

# POSTOPERATIVE PULMONARY DYSFUNCTION IN ADULTS AFTER CARDIAC SURGERY WITH CARDIOPULMONARY BYPASS: CLINICAL SIGNIFICANCE AND IMPLICATIONS FOR PRACTICE

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*Postoperative pulmonary complications are the most frequent and significant contributor to morbidity, mortality, and costs associated with hospitalization. Interestingly, despite the prevalence of these complications in cardiac surgical patients, recognition, diagnosis, and management of this problem vary widely. In addition, little information is available on the continuum between routine postoperative pulmonary dysfunction and postoperative pulmonary complications. The course of events from pulmonary dysfunction associated with surgery to discharge from the hospital in cardiac patients is largely unexplored. In the absence of evidence-based practice guidelines for the care of cardiac surgical patients with postoperative pulmonary dysfunction, an understanding of the pathophysiological basis of the development of postoperative pulmonary complications is fundamental to enable clinicians to assess the value of current management interventions. Previous research on postoperative pulmonary dysfunction in adults undergoing cardiac surgery is reviewed, with an emphasis on the pathogenesis of this problem, implications for clinical nursing practice, and possibilities for future research. (American Journal of Critical Care. 2004;13:384-393)*

Since the advent of cardiac surgery in the 1950s, the number of cardiac procedures done worldwide has increased exponentially. Soon after cardiac surgery commenced, the contribution of postoperative pulmonary complications (PPCs) to morbidity and mortality<sup>1-3</sup> was recognized. Cardiac surgical patients are subject to distinct surgery-related factors that predispose them to the pathogenesis of PPCs. Unique to cardiac surgery are the effects of the median sternotomy incision, topical cooling for myocardial protection, internal mammary artery dissection, and the use of cardiopulmonary bypass. Pulmonary dysfunction is a ubiquitous consequence of cardiac surgery, and every clinician familiar with the postoperative care of cardiac surgery patients anticipates complications.<sup>4</sup>

Clinical manifestations of postoperative pulmonary dysfunction (PPD) range from arterial hypoxemia in 100% of patients<sup>5</sup> to acute respiratory distress syndrome, which occurs in 0.4%<sup>6</sup> to 2.0%<sup>7</sup> of patients.

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In the literature, the terms dysfunction and complication are frequently used interchangeably. We maintain that a distinction between pulmonary dysfunction and pulmonary complications is necessary. PPD refers to expected alterations in pulmonary function such as increased work of breathing, shallow respiration, ineffective cough, and hypoxemia. The diagnosis of PPC requires symptomatic pulmonary dysfunction and associated clinical findings, such as atelectasis, that meet the specified criteria of a particular diagnosis.

*Postoperative pulmonary dysfunction results in increased work of breathing, shallow respirations, ineffective cough, and hypoxemia.*

For most patients then, some degree of PPD is an inevitable consequence of cardiac surgery. However, PPD is poorly defined and not well recognized.<sup>8</sup> This ambiguity has several implications. First, information on the course of events of PPD in adults during the postoperative inpatient phase is sparse, and second, the

point at which PPD becomes a pulmonary complication is not clear and is often difficult to establish.

Although much research on PPCs after cardiac surgery is available, investigators have studied risk factors, predictors, management interventions, and subsequent outcomes of the complication rather than the progression toward these complications. This approach does not recognize the inevitability of PPD after cardiac surgery or the sequence of dysfunction patients may encounter before a PPC is diagnosed. In other words, previous research provides few clues about the expected course of events associated with pulmonary dysfunction in postoperative cardiac surgical patients.

In this review, we provide an integrated discussion of the pathogenesis of PPD unique to cardiac surgery to support the proposition that pulmonary dysfunction is an inevitable consequence of cardiac surgery. The search for studies in this review included a series of successive steps as recommended by the *Cochrane Reviewers' Handbook*, version 4.1.5.<sup>9</sup> We searched the following databases via SilverPlatter and WebSPIRS: CINAHL, MEDLINE, PubMed, Current Contents, PsycINFO, Cambridge Scientific Abstracts, Dissertation Abstracts, and HealthSTAR. The search was restricted to the adult population, the English language, and the years 1980 through 2002 inclusive. In addition, we searched tables of contents of relevant journals and reference lists in various articles by hand and via the Internet. Our strategy included exploding the terms pulmonary or respiratory, complication or dysfunction, and cardiac or cardiovascular surgery.

