PIRT classification system has recently been proposed as a tool that can be used to identify ICU patients who would benefit from continuous lateral rotation therapy (CLRT).⁸ PIRT was developed using the APACHE II as the framework and is undergoing psychometric evaluation. It is foreseeable that the clinical effectiveness of combination LAL therapy and CLRT for CVS patients with IABP will be evaluated in future studies; therefore, the researchers attempted to establish construct criterion validity of PIRT during the pilot study.

Statistical Analyses

Statistical analyses were performed using the Statistical Package for the Social Sciences⁹ for Microsoft Windows Release 6.1 (1991). Descriptive statistics were used to evaluate preoperative, intraoperative, and postoperative variables between treatment and control

groups and between subjects who developed pressure ulcers and those who did not. Because the sample size was small and had unequal groups, comparison of the two independent samples with respect to a dichotomous dependent variable was performed using the Fisher's exact test. Likewise, nonparametric Mann-Whitney U tests were used to examine the distributions among the groups in regard to categorical or ranked data. Prior to comparing the differences between group means for interval-ratio level data, Levene's test was performed to test the assumption of homogeneity of variance. Independent sample t-tests, using either equal or unequal variance as appropriate, were performed on interval level data to explore the differences among subjects regarding the development of pressure ulcers. The level of significance was set at $P < 0.05$ (two-tail). Due to the small sample size, univariate or multivariate logistic regression analysis was not feasible for the

Variable	Pressure Ulcers (n = 6)			No Pressure Ulcers (n = 30)			P Value
Comorbidities							
CAD	5 (83%)			30 (100%)			0.16*
HTN	3 (50%)			20 (67%)			0.64*
PVD	2 (33%)			5 (17%)			0.57*
MI	3 (50%)			18 (60%)			0.67*
CVA	4 (67%)			3 (10%)			0.00*
Renal INS	3 (50%)			2 (7%)			0.02*
COPD	0			7 (23%)			0.31*
DM	2 (34%)			7 (23%)			0.42†
CV Surgical Procedures							0.75†
ACB	4 (67%)			23 (77%)			
ACB and valve	2 (33%)			5 (17%)			
Valve repair/replacement	0			1 (3%)			
Aneurysm	0			1 (3%)			
Emergent	1 (17%)			6 (20%)			1.00*
Reoperation	4 (67%)			11 (37%)			0.20*
Return to OR	3 (50%)			8 (27%)			0.34*
Open sternum	3 (50%)			4 (13%)			0.07*
Duration of OR	X	SD	ME	X	SD	ME	t-test (p)
Anesthesia time (min)	335	167.43	310	280	81.2	263	0.43
CPB (min)	127	87.89	97	86	37.73	76	0.29
Cross clamp (min)	60	27.49	62	51	24.84	44	0.40

Comparison of admission diagnoses, comorbidities, and surgical procedures between subjects who developed pressure ulcers and those whose skin remained intact.

Note: Percentage may not equal 100% due to rounding error.

* Fisher's exact test.

† Chi-square test.

CAD = coronary artery disease; HTN = hypertension; PVD = peripheral vascular disease; MI = myocardial infarction; CVA = cerebrovascular accident; INS = insufficiency; COPD = chronic obstructive pulmonary disease; DM = diabetes mellitus; AMI = acute myocardial infarction; CHF = congestive heart failure; CV = cardiovascular; ACB = aortocoronary bypass; OR = operating room; CPB = cardiopulmonary bypass; ME = median; X = mean; f = frequency.

pilot study. Construct validity for the PIRT instrument was established ($r \geq 0.95$) using Pearson's product moment correlation to measure the degree of association between paired PIRT and APACHE II scores. Evidence of adequate inter-rater reliability for data collection and calculation of APACHE II, PIRT, and Braden scores was set at $r \geq 0.95$.

