

NEWSLETTER

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February 2007

President's Message

Remembering John Arthur Hinckley, 1932 - 2006

By James G. Ramsay, MD
President, 2005-2007

Those of you who attended our annual meeting last year in San Diego will recall the SCA created a lifetime outstanding service award in the name of John Hinckley, and he was the first recipient. John was the founder of Ruggles Service Corporation 32 years ago, and was instrumental in the early development and continued success of our society. It is with great sadness I announce to you his peaceful passing away after a long struggle with lung disease, on December 18, 2006. John is survived by his daughter Heather and son Stewart, who continue to operate Ruggles. There are three other children and eight grandchildren who survive John.

When we created John's award last year, I spoke with past presidents Earl Wynands and Jerry Reves as well as the founders of the society who are lifetime Board members, George Burgess and Bob Marino, to find more about John's history. My brief remarks in giving the award really did not do justice to his life and contributions, and my intention is to let you all know what a wonderful man he was and how he helped us create the very successful organization we have today. I have drawn from these individuals as well as from eulogies given by Heather's husband, Bruce Spiess, and John's son Stewart.

John was a native of Fauquier County, Virginia. He was a direct descendant of Presidents James Monroe, Theodore Roosevelt and Franklin D. Roosevelt. A graduate of St. Paul's School (NH) and the University of Virginia, he completed studies at the Stonier Graduate School of Banking. Commissioned in the U.S. Army, he served in Wurzburg, Germany and Fort Riley, Kansas with the First Infantry Division. After his work in the service John worked for State Planter's Bank in Richmond, Virginia, until he tired of the corporate world and decided to start a family business in 1974. He and his wife Elizabeth founded the "Institute for Continuing Education," but he soon changed the name to Ruggles Service Corporation because the former was too hard to say when he answered the phone (he lived in a home on Ruggles Road). His first clients included the Virginia Society of Anesthesiologists, the American Academy of Cerebral Palsy and Developmental Medicine, the American Society of Post Anesthesia Nurses, and the American Society of Regional Anesthesia. A few

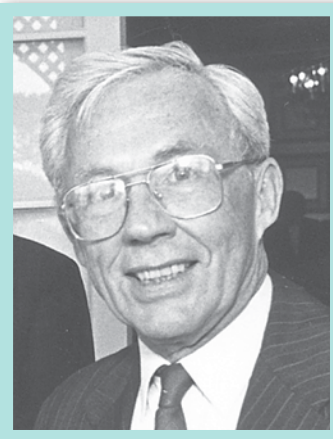
years later George Burgess was searching for a meeting planner for the SCA, and Ruggles was recommended to him through the search process. An excerpt from a letter George wrote in 2001 describes his early interactions with John:

"...Then came the first of what would be many contract negotiations. The SCA had little in the way of reserves and wanted no risk; John was understanding... to a point. Each year or two, the contest started a few months before contract expiration. Of course, John was also our compiler of finances and knew exactly what SCA could and could not afford. He invariably started out with his desired proposal for contract renewal; which invariably was just as courteously rejected, followed by a counterproposal. This ritualistic negotiation always led to a final capitulation stage on both sides, accompanied by statements that neither John nor SCA could possibly afford the contract terms. Yet, both parties always seemed to do just fine under the final terms... year after year. These were very enjoyable negotiations; and, he always got the contract. I wish that my managed care negotiations were as enjoyable."

Successive SCA presidents all relate stories of their interactions with John, and these reflect John's great humanity, his genuine interest in the well being and success of people he worked with and for, his integrity and honesty, his focus on family, and a wonderful sense of humor. From his son Stewart:

"A number of years ago, while running a meeting in Montreal, Dad asked Kevin Johns, our Director of Meetings, to put "foot prints" down on the sidewalks between the hotels that we were using. I guess Dad thought this would make our convention delegates feel closer to one another. Well... Kevin was in the middle of marking up the sidewalks when the Montreal police arrested him. Kevin called Dad from jail, and when Kevin handed the phone to the policeman, Dad told them in fluent French that he had no idea who Kevin was."