Overall, the pattern of pulmonary dysfunction in patients before any PPC is diagnosed appears to be a neglected but fundamental issue in the development of these complications. With an aging population and the prevalence of delaying surgical treatment, patients referred for cardiac surgery today tend to be older and sicker, have increasingly more complex problems,<sup>10</sup> and therefore it would seem are at much greater risk for PPCs than were previous patients. Contemporary clinical practice involves dynamic and at times rapid advances in the technological and surgical techniques involved in cardiac surgical procedures; however, associations between improved techniques and a reduction in the extent of PPCs have not been identified.<sup>11</sup> In addition, examining PPCs rather than the course of PPD does little to foster interventions that have a preventive rather than a curative focus.

Because no evidence-based practice guidelines for the care of cardiac surgical patients with PPD are available, an understanding of the pathophysiological basis of the development and continuum of PPD is crucial for several reasons. Appreciating the course of

events associated with PPD might result in earlier recognition of patients at risk and facilitate preemptive clinical practice. The capacity to recognize variability in PPD will promote appropriate monitoring of this problem from the immediate postoperative phase until resolution and recovery or the diagnosis of a PPC.

This approach provides an opportunity to ensure outcomes-based continuous quality improvement in clinical practice. With an increased understanding of routine PPD, the development of useful quality indicators of efficient and effective practice can be expected. Finally, an approach that provides knowledge and recognition of pulmonary dysfunction after cardiac surgery will provide the impetus for a research agenda that will offer a better understanding of the continuum between PPD and PPCs.

### **Pathogenesis of PPD**

The pathogenesis of PPD is associated with anomalies in gas exchange, alterations in lung mechanics, or both. Anomalies in gas exchange are evidenced by a widening of the alveolar-arterial oxygen gradient, increased microvascular permeability in the lung,<sup>12</sup> increased pulmonary vascular resistance,<sup>4</sup> increased pulmonary shunt fraction,<sup>5</sup> and intrapulmonary aggregation of leukocytes and platelets.<sup>13</sup> Alterations in the mechanical properties of the lung lead to reductions in vital capacity,<sup>14</sup> functional residual capacity,<sup>15</sup> and static and dynamic lung compliance.<sup>16</sup> A review of the etiology of PPD in the context of cardiac surgery is useful in understanding the relationship between the pathogenesis of abnormalities in gas exchange and pulmonary mechanics and the subsequent pathophysiological manifestations associated with PPD.

### **General Anesthesia**

Factors associated with the development of PPCs and cardiac surgery are summarized in Table 1. The immediate contribution of anesthesia to abnormalities in gas exchange is well documented. Anesthesia combined with prolonged supine positioning results in an upward shift of the diaphragm, relaxation of the chest wall, altered chest wall compliance, and a shift in blood volume to the abdomen from the thorax.<sup>62,81-83</sup> These factors in combination result in ventilation-perfusion mismatch<sup>63,64</sup> and abnormal pulmonary shunt fraction. The ventilation-perfusion mismatch is evidenced by a widening alveolar-arterial oxygen gradient<sup>36</sup> and reductions in the vital capacity and functional residual capacity of the lungs.<sup>62</sup> In addition, inhalation anesthetics inhibit hypoxic pulmonary vasoconstriction, and narcotics used for induction of anesthesia reduce hypoxic and hypercapnic ventilatory drive, fur-