Sample

Thirty-six adult CVS patients requiring IABP for failure to wean from cardiopulmonary bypass surgery were consecutively entered into the pilot study from December 1, 1995 through May 31, 1996. Five other patients who initially met inclusion criteria were not entered into the study due to a breach in the research protocol. All 36 patients completed the pilot study. Subjects were predominantly Caucasian (81%), male (72%), and had an average of four comorbidities at the time of admission into CVRR. Age in years extended

from 47 to 84 with a mean of 63 years. Unstable angina (50%) or coronary artery disease (19%) was the admitting diagnosis for the majority of the subjects, for which 75% underwent aortocoronary bypass surgery. Forty-two percent of the subjects had a reoperation and 19% were classified as requiring emergent surgical intervention by the cardiovascular surgeon. The average preoperative length of stay was 1.7 days.

To confirm the severity of the subjects' acuity and risks of developing pressure ulcers, the initial mean APACHE II score obtained on the first postoperative day (POD 1) was 25 and the initial mean Braden score on POD 1 was 9.56. Demographic data are summarized in Tables 2 and 3 for the treatment and control groups. Except for the preoperative albumin level, which was significantly lower in the treatment group, there were no major differences between the groups with regard to age, ejection fraction, number of comorbidities, preoperative hospitalization of stay (LOS), or

Variable	Skin Breakdown (n = 16)		No Skin Breakdown (n = 30)		P value
	X or f (%)	SD	X or f (%)	SD	
Nutritional Support					
POD 1	2 (33%)		9 (30%)		1.00*
POD 2	4 (67%)		14 (47%)		0.05*
POD 3	5 (63%)		10 (55%)		
Lactic Acid (mg/dL)					
POD 1	68	35.96	52 (n = 28)	29.35	0.265†
POD 2	65.2 (n = 5)	54.20	35.73 (n = 26)	31.17	0.09†
POD 3	22.4	7.16	20.52	10.63	0.71†
Hemoglobin (g/dL)					
POD 1	8.78	1.85	10.69	1.70	0.01†
POD 2	10.26	1.26	11.00	1.74	0.28†
POD 3	11.01	0.64	11.38	1.76	0.40†
Albumin (g/dL)					
POD 1	2.30	0.48	2.59 (n = 28)	0.51	0.36†
POD 2	2.5 (n = 4)	0.49	2.66 (n = 14)	0.46	0.74
Creatinine (mg/dL)					
POD 1	3.04	2.34	1.43	0.80	0.20†
POD 2	2.98	2.24	1.44 (n = 29)	0.88	0.15†
POD 3	3.06 (n = 5)	1.98	1.47 (n = 27)	0.87	0.00†
Altered LOC					
POD 1	6 (100%)		24 (80%)		0.56*
POD 2	5 (63%)		11 (37%)		0.06*
POD 3	5 (63%)		9 (31%)		0.02*
Repositioned/turned					
POD 1	0.16	0.40	1.80 (n = 27)	3.40	0.25†
POD 2	1.16	1.83	6.16	4.07	0.00†
POD 3	3.16	3.31	7.58 (n = 29)	5.94	0.06†
CPR					
POD 1	1 (17%)		3 (10%)		0.53*
POD 2	0		3 (10%)		1.00*
POD 3	6 (100%)		27 (100%)		1.00*
No. of Vasoactive Infusions					
POD 1	3.16	0.75	2.03	0.85	0.00†
POD 2	2.83	0.75	1.63	0.96	0.00†
POD 3	2.5	0.54	1.41	0.93	0.00†

Comparison of physiologic parameters and interventions on postoperative days 1 to 3 between subjects who developed pressure ulcers and those whose remained intact.

* Fisher's exact test.

† Most independent samples.

LOC = level of consciousness; CPR = cardiopulmonary resuscitation; POD = postoperative day; ME = median; X = mean; f = frequency.

initial APACHE II and Braden scores. The groups appeared to be homogenous.