Over the years Ruggles took on many more societies, yet John and his family's commitment to SCA remained stronger than ever, and with their help we grew from one annual meeting to three meetings per year, as well as an international meeting every other year. When John retired in 2002 he stayed connected with



John A. Hinckley

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the society but had already effectively "handed the reins" to Heather and Stewart who had been helping him run the society's business for many years. He continued to come into the office and contribute thoughts and ideas about the SCA virtually until the end. This smooth transition, Heather's knowledge of our history and key players, and her considerable organizational and people skills have greatly facilitated our growth and development in recent years. Heather and Stewart now have 20 employees and manage 13 societies, with Heather focused almost exclusively on our very successful SCA.

Our relationship with John Hinckley was clearly more than a business contract. His humanity fostered relationships with successive leaders of the SCA that continued long after their terms of office. We all grew to respect not only his business and organizational skills, but also his ability to help us make decisions that were in our own best interest, his unfailing integrity and honesty in dealing with us and helping us deal with each other, and in our contractual relationships with the hotel and travel industry as well as our own medicine related industries. He will be remembered as a great human being, and is missed by all those who had the good fortune to know him.



Nadir hematocrits lower than 21% on cardiopulmonary bypass are associated with greater cerebrovascular and renal complication rates and should trigger transfusion.

PRO:

The management of nadir hematocrits during cardiopulmonary bypass (CPB) has changed dramatically over the years. Initially, all CPB circuits were primed with whole blood, reducing the level of hemodilution and limiting how low the patient's hematocrit would drop.¹ Concerns arose over exposing patients to blood transfusions from numerous donors (triggering antibodies, increasing risks of transfusion related reactions, and infection) and depleting the relative scarce commodity of available blood.



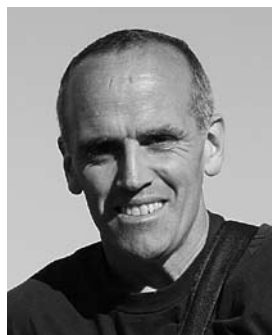
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Currently, while variable, most CPB prime is composed of crystalloid and colloid solutions that can lead to marked levels of hemodilution. There are advantages of hemodilution: a reduction in the viscosity of blood allows for maintenance of blood flow without elevated arterial pressures (which can lead to dissection). Hypothermia also aids in reducing oxygen requirements of vital organs during CPB, allowing for the lower hematocrit, but the question remains: to what nadir hematocrit should we hemodilute?^{2,3}

Several recent studies have found that the severity of hemodilution during CPB correlates strongly with increased perioperative morbidity and/or mortality.^{3,4,5,6} These clinical studies have built upon the animal models in the literature that suggested severe hemodilution could lead to increased end-organ damage and touted potential benefits of maintaining higher hematocrits.^{7,8} Additionally, the definition of "severe" hemodilution in these studies varies. Increased complications are seen with nadir hematocrits anywhere in the range of 17-23.^{2,3,4,5,6} For the purposes of this discussion, I will consider nadir hematocrits less than 21 severe, and highlight the evidence in the literature that exists for maintaining patients at or above this number.

In 1997, Fang et al. found that so-called "high-risk" patients (defined as those who had one or more of 8 significant independent predictors of mortality including: previous open heart surgery, renal failure, congestive heart failure, etc) had increased mortality with nadir hematocrits less than 17, while "low-risk" patients had increased mortality at < 14. This was one of the first studies in a human population that actually identified a number where morbidity/mortality was increased at a certain nadir hematocrit. Previous work had all shown either no benefit or safety at hematocrits even as low as 13!⁴ It should be noted, however, that these studies, including even Fang's work, were done in a surgical population that is very

CON:



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An 82 year old underwent replacement of the ascending aorta and aortic arch with reimplantation of the head and neck vessels. Cardiopulmonary bypass (CPB) lasted 3.5 hours including 60 minutes of deep hypothermic circulatory arrest. The lowest HCT during CPB was 16%. During the re-warming phase he received four units of packed red blood cells. The preoperative serum creatinine was 1.1 and the peak postoperative creatinine was 1.2 mg/dl. The patient was discharged from the hospital on postoperative day 7 neurologically intact and without evidence of any end-organ injury.