**Table 1** Factors associated with the development of postoperative pulmonary complications and cardiac surgery

<b>Preoperative</b>
Chronic obstructive pulmonary disease <sup>17-21</sup>
Obesity <sup>17,22-24</sup>
Age: >60 years, <sup>25,26</sup> >70 years, <sup>19,20,23</sup> >80 years <sup>17,22,27,28</sup>
Diabetes <sup>29</sup>
History of smoking <sup>18,29,30</sup>
Chronic heart failure <sup>17,20,22,29,31-33</sup>
Emergency surgery <sup>22,23,25,34</sup>
Previous cardiac surgery <sup>20,25</sup>
Immobility <sup>35</sup>
<b>Intraoperative</b>
Respiratory depression <sup>36</sup>
Neurological injury <sup>37</sup>
Lung deflation <sup>38</sup>
Cardiopulmonary bypass <sup>36,39</sup>
Topical cooling <sup>40,41</sup>
Internal mammary artery dissection <sup>15,36,42-47</sup>
Sternotomy incision <sup>48,49</sup>
Increased number of bypass grafts <sup>44,50,51</sup>
Increased duration of cardiopulmonary bypass <sup>22,23,31,34,44,50,52</sup>
Lower core temperature <sup>22,34,50,53</sup>
<b>Postoperative</b>
Respiratory depression associated with nonreversal of anesthesia <sup>36</sup>
Phrenic nerve dysfunction <sup>54</sup>
Diaphragmatic dysfunction <sup>55,56</sup>
Pain <sup>57-60</sup>
Constant tidal volumes/short shallow respiration <sup>48</sup>
Reduced compliance <sup>61</sup>
Reduced vital capacity and functional residual capacity <sup>62</sup>
Ventilation-perfusion mismatch and physiological shunt <sup>36,63,64</sup>
Fluid imbalance <sup>27,31,39,65</sup>
Immobility, <sup>66,67</sup> position <sup>68</sup>
Chest tubes <sup>69</sup>
Nasogastric tubes <sup>70</sup>
Impaired mucociliary clearance, <sup>71</sup> ineffective cough <sup>14,72</sup>
Pleural effusion <sup>47,73,74</sup>
Atelectasis <sup>72,75-77</sup>
Pulmonary edema <sup>4,7,78,79</sup>
Aspiration <sup>80</sup>

ther predisposing patients to widening of the alveolar-arterial oxygen gradient and the onset of hypoxemia and atelectasis.<sup>36</sup> Increasingly, the benefits of combined intrathecal anesthetics and reduced doses of general anesthetics for a reduction in postoperative pulmonary morbidity have been investigated, with inconsistent findings.<sup>84-87</sup>

*Inhalation anesthetics inhibit hypoxic pulmonary vasoconstriction, which increases hypoxemia.*

## Surgical Approach

The effects of the median sternotomy incision, hypothermia for myocardial protection, dissection of the internal mammary artery, and the use of cardiopulmonary bypass are intraoperative factors unique to cardiac surgical procedures. The impact of the median sternotomy incision on PPD is not yet clear. Sternotomy with rib retraction logically leads to reduced airway pressures and increased lung compliance, because the chest wall no longer impedes lung expansion. Closure of the chest wall, however, produces changes in the opposite direction that are particularly enhanced in patients with chronic obstructive airway disease or obesity.<sup>61</sup>

Researchers<sup>36</sup> who compared the sternotomy incision with a thoracotomy incision think that minimal interruption to the chest wall, less trauma, and negligible lung compression make the sternotomy a relatively benign procedure. Barnas et al<sup>78</sup> support this stance. In a study of 11 patients undergoing median sternotomy, they found that the incision did not affect the mechanical properties of the chest wall.<sup>78</sup> Ranieri et al<sup>88</sup> reported that sternotomy produced immediate changes in chest wall mechanics that were completely resolved 4 hours after sternotomy in 8 adults who had valvular correction. In contrast, Auler et al<sup>89</sup> found marked alteration in respiratory mechanics in 12 patients after median sternotomy, and Tulla et al<sup>48</sup> found that surgical trauma associated with sternotomy led to shallow breathing, impaired gas exchange, and a predisposition to PPCs. Previous research<sup>63</sup> in patients who had abdominal surgery clearly indicated the relationship between the proximity of the surgical incision to the thorax and the development of PPCs.

The effectiveness of less invasive surgery and use of a partial inferior midline sternotomy rather than the standard full midline approach in reducing the occurrence of PPCs is equivocal. Bauer et al<sup>90</sup> reported that a smaller sternotomy incision had no beneficial effect on PPCs. However, Lichtenberg et al<sup>49</sup> found an association between the preservation of pulmonary function and minimally invasive direct coronary artery bypass that required an 8-cm incision rather than the standard 20-cm incision. Of note, however, minimally invasive direct coronary artery bypass procedures are simpler than the standard full midline approach, involve grafting of fewer and more accessible vessels, and therefore in most cases are associated with a shorter duration of surgery.<sup>91</sup>