Findings

Although the results must be interpreted with caution because of the small sample size, there did not appear to be a statistical or clinical difference observed between the treatment (n = 16) and control (n = 20) groups regarding the incidence of pressure ulcer development (P = 1.00) during the early postop-

erative period prior to IABP removal. Three patients (15%) on the standard bed developed a total of five pressure ulcers. Similarly, three patients (19%) who were placed on the LAL bed developed a total of seven pressure ulcers (Figs. 2 and 3). The number of pressure ulcers that developed during the early phase in each group (5 versus 7) was not significantly different. Table 7 summarizes the characteristics of the pressure sores. Altogether, six of the 36 subjects (16.7%) developed a total of 12 pressure ulcers during the early phase of their illness while requiring IABP support.

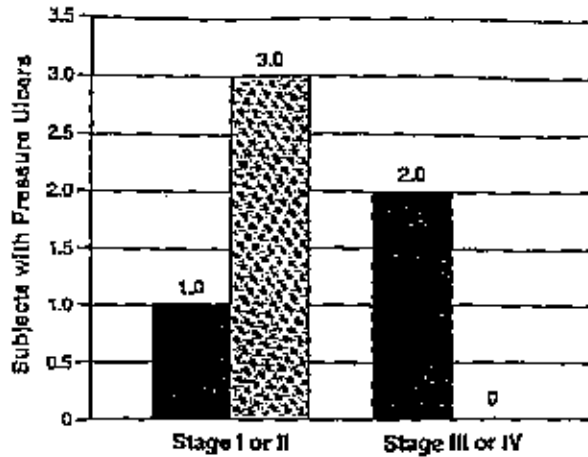


Figure 2. Classification of ulcers. Dark bar = LAL bed; Light bar = standard bed.

An additional three patients developed a total of five ulcers during the late phase of their ICU stay following LABP removal, for a 25% incidence of pressure ulcer formation. Assessing the acute and late postoperative periods together, five subjects (31%) in the treatment group and four subjects (20%) in the control group developed pressure ulcers. Early and late ulcer development did not appear to be significantly different between the groups ($P = 0.46$).

Ten of the ulcers (83%) that developed during the early phase were identified on or before postoperative day four. Of the six subjects who developed pressure ulcers during the early phase, three subjects had bilateral heel ulcers and five subjects developed sacral ulcers. Regardless of early or late development, 16 of the 17 ulcers were located on either the heels or sacrum. Interestingly, the standard mattress was equipped with extra pressure reduction capabilities for the heel

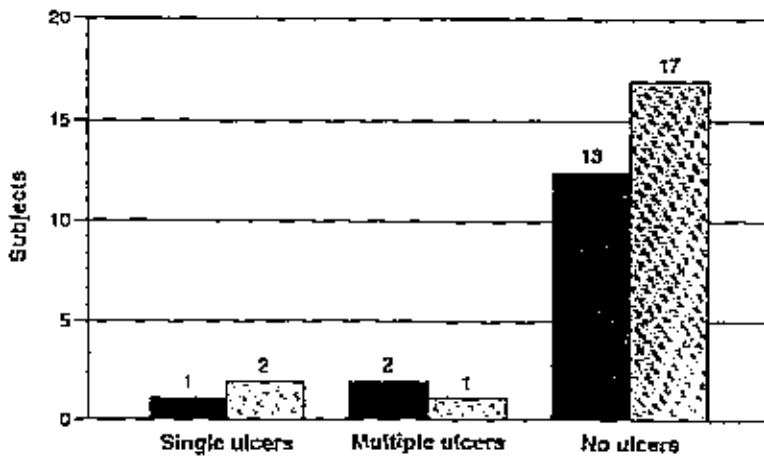


Figure 3. Occurrence of ulcers. Dark bar = LAL bed; Light bar = standard bed.

Subject	Ulcer	Initial Stage Assessment	Location	PODP	Resolving	Progressed to Stage	Status	Bed Type
1	1	I	Sacrum	4†	No	III	DC	LAL
	2	I	L) Heel	4	Yes	NA		
	3	I	R) Heel	6	Yes	NA		
2	1	I	L) Heel	4	No	I	Expired	LAL
	2	I	R) Heel	4	No	I		
	3	I	Sacrum	4	Yes	NA		
3	1	II	Sacrum	4†	No	III	Expired	LAL
4	1	I	L) Heel	2	No	I	Expired	S
	2	I	R) Heel	2	No	I		
	3	I	R) Elbow	2	No	I		
5	1	I	Sacrum	5	Yes	NA	DC	S
6	1	I	Sacrum	4	No	I	Expired	S

*N = 6.