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Hemodilution occurs at the onset of CPB due to mixing of the patient's blood with the CPB prime. Depending on the patient's red blood cell mass, blood volume, and the volume of the CPB prime, resultant hematocrits (HCT) commonly range from 16-28%. Although oxygen content is reduced, blood flow is increased due to favorable rheologic changes helping to maintain systemic oxygen delivery and oxygen balance, as globally assessed by the mixed venous oxygen saturation, acid-base balance, and lactate levels. Recently, several retrospective, un-

controlled, and/or data-base driven studies suggest that this hemodilution is not safe and that clinicians should consider transfusing packed red blood cells (PRBCs) for a HCT < 21%. These investigations report an association between the nadir HCT (nHCT), which is defined as the lowest HCT during the CPB period, and perioperative end-organ dysfunction (specifically renal and neurocognitive) (1-6). The nHCT is thought to reflect the severity of hemodilution during CPB.

The reported incidence of renal dysfunction varies with its definition but may be as high as 10-20% when defined by a > 20% increase in serum creatinine (SCr) or creatinine clearance (CrCl). However, significant renal failure (>50% change from baseline) occurs in less than 5% of patients, and renal failure requiring dialysis occurs in < 1% of cases (1-13). Stroke as defined by a new gross deficit occurs in less than 2%, delirium in < 10%, and neurocognitive dysfunction

different than the one we deal with today- almost all of our patients would fall into the “high-risk” category.

Additionally, subsequent, more recent work in larger groups of patients has demonstrated that increased complications occur well above the first nadir established by Fang. DeFoe et al. reported a trend towards increasing risk of death at all hematocrits < 23 in a much larger group of patients. The mortality at a nadir hematocrit of < 19 was double that of when the nadir hematocrit was 25 or above. Additionally, patients with low hematocrits had an increased risk of intra- or post-operative placement of intra-aortic balloon pumps and return to CPB after initial separation.² Smaller patients had increased risk and inherently this makes sense as their hemodilution would presumably be more severe if identical CPB circuits were employed regardless of weight.

Habib et al. found that at nadir hematocrits lower than 22, patients had significantly increased risk for myocardial infarction, renal failure, prolonged ventilation, sepsis and longer intensive care unit (ICU)/hospital stays. Long term survival also was improved systematically with increasing nadir hematocrits. Both early and late mortality figures were worse for patients with nadir hematocrits < 20, and worsened significantly the lower the hematocrit fell versus patients with hematocrits in the high 20's.⁶

In addition to mortality outcomes, evidence for significant morbidity associated with hematocrits lower than 21 is prevalent throughout the literature and the pathways for such injury better delineated. Of note, the renal and neurological organ systems seem particularly susceptible to injury after excessive hemodilution.

The effect of hemodilution on brain function is thought to be related to the increase in the embolic load to the brain.⁹ Additionally, while oxygen delivery to the brain is maintained during hemodilution in normal brain cells, ischemic cells may lack the compensatory measures needed for survival and extraction of oxygen decreases with decreased hematocrit.³

Karkouti et al examined almost 11,000 patients prospectively undergoing cardiac surgery with CPB. For every percent decrease in nadir hematocrits during CPB, patients had a 10% increase in the odds of suffering a perioperative stroke. If patients had a nadir hematocrit < 18, their risk of stroke was 3x that of patients who had nadir hematocrits 21-23.³ In another retrospective review of 5000 consecutive patients, Habib et al. found a significantly increased risk of stroke with nadir hematocrits of less than 22.⁵

Some authors maintain that hematocrits should actually be maintained much higher than 21, even as high as 30 for preservation of neurological function.¹⁰ Jonas et al., found that in a pediatric population, patients with hematocrits higher than 27 had significantly better motor skills at one year than their counterparts who had nadir hematocrits of 21.^{5,11} Noting that the adult brain may be even more sensitive to hypoxic brain injury than the neonatal brain, he advocates hematocrits of at least 25.¹⁰

Like neurological injury, renal failure as sequelae of cardiac surgery is a dreaded complication. It can occur in 1-5% of patients undergoing cardiac surgery and has been identified as an independent risk for increased mortality.^{12,13} With respect to renal function, even slight reductions in renal oxygen delivery (as a result of hemodilution) can cause ischemic damage. This may actually be exacerbated by increased renal blood flow, as this increases renal energy requirements which cannot be met in the face of diminishing

in as many as 40-60% of cases depending on the battery of neurological tests employed (3,4,14-16). Although significant renal or neurocognitive dysfunction is uncommon, they are associated with increased perioperative (30 day) mortality, prompting clinicians to find preventative methods.