## Internal Mammary Artery Dissection

Because of the complexity of cardiac surgical procedures, the influence of the initial incision on PPD cannot be considered in isolation. In particular, attention has been given to the influence intact pleura may

have on pulmonary mechanics after cardiac surgery. The internal mammary artery has been accepted as the conduit of choice for bypass grafting because of its superior patency rate. The results of some recent studies,<sup>15,36,42-47</sup> however, suggest that retrieval of the internal mammary artery, which typically necessitates pleural dissection, may contribute to increases in PPCs. What is not clear is whether the increased occurrence of PPCs is due to the pleurotomy itself, subsequent pleural effusion, or pericardial inflammation contributing to the development of pleural effusion.<sup>92</sup> Remarkably, compared with unilateral internal mammary artery grafting,<sup>93,94</sup> bilateral internal mammary artery grafting does not increase the occurrence of PPCs but may increase the need for acute respiratory support.<sup>95</sup>

### **Topical Cooling for Myocardial Protection**

Specific intraoperative strategies to ensure myocardial protection include moderate systemic cooling of circulating blood via the cardiopulmonary bypass circuit and profound myocardial hypothermia. Although the necessity of and optimal approach to achieving profound myocardial hypothermia are the subject of technical debate,<sup>35</sup> few publications confirm the impact of hypothermia on PPD. Myocardial hypothermia is achieved by using topical iced slush,<sup>96</sup> a cooling jacket, or infusion of the coronary arteries with chilled cardioplegic solution.<sup>38</sup> Cooling jackets and direct infusion of chilled solutions are advantageous because their containment aids in avoiding hypothermic injury to the phrenic nerve. The sequelae of phrenic nerve dysfunction include diaphragmatic paralysis and alterations in pulmonary mechanics that can have a marked impact on the course of PPD<sup>36,55</sup>; however, phrenic nerve dysfunction does not always prolong patients' length of stay in the hospital<sup>97</sup> and it occurs relatively infrequently.<sup>56</sup>

Phrenic nerve paralysis was attributed to the use of topical cooling in a retrospective comparative study<sup>54</sup> of 100 patients, 50 of whom received topical slush and 50 who did not. The frequency of phrenic nerve paralysis was greater than 30% in the slush group and less than 5% in the other group. In addition, more than 80% of the patients who received slush and 32% of patients who did not had subsequent collapse of the left lower lobe. In a sophisticated electrophysiological study by Dimopoulou et al,<sup>40</sup> logistic regression analysis of intraoperative risk factors for postoperative phrenic nerve dysfunction indicated the use of ice slush as the only independently related risk factor. In contrast, in a prospective study by Markand et al,<sup>56</sup> 43 of 44 patients had atelectasis but only 5 had diaphragmatic dysfunction as a result of phrenic nerve paralysis.

The suspicion that factors other than topical cooling are responsible for PPCs is further supported by Wilcox et al,<sup>50</sup> who found unequivocal phrenic nerve paralysis in only 10% of their study patients who had a 93% incidence of atelectasis of the left lower lobe. A discriminant analysis of intraoperative variables indicated that more severe atelectasis was associated with a larger number of bypass grafts, longer operative and bypass time, pleurotomy, absence of a right atrial drain and cardiac insulating pad, and lower systemic temperature.<sup>50</sup>

*Phrenic nerve paralysis has been associated with the use of myocardial topical cooling.*

### **Cardiopulmonary Bypass**

After the induction of anesthesia, creation of the operative incision, and retrieval of the internal mammary artery pedicle, cardiopulmonary bypass is achieved before direct myocardial hypothermia is established. Use of cardiopulmonary bypass has clear consequences for postoperative pulmonary function. Compared with other types of major surgery, it appears to cause additional lung injury and a delay in pulmonary recovery. General surgical patients with PPD usually have hypoxia but no alteration in the alveolar-arterial oxygen gradient; this situation implies that hypoventilation rather than abnormalities of ventilation and perfusion is the causative factor.<sup>5</sup> The PPD attributed to cardiopulmonary bypass is both common and severe and has been the subject of several recent reviews.<sup>4,11,89-100</sup> The dysfunction is thought to be due to the effects of an acute systemic and pulmonary inflammatory response<sup>13,98,101</sup> commonly referred to as "pump lung"<sup>102</sup> or "post pump syndrome."<sup>36</sup>

*Pulmonary dysfunction may also result from lung injury caused by cardiopulmonary bypass.*