† Posterior surfaces were not evaluated until POD 4 due to hemodynamic instability/open sternum.

PODP = postoperative day of presentation; L = left; R = right; NA = not applicable; S = standard; LAL = low air loss; DC = discharged.

area. Future analysis will determine if unilateral heel ulcers develop in the same extremity as the IABP insertion site. The location of the ulcers probably was associated with prolonged supine positioning. Of the five subjects that expired prior to discharge, all (100%) had developed one or more pressure ulcers ($P = 0.0003$).

Inferential and descriptive statistics for continuous and categorical variables as possible indicators of pressure ulcer formation are presented in Tables 4 through 6. Subjects who had a medical history of cerebrovascular disease ($P = 0.00$) or renal insufficiency ($P = 0.02$) were more likely to develop pressure ulcers postoperatively. Subjects who developed pressure ulcers were an average of 6 years older (68 versus 62) than those whose skin remained intact ($P = 0.04$). Higher APACHE II ($P = 0.04$) and PIRT ($P = 0.01$) scores (greater than 32 and 37, respectively) and a Braden score of nine or less ($P = 0.02$) obtained on the first postoperative day appeared to be predictors of pressure ulcer development. Postoperative variables, including a lower hemoglobin level, a higher serum creatinine level, and an altered level of consciousness were also associated with a greater risk of pressure ulcers. Frequency of repositioning or lateral rotation and number of vasoactive infusions were indicators of pressure ulcer formation. Hence, patients who arrived to CVRR with an open sternum were more

likely to develop a pressure ulcer that probably was associated with complete immobility although statistical significance was not reached ($P = 0.07$). Interestingly, neither intraoperative or postoperative cardiac arrest, length of time on OR table, preoperative albumin level, nutritional support, or type of bed surface appeared to be associated with pressure ulcer development. Number of comorbidities, preoperative length of stay, number of days requiring IABP support, and serum lactate levels on postoperative day two approached statistical significance and may become important predictors of pressure ulcer formation with the addition of more subjects in the study. Coincidentally, total length of stay was not significantly different between those who developed pressure ulcers and those who did not ($P = 0.53$). This finding may be skewed by the fact that five subjects who had pressure ulcers expired prior to discharge. Death usually occurred on or about postoperative day 11 compared to the median total of stay of 14 days. Because the majority of pressure ulcers occurred on or before postoperative day four, variables were analyzed daily from postoperative day one through four.

Using Pearson's product-moment correlation, the association between APACHE II and PIRT scores revealed an $r = 0.89$ ($P = 0.000$, two-tailed). Although these results appear favorable in establishing the psychometric va-

lidity of PIRT, further testing of this instrument is indicated with a larger sample size. PIRT not only quantifies the severity of illness, but may be useful in identifying patients with pulmonary conditions that may benefit from continuous lateral rotation therapy. Surprisingly, CLRT has not demonstrated significant effects on the incidence of pressure ulcers.⁴

Discussion

Pressure-relieving devices are prescribed based on the assumption that pressure must be reduced or eliminated to prevent ulcer formation. Although this assumption is likely true, other preventive measures aimed at supporting the skin's integrity or ability to interface with external pressures may actually be more effective than the support surface in certain situations. For example, metabolic interventions designed to improve oxygen and nutrient delivery to the tissues and promote diffusion of cellular nutrients and wastes warrant further investigation into the effects on pressure ulcer development. Depending on the severity of the pathologic state, pressure ulcers are an inevitable outcome of the patient's progressive deterioration regardless of the type of surface provided.⁴⁵ In these situations, specialty support surfaces may only increase the amount of time that the patient can remain in the same position without irreversible ischemic damage.⁴⁶