Investigators touting the importance of the nHCT describe an nHCT < 21% as a strong predictor, or risk factor for renal and/or neurologic injury (1-6). Some have reported that the threshold for injury may be as high as 24 or 25% (3,4). Habib et al further reported that a nHCT > 27.5% was associated with an improved 6 year outcome (1). Although there is some recognition that a well thought-out prospective randomized study is important, the undertone of these articles suggests that CPB management should be altered to prevent such hemodilution, perhaps involving blood transfusion.

So, how can it be that the patient presented above did not suffer any significant end organ injury despite a nHCT of 16%? There is clearly a lot more to end-organ injury than the nHCT (17). A number of logistic issues with the design of these manuscripts are evident. Because these investigations are retrospective, uncontrolled, and/or data-base driven, the authors employ complicated statistical analyses in order ‘to control for’ or ‘filter out’ numerous other variables also found to be associated with adverse outcome (Table 1) (1-6). Despite a rigorous application of complicated statistics, the authors do not report any predictive positive or negative values. Due to the low incidence of significant and serious renal and neurologic outcomes, and the overlap of nHCT among complicated and uncomplicated cases, the predictive positive value of the nHCT would be low, while the predictive negative value would be well below 100%. This alone suggests that acceptance of a HCT of 21% as the transfusion trigger would prompt unnecessary blood transfusion to a very larger majority of patients. Furthermore, when patients are grouped into ranges of nHCT, patients with lower nHCT are markedly different, and carry a higher risk profile than those with a higher nHCT (1,4). Patients with lower nHCT are characterized by a host of demographic, surgical and perioperative variables (TABLE 1), which are also associated with, predictive of, or a risk factor for adverse outcome. Readers should also be aware that there is no ‘real control’ during these retrospective analyses when considering decisions that are made day-to-day, by different clinicians on a case-by-case basis. They also do not control for selection of surgical patients, the type and timing of surgical procedures, decisions regarding the placement of intra-aortic balloon pumps (IABP), choice of hemodynamic monitors, selection of vasoactive medications, transfusion of blood products, evaluation, and manipulation of the aorta, management of CPB (hypothermic, normothermic, mean blood pressure, acid-base management, systemic flows), as well as a host of postoperative clinical care protocols. All these variables and more are associated with patient outcome (TABLE 1). While the authors of these manuscripts make every effort to convince the reader that the nHCT is an important risk factor or is predictive of renal or neurologic outcome, they, in the end, are only able to describe an association between nHCT and outcome.....nothing more than an association!!!!

Proposed (not proven) mechanisms for the association of nHCT and outcome include increased embolic events and reduced oxygen delivery (DO₂) to organ beds. Blood flow increases linearly with hemodilution, which helps compensate for reduced oxygen content to

oxygen delivery.¹³ Additionally, much like in the brain, increased embolic load is also thought to contribute to renal dysfunction perioperatively.

Recent studies have confirmed the deleterious effects of low nadir hematocrits on renal function. Karkouti et al. studied over 9000 patients prospectively and found an independent relationship between nadir hematocrit on CPB and acute renal failure requiring dialysis. Hematocrits lower than 21 had the highest risk; interestingly, hematocrits above 25 also had greater risk compared to those between 21-25.¹³ Habib et al. found that patients with CPB hemodilution to hematocrits < 24 had an increased likelihood of renal injury and worse operative outcomes.⁵ In another study, the same authors found that as nadir hematocrits on CPB decreased lower than 22, the risk of postoperative renal dysfunction increased.⁶

It is clear from these studies that the current practice of hemodilution, while with some benefits, carries inherent risks of increased

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help maintain oxygen delivery (18,19). Previous literature describes the benefit of hemodilution in preventing organ injury by increasing blood flow, reducing red cell trapping, and providing more uniform delivery of oxygen throughout the tissue beds (20-23). It is now theorized that this hemodilution, which results in increased blood flow, results in greater systemic delivery of emboli during high-risk periods as during aortic manipulation (1-5). The association between micro-emboli and hemodilution was reduced when the CPB prime volume was decreased from 1500 ml to 500 ml, the latter being applied to a Miniature Extracorporeal Circulation (MECC) (24). Although patients in the MECC group had higher mean HCT during MECC (34.5% vs. 29.3%) and a reduction in microemboli, there were no differences in end-organ outcome (24). Although systemic emboli have long been thought of as a reason for end organ injury, methods to reduce or catch emboli (Aortic Embol-X Catheter) have not been shown to reduce neurologic outcome or other systemic end-organ injury (25-27). Finally, despite a reduction in cerebral embolization during off-pump CABG surgery, no clear and consistent difference in neurologic function or cerebral MRI were noted in a prospective randomized study comparing off-pump to on-pump CABG surgery (28). To complicate the matter further, embolic delivery is also dependent on ventilator management and PaCO₂, neither of which is reported in the nHCT studies. Hypocapnia reduces emboli delivery (29). These studies highlight the complex nature of systemic injuries, and that microembolization may not play a significant role in significant and/or sustained injury.