Once cardiopulmonary bypass commences, the cessation of pulmonary ventilation results in collapsed lungs; insufficient alveolar distention to activate the production of surfactant, a situation that potentiates alveolar collapse; abnormal pulmonary mechanics; retention of secretions; and atelectasis. Pulmonary circulation is stopped; blood is exposed to hypothermic conditions,

cardioplegic solution, foreign mechanical surfaces, and shearing forces.<sup>39</sup> Sequestration of blood in the microcirculation, pulmonary ischemia, injury to capillary walls, release of inflammatory mediators,<sup>100</sup> increased pulmonary capillary permeability,<sup>103</sup> flooding of the pulmonary interstitium,<sup>104</sup> increased intrapulmonary shunt,<sup>105</sup> and the formation of microthrombi occur, all of which increase abnormalities in gas exchange and lead to closure of the small airways. In an attempt to minimize microcirculatory disturbances and to augment tissue perfusion and oxygen delivery, induced hemodilutional anemia is a routine facet of cardiopulmonary bypass.<sup>106</sup> Notably, recently researchers linked low hematocrit levels to requirement for reintubation,<sup>52</sup> respiratory failure,<sup>107</sup> and increased lengths of stay<sup>106,108</sup> and massive blood transfusion to acute respiratory distress syndrome.<sup>6</sup>

Continued refinement of cardiopulmonary bypass materials and anesthetic and operative techniques has largely limited lung injury.<sup>94</sup> Although the degree of lung injury may be reduced by factors such as shorter duration of cardiopulmonary bypass, the effect on the occurrence of PPCs may not be immediately apparent. Currently, the frequency of PPCs remains similar for patients who have cardiopulmonary bypass and patients who do not.<sup>109-111</sup> The resurgence in popularity of off-pump surgery for cardiac patients does, however, offer greater potential for reductions in the release of inflammatory mediators and subsequent PPD.<sup>98,110,112-114</sup>

### Summary

In summary, the pathogenesis of PPD after cardiac surgery is multifactorial and complex. In individual patients, the variables that influence the course of events associated with PPD may occur alone or in combination. Continued moves toward improving technology and technique offer the potential to improve patients' outcomes. Unfortunately, what the literature does not address in detail is the expected occurrence of problems with gas exchange and lung mechanics in the postoperative period and how these problems may affect patients' recovery.

The onset and course of events of the pathophysiological manifestations of PPD could be determined by carefully mapping the clinical manifestations of PPD, something that has not been done. This mapping would involve investigating aspects of clinical decision making in the context of PPD after cardiac surgery, including the subjective indicators nurses use as manifestations of PPD.

### Pathophysiological Manifestations of PPD

Clearly, some degree of pulmonary dysfunction after cardiac surgery can be expected. The occurrence of PPD is evidenced by pathophysiological manifesta-

**Table 2** Frequency of pulmonary complications after cardiac surgery

Complication	Frequency, %
Pleural effusion	27-95 <sup>118,119</sup>
Atelectasis	16.6-88 <sup>47,50</sup>
Phrenic nerve paralysis	30-75 <sup>54</sup>
Prolonged mechanical ventilation	6-58 <sup>26,71</sup>
Diaphragmatic dysfunction	2-54 <sup>55,120</sup>
Pneumonia	4.2-20 <sup>30,121</sup>
Diaphragmatic paralysis	9 <sup>50</sup>
Pulmonary embolism	0.04-3.2 <sup>122,123</sup>
Acute respiratory distress syndrome	0.4-2 <sup>6,7</sup>
Aspiration	1.9 <sup>124</sup>
Pneumothorax	1.4 <sup>125</sup>
Chylothorax	18 individual case reports <sup>126</sup>
Trapped lung syndrome	Single case report <sup>127</sup>

tions. Alterations in gas exchange and lung mechanics are indicated clinically by short shallow respiration without periodic sighs,<sup>48</sup> changes on chest radiographs,<sup>115</sup> hypoxemia,<sup>22,116</sup> increased respiratory rate, increased work of breathing, additional breath sounds, and a productive cough.<sup>14,71,117</sup> Signs and symptoms that accompany pulmonary dysfunction are the result of alterations in gas exchange and pulmonary mechanics discussed previously. In clinical practice, manifestations of PPD do not usually have a marked effect on a patient's postoperative course. The question of when PPD becomes clinically important is difficult to answer. However, the finding that a patient cannot adequately and independently ambulate because of symptomatic shortness of breath and hypoxemia associated with postoperative pathophysiological changes in pulmonary function is clinically important.