Although pressure-reducing surfaces may contribute to pressure ulcer prevention, they do not independently prevent pressure ulcer formation. Instead of being considered a primary intervention for high-risk patient populations, therapeutic surfaces should be used with patient selection criteria in addition to other measures designed to relieve external forces and maintain the integrity of the skin's supporting structures. Furthermore, when an institution implements a support surface, quality initiatives should include ongoing evaluation of new and used product characteristics in regard to interface pressures over specific bony prominences.⁴⁷

Except when using CLRT, patients on LAL beds must be routinely turned and repositioned at least every 2 hours to prevent skin breakdown. Interestingly, in this study, patients on the LAL bed were consistently turned less frequently on postoperative days one

through four than patients on the standard bed. Perhaps some health care providers believe that LAL therapy eliminates or reduces the need for patients to be turned regularly at scheduled intervals. Clearly, these results support the need for improved education and clinical support for health care providers.

Using the findings from previous studies as well as the pilot study, it was determined that LAL therapy would be clinically effective if it were responsible for a reduction in the incidence of pressure ulcers from 25% to 10% in the CVS population with IABP support. Using a large effect size of 0.60 (alpha 0.05, power 0.80, two-tailed), 113 subjects per group are needed for the main study to test the hypothesis that there will be a reduction in the incidence of pressure ulcers due to LAL therapy.⁴⁸ For future analysis, multivariate logistic regression will be used to determine preoperative, intraoperative, and immediate postoperative variables that predict pressure ulcer development in CVS patients with IABP support.

During the early and late postoperative periods, the CVS population with IABP support had a 25% incidence of pressure ulcer development. These observations are similar to those of Kemp et al.¹⁷ and Papanonio et al.¹⁸ who reported a 27% incidence of pressure ulcers in cardiovascular surgical patients who had been placed on extracorporeal circulation. Subjects in this pilot study had a 19% incidence of sacral ulcers ($n = 7$) that was lower than the 27% incidence of sacral ulcers reported by Papanonio et al.¹⁸ Furthermore, in this pilot study 57% (4/7) of the sacral ulcers progressed to at least Stage III.

Because this pilot study began on December 1, 1995, the use of specialty beds in the CVS population with IABP support has decreased due to treatment randomization by 60%. Subsequently, specialty bed costs have been reduced by \$68,000. Despite a reduction in LAL bed usage, the incidence of pressure ulcers has not changed in this patient population.

Similar to the findings of Kemp et al.¹⁷ and Stotts and Paul,²⁰ but unlike those of Papanonio et al.,¹⁸ preoperative albumin level as an indicator of nutritional status was not a predictor of pressure ulcer formation. Other determinants of nutritional status, such as total lymphocyte count and total protein levels, were not available in this study to determine

whether preoperative nutritional status was a significant variable in ulcer formation.

Metabolic support has been a part of the postoperative management protocol for CVS patients with IABP support since 1994. Postoperatively, 70% of our sample did not have any nutritional or metabolic support on the day of surgery, which was usually related to extremely high serum glucose levels or acute renal failure. By postoperative day two, however, 60% of the subjects were receiving nutritional support, typically in the form of a 50% glucose-insulin-potassium solution containing amino acids, trace elements, and multivitamins. To prevent ischemic injury, oxygen and substrate must be delivered to and used by the tissues. Interestingly, subjects who had a lower hemoglobin level on postoperative day one had a higher incidence of pressure ulcer development. In contrast, Stotts and Paul²² determined that blood loss and hypovolemia, rather than hemoglobin, were a significant predictor of pressure ulcers.

Age is commonly considered a risk factor for the development of pressure ulcers. Our findings support those of Kemp et al²³ and Papanonio et al,²⁴ who found that older patients are at greater risk for skin breakdown. In contrast, other researchers, including Hoyman and Gruber,²⁵ Stotts and Paul,²⁶ and Olson et al,²⁷ did not identify age as a significant factor in the development of pressure ulcers.