Another proposed mechanism of injury is a critical reduction in oxygen delivery (DO₂) to systemic organs. According to the nHCT investigations, this occurs when the HCT is reduced below 21%. It is reasonable to accept that there exists a critical value of hemoglobin, below which oxygen demands are not matched by delivery. Although transient decreases in DO₂ occur, sustained injury is more likely to result from a prolonged period of critically reduced DO₂, and not based on a one-time measured, uni-dimensional nHCT. Furthermore, hemoglobin is only one component of DO₂ and oxygen balance. Others include metabolic demand (VO₂), blood flow and hemodynamics, oxygen saturation, and vascular tissue caliber. These investigations include a variety of CPB techniques including systemic normo-, hypo-, and deep hypo-thermia, the latter with circulatory arrest, and CPB flows ranging from 2 to 2.5 L/min/m² all of which impact on DO₂ and oxygen balance (1-6).

morbidity and mortality. While most of these studies have been observational or retrospective in their design, their results are consistent. There exists a need for randomized, controlled trials that may better elucidate how low we should allow hematocrits to fall during CPB (as has been called for by many of these authors). Certainly there are concerns with transfusions, but there exist possible alternatives where excessive hemodilution as well as transfusion can be minimized. These include the practice of retrograde, autologous priming of the CPB circuit or the use of smaller circuits, especially in smaller patients. When these measures cannot be undertaken, hematocrits below 21 should spur transfusions as the neurological/renal morbidity and overall increases in mortality outweigh the benefits of hemodilution.

References available at www.scahq.org

One investigation demonstrated the greater importance of DO₂ in predicting renal outcome. The authors described a critical nadir DO₂ (nDO₂) of 272 ml/min (calculated) below which an association with renal dysfunction was found (6). This resulted in a sensitivity and specificity of only 68%. In the same study an nHCT of 26% resulted in 64% sensitivity and 65% specificity (6). Although their argument lends greater support to a reduced oxygen delivery as a contributing component of renal injury, this measure is also of ill-defined duration and severity. Furthermore, significant overlap existed between those patients with and without renal dysfunction. Similar to other investigations, a number of additional commonly described variables were significantly associated with outcome (older age, higher preoperative creatinine, COPD, DM, longer CPB, reduced LVEF, and blood transfusion). When blood transfusion was included in the analysis, the nHCT was only borderline significantly ($p = 0.06$) associated with outcome in some of the statistical models used, and not significant in others (6). Although the concept of nDO₂ is attractive, a prolonged reduction in DO₂ is more likely to cause injury. This could occur prior to CPB, during CPB, especially when prolonged, or after CPB when oxygen delivery is dependent on baseline and resultant cardiovascular and pulmonary functions. In these investigations, prolonged CPB, reduced baseline heart function, pulmonary disease, low cardiac output, and a greater need for vasoactive agents and/or IABP, are all significantly associated with adverse outcome (1-6).

Reduced baseline (pre CPB) renal and neurologic function and reserve are important in predicting the ability to tolerate transient periods of insult. Patients with preoperative renal and neurologic dysfunction are more likely to have increased postoperative organ dysfunction (1-6, 8,11,13-15,30,31). Several investigations have demonstrated the strong and independent correlation between preoperative testing, including MRI, and the incidence of both stroke and neurocognitive dysfunction (32,33). These preoperative assessments were found to be the most predictive variable. In one investigation comparing off- (no associated hemodilution) to on-pump CABG surgeries, no differences in neurocognitive outcome were found both immediately after surgery and for 12 months thereafter (24.1% vs. 23.1%) (33). Not only was the preoperative MRI a

predictor of neurocognitive dysfunction, but patients with a normal preoperative MRI had no new cognitive dysfunctions 3 months after surgery (33). A recent investigation demonstrated increases of the neurologic protein, S β -100, after induction of anesthesia and prior to CPB suggesting that a cause of neurocognitive dysfunction may result from a number of variables not yet fully recognized and not related to CPB and hemodilution (34). This helps explain similar incidences of neurocognitive dysfunction recorded for patients undergoing off-pump cardiac surgical or non-cardiac surgical procedures during which the severity of hemodilution associated with CPB does not occur (34-40).