Until due attention is given to identifying the onset and describing the progression of the pathophysiological manifestations of PPD, appropriate interventions to manage PPD will remain ambiguous. Clinical decision making in the treatment of patients with PPD after cardiac surgery is poorly documented. Subjective indicators that nurses recognize as manifestations of PPD specifically after cardiac surgery have not been described. The expectation that objective symptomatic indicators are identified as a component of patients' assessments needs validation. Finally, mapping the course of PPD will enable the determination of clinical interventions that can hasten the resolution of PPD and the potential identification of key indicators that differentiate PPD from diagnosed PPCs.

### Diagnosis and Frequency of PPCs

Common pulmonary complications after cardiac surgery are outlined in Table 2. Accuracy and speci-

ficity in diagnosing pulmonary complications after cardiac surgery can be complex. Distinct diagnostic groups are difficult to define clearly because of the variability in diagnostic criteria.<sup>128</sup> This difficulty is particularly evident when evaluating the literature on atelectasis and pneumonia. Although each diagnosis has a specific definition, the definition of each diagnosis is variable in the literature. In addition, the distinction between what constitutes a clinically important pulmonary finding and a pulmonary complication is not always clear.<sup>129</sup> Hypoxemia, for example, has significant clinical implications, yet hypoxemia in itself is not a diagnosis but a component of other diagnoses such as atelectasis or pneumonia.

Understandably then, the frequency of pulmonary complications and their severity in clinical practice are not clearly documented.<sup>5</sup> Variations in the reported occurrence of pulmonary complications after cardiac surgery range from 8% to 79%<sup>71,130</sup> and can be attributed to a combination of factors. In addition to the diverse definitive criteria that affect diagnostic specificity, a variety of terms are used to classify PPCs. Previous research on the incidence and prevalence of PPC after cardiac surgery in adults was often limited by inadequate sample sizes and equivocal definitions and outcome measures. Risk prediction models often focused on heterogeneous groups of patients with multiple or specific singular outcomes. These risk models were rarely validated in uniform surgical groups, and this presents difficulties for clinicians who are attempting to translate research findings into practice. Further, definitions of pulmonary complications that contain multiple outcomes narrow the spectrum of clinical relevance, because the risk indices developed are not complication specific.<sup>131</sup> Finally, the benefits of advancements in anesthesia and surgical technique since the advent of cardiac surgery have been offset by the increased acuity of the population of patients who have this type of surgery.<sup>119</sup> Thus, comparisons with earlier investigations of PPCs to evaluate the contemporary occurrence and outcomes of PPCs are difficult.

### **Effectiveness of Pulmonary Interventions**

Routine facets of postoperative cardiac care to improve patients' pulmonary function have been described. Most of these interventions focus specifically on airway management and include various techniques of mechanical ventilation, endotracheal suctioning, extubation and physiotherapy that includes deep breathing and coughing exercises, incentive spirometry, and the application of a range of maneuvers to achieve positive airway pressures and alveolar recruitment. More recently,

the effects of postoperative position, pain management, and early ambulation on PPCs have been evaluated.

In previous research, investigators focused on the efficacy of interventions to reduce the occurrence of PPCs rather than improvements in pulmonary function as a result of the intervention. Interventions that reduce the occurrence of PPCs will also improve pulmonary function; however, a focus on reducing complications that includes the use of outcome measures that solely reflect incidence means that the multifactorial context in which complications arise may be overlooked. Previous investigations, with complication-related end point measures, were not successful in determining the effectiveness of pulmonary interventions for the attenuation of PPD and subsequent promotion of patients' recovery. Consequently, it is difficult to determine the effectiveness of pulmonary interventions for postoperative cardiac surgical patients in whom early pulmonary dysfunction spontaneously resolves. Further, treatments are often administered infrequently, evaluated in isolation in poorly controlled environments, and administered to patients who have clinically insignificant dysfunction.<sup>132</sup>

The large amount of literature devoted to chest physiotherapy is contradictory and requires clarification. The impact of physiotherapy on patients' pulmonary function after cardiac surgery was the subject of several investigations, with equivocal results. When the efficacies of preoperative prophylactic inspiratory muscle training,<sup>133</sup> breathing techniques,<sup>117,128,134-136</sup> incentive spirometry,<sup>137,138</sup> the application of positive inspiratory and expiratory pressures via mask,<sup>66,139</sup> and early mobilization<sup>140,141</sup> for the prevention of PPC are compared, no single method is superior.