Although patients who developed pressure ulcers tended to have longer anesthesia, CPB, and cross-clamp or ischemic times during the surgical procedure, there did not appear to be a relationship between these intraoperative variables and the formation of pressure ulcers. Results of this study contradict those of Hoyman and Gruber,²⁵ Kemp et al,²⁶ and Papanonio et al,²⁷ who determined that length of the operative procedure is associated with increased pressure ulcer formation. Presentation of pressure ulcers occurred much later in this pilot study when compared to Kemp et al,²⁶ who detected 70% of pressure ulcers at the end of the surgical procedure.

When evaluating the effects of intraoperative variables, the OR table surface and patient-positioning practices must be considered. The subjects in this pilot study were positioned on a standard 21 by 76-inch OR table with a 2-inch Staph check mattress.

Sterile cotton linen covering a hyperthermia pad (from the thoracic spine to the heels) lay beneath the supine patient. The patients' arms and heels were padded with a 2-inch piece of eggcrate. The occiput rested on a stockinette donut made by the OR staff. It is possible that pressure-reducing devices should be initiated in the OR to provide optimal protection for patients undergoing prolonged surgical procedures. For example, the subjects in this study were noted to have prolonged OR times most likely attributed to difficulty weaning from CPB and hemodynamic instability that required intervention prior to transfer. Further research of the relationship between ulcer risk and pressure-reducing capabilities of OR table pads is warranted.

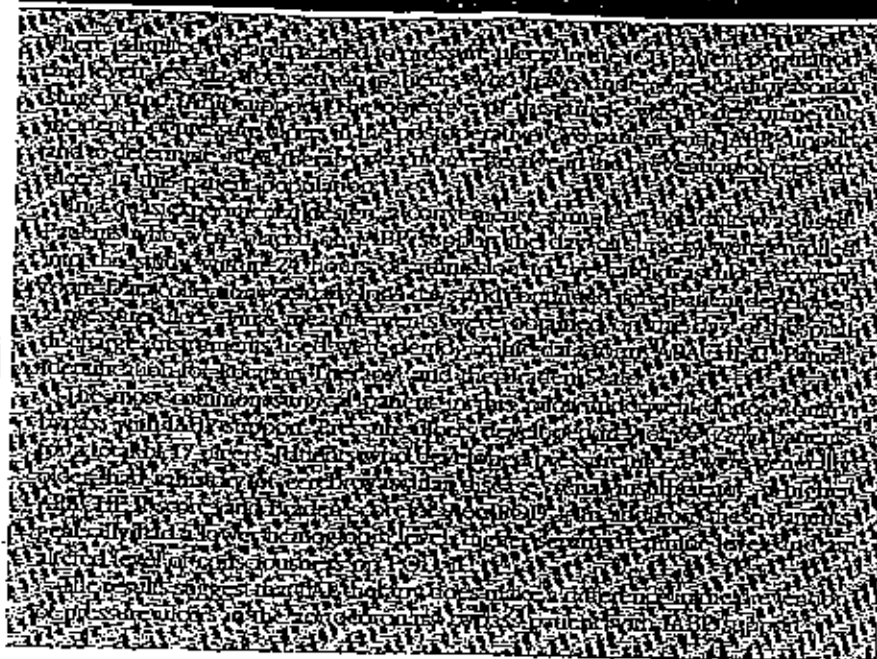
Analogous to the findings of Olson and colleagues,²⁷ patients who developed pressure ulcers required an average of 36 more hours of IABP support, and therefore remained in bed for a longer period of time. Dissimilarly, Papanonio et al²⁴ did not find immobility to be a risk factor of pressure ulcer development. In fact, 67% of patients who developed pressure ulcers in the study were out of bed by 24 to 48 hours after undergoing cardiac surgery.

In summary, a multitude of factors including age, higher severity-of-illness index, altered level of consciousness, number of vasoactive infusions, frequency of repositioning, and renal insufficiency tended to be significantly associated with pressure ulcer development. Additional testing of these variables with larger sample sizes is warranted. Costs of preventing pressure ulcers are substantial. It is financially beneficial to target research-based interventions at high-risk groups to ensure efficient use of resources. Besides the small sample size, other limitations of the study included a lack of data availability on a daily basis to determine risk scores. Results are only generalizable to the CVS population with IABP support.

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SUMMARY



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