Patients characterized by a number of high risk demographic and procedural variables (TABLE 1) are also more likely to have lower nHCT, receive blood transfusions, have perioperative renal and neurocognitive dysfunction, and suffer significant morbidity and mortality (1-8, 11, 13-16, 30-32, 41). It seems more accurate to state that the nHCT reflects the sicker and more complicated patient, than to speculate any cause and effect relationship. Although Habib et al reports an association between nHCT and perioperative adverse outcomes (reoperation, septicemia, stroke, coma, prolonged ventilation, pulmonary edema, perioperative myocardial infarction, tamponade, cardiac arrest, placement of IABP, renal failure, multiorgan failure, operative mortality, increased ICU and hospital stays), data from multiple studies show that these patients are at higher risk, with greater co-existing morbidities (e.g. renal or neurologic), possibly undergoing more complicated procedures with longer CPB times (Table 1) (1-4). This is further supported by data from off-pump CABG for which a similar incidence of neurocognitive and renal dysfunction was observed as on-pump CABG, despite the absence of hemodilution associated with CPB, and a reduction in blood transfusion (38-40, 42). In all these studies, a similar demographic and perioperative description (TABLE 1) of the high-risk patient is consistently reported, and associated with adverse neurologic, renal, and overall patient outcomes. High-risk patients are more likely to have lower nHCT, receive blood transfusion, and suffer end-organ dysfunction.

Possible techniques to prevent or treat hemodilution include reducing the CPB prime volume or transfusing blood products respectively. Blood transfusion has emerged as an important variable associated with organ dysfunction and patient outcome (43). Although investigators have stated that their statistical analyses controlled for blood transfusions, it is difficult to control for something that is directly linked to nHCT (1-6, 8, 13-15). Patients with lower HCTs get transfused!! Furthermore, predictors of a lower nHCT are similar to predictors of perioperative blood transfusions (surgical procedure, reoperation, urgency of surgery, elevated serum creatinine, age, CPB time, preoperative HCT, reduced LV function, and poor nutrition (albumin <4 mg/dl)) (1, 3, 4). These are also known risk factors for renal and neurocognitive dysfunction, and patient adverse outcome (44-46). In fact, transfusion had the highest odds-ratio for end-organ dysfunction in several studies (8, 13). Although it may be statistically possible, it is clinically difficult to separate the nHCT from other patient and surgical variables, also associated with patient outcome. Transfusion has been associated with mistransfusion, transmission of infectious agents, hemolytic reactions, transfusion related lung injury, and immunosuppression with an increased risk of postoperative infections, higher incidence of multisystem organ failure, longer hospital stays, and greater use of hospital resources and cost (43, 47). One proposed mechanism for end-organ injury associated with blood transfusion includes a reduced release of oxygen from transfused hemoglobin due, in part, to lower levels of 2,3 DPG. Stored red blood cells are deficient in

2,3-DPG and are unable to unload bound oxygen, thus impairing oxygen delivery to the tissues. Tsai et al. showed that transfusion with stored red blood cells under conditions of normovolemic anemia actually decreased tissue oxygenation by 400% (48). Stored red blood cells are also less supple and flexible and may physically obstruct capillaries, leading to further organ ischemia (2, 43). Additionally, banked blood carries a large load of inflammatory cytokines, white blood cells, bradykinin, platelet activating factor, and activated complement (43), all complicating the inflammatory response. Engoren et al reported on 1,915 patients who underwent first time coronary artery bypass surgery. Six hundred and fifty-nine (34%) were transfused perioperatively. The mortality at two years after surgery was 15% for those who received transfusion compared to 6% for those who did not. After correction for co-morbidities, those patients who received transfusions had a 70% increased risk for long-term mortality (49).