In addition, when use of a particular technique does result in a measurable difference in pulmonary function, the difference may be sustained for as little as 1 hour, as in the case of biphasic inspiratory positive airways pressure,<sup>142</sup> or be immediately lost when the therapy is discontinued, as in the case of positive-end expiratory pressure.<sup>143</sup> Notably, however, the premise underpinning treatments associated with physiotherapy is intermittent administration. The possibility that more frequent treatments<sup>132</sup> applied for a longer time<sup>144</sup> would be beneficial in the management of PPD has received little attention, most probably because of the financial burden this intervention would involve.

The interaction between postoperative pulmonary function and pain management is poorly understood. For many years, narcotic analgesics have been used with caution because of early research that indicated an association between administration of analgesics and an increased incidence of respiratory dysfunction.<sup>145</sup> Some investigators<sup>36,38</sup> claimed that pain control is rarely a

problem after cardiac surgery because the surgery requires minimal muscular interruption and the sternum is well supported when wired at the end of the procedure. However, research by Watt-Watson et al<sup>146</sup> highlighted the fact that although patients reported moderate to severe pain, they received only 47% of the prescribed analgesics. More than 50% of patients reported severe pain before their next dose of analgesic, and 80% of patients received only 16 mEq rather than the recommended 50 to 60 mEq of morphine for each 24-hour interval in the first 3 postoperative days.

Greater pain intensity is linked to an increased frequency of atelectasis.<sup>57</sup> Poorly controlled pain postoperatively is manifested as an ineffective breathing pattern, impedes patients' mobility, and prolongs recovery. Recent research<sup>147</sup> established the benefits of refined administration of analgesics to enhance cardiac surgical patients' ability to be weaned from mechanical ventilation with minimal pain or respiratory side effects. Other investigators focused on the site of pain after surgery,<sup>58,59</sup> therapies to reduce pain,<sup>60,84,148-154</sup> and the influence of attachments such as temporary pacing wires<sup>155</sup> and chest tubes<sup>156-159</sup> on pain after cardiac surgery.

Finally, although the link between treatment interventions and patients' outcome is well established in terms of airway maintenance, the effect of additional physiological injuries such as neurological deficits,<sup>37</sup> fluid imbalance,<sup>65,102</sup> immobility,<sup>67,137</sup> impaired mucociliary clearance,<sup>71</sup> ineffective cough, and subsequent sputum production and retention<sup>14,72</sup> on the course of events associated with PPD requires further investigation. The compounding pathophysiological effect of each of these factors on the course of events in PPD and the development of PPCs is not well understood.

*No single method of pulmonary physiotherapy is superior to others in preventing pulmonary complications.*

### Clinical Implications and Conclusion

The focus of this review is the pathogenesis and pathophysiological manifestations of PPD after cardiac surgery in adults. The emphasis is on the importance of recognizing the continuum between PPD after cardiac surgery and the diagnosis of PPCs. The course of events associated with PPD after cardiac surgical procedures has not been investigated. Currently, clinicians rely on the evidence that supports interventions for the prevention of PPCs to make management decisions. This reliance has several implications for clinical practice and future research.

Indicators nurses use to identify PPD and PPCs and to instigate treatment interventions, the actual interventions, and the effectiveness of nursing interventions must be explored. Because PPCs have been used as an end point to assess the effectiveness of clinical interventions, the value of interventions to actually prevent the progression of PPD and the development of PPCs is not known. The effect of interventions on the course of events in PPD requires immediate scrutiny. The context in which clinical interventions take place and its effect on processes of postoperative care and thus PPD is largely ignored. Further, in many instances, nursing interventions are ritualistic and founded on anecdotal substantiation, as reflected in the inaccessibility of sound evidence-based information.

Risk prediction models that identify low, medium, and high risk for the development of PPCs will indicate those patients in whom preemptive interventions may have the greatest effectiveness. Models with well-defined single outcomes must be developed and tested in homogeneous subsets of surgical patients to enhance the clinical applicability of research findings.

PPD is an inevitable consequence of cardiac surgery that should be recognized and expected. The continuum between PPD and the development of PPCs needs to be managed with reliable preventive treatment strategies. Within the spectrum of postoperative care, nurses can make a significant difference to patients' outcomes. Mapping the continuum of PPD and determining the effectiveness of preventive nursing interventions are possibly the most essential but most underinvestigated aspects of postoperative pulmonary management. Once the course of events associated with PPD is understood, prospects for future research and opportunities to clarify the role of nursing interventions in the resolution of PPD will be ample.

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