Previously described 'critical hematocrits' are significantly lower than the proposed range of 21-24%. Using a rabbit model of normothermic and hypothermic CPB, the critical hematocrit (sustained cardiac dysfunction) was not reached until 10-12% at which time the SvO₂ fell below 46% and the heart rate decreased to unsafe levels defined by hemodynamic instability and difficult resuscitation (50). Using a dog model of normothermic CPB, systemic oxygen delivery and uptake were matched until the HCT fell below 18% during CPB. Mixed venous saturation, however, did not significantly change until the HCT was reduced to 9% at which time lactate levels increased (51). In another investigation, a HCT between 9 and 14% resulted in an imbalance between cerebral oxygen delivery causing a decline in cerebral metabolic rate and consumption (52, 53). Finally, in humans, myocardial oxygen balance, during normothermic CPB, was maintained until a HCT of 15% at which time myocardial oxygen extraction was not able to increase to match demand, and, subsequently, myocardial ischemia occurred as reflected by an increase in coronary sinus lactate (54). The retrospective 'nHCT' investigations state that adverse outcome was associated with a nHCT of < 21%, perhaps as high as 25% (2, 4, 6). There was, however, significant overlap between patients with and without end-organ injury. In the study by Karkouti et al, the mean nHCT for patients complicated by renal failure was 21 +/- 4% compared to 23 +/- 4% in patients without renal failure (3, 4). Differences in nHCT of patients with no renal insufficiency, renal insufficiency, and acute renal failure, in the study by Habib et al was 22.4 +/- 4.6%, 21.3 +/- 4.7%, and 20.6 +/- 4.1% respectively (2). Differences between patients with and without complicated perioperative periods were 19.9 +/- 4.0% vs. 21.8 +/- 4.1% respectively (2). In these investigations there is also a large range in which pre CPB and nHCT fall (22-52% and 10-33% resp.) (5).

The critical HCT during CPB may be as low as or lower than 15%, depending on CPB management such as permissive, or induced hypothermia. In considering the standard deviations listed above, and the low incidence of significant long-lasting organ dysfunction a blanket transfusion policy based on a trigger of 21% would needlessly transfuse a large number of patients. Although using statistical methodology a statistician may be able to justify a transfusion trigger of 21%, clinicians should consider patient demographics and co-morbidities, baseline end-organ functions, perioperative cardiopulmonary function and procedural variables prior to making such decisions. The data on nHCT should be viewed as an association with outcome, which, in the absence of causality, reflects a higher risk patient, who may be undergoing a more complicated surgical procedure. Comments otherwise are speculative.

References available at www.scahq.org



Literature Reviews

Is early too early? Effect of shorter stays after bypass surgery

Cowper PA, DeLong ER, Hannan EL, Muhlbaier LH, Lytle BL, Jones RH, Holman WL, Pokorny JJ, Stafford JA, Mark DB, Peterson ED. *Ann Thorac Surg* 83:100-107, 2007.

Reviewer:

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University of Chicago

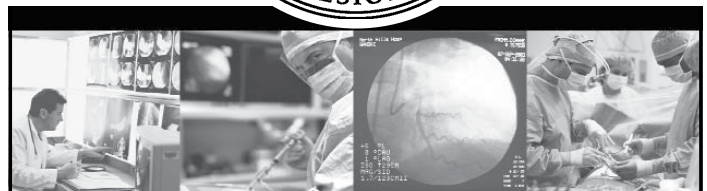
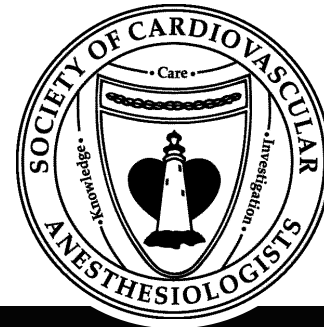
Abstract Excerpt

Postoperative stays after coronary artery bypass graft surgery (CABG) decreased substantially in the 1990s. Although shorter stays offer clinical benefits, premature discharge could increase adverse events and offset initial savings. This study examined the effect of early discharge after CABG on readmission/death and cost within 60 days of discharge home. Variability in hospitals' tendencies for early discharge and adverse outcomes was also explored. Analyses were based on clinical and claims data for 55,889 New York CABG patients discharged home 1995 to 1998. Early discharge was defined as a postoperative stay below the 15th percentile for patients with similar risk. The likelihood of early discharge and its effect on readmission/death were examined using hierarchical logistic regression, accounting for patient risk and within-hospital correlation. The correlation between early discharge and adverse outcomes at the hospital level was assessed. The effect of early discharge on subsequent inpatient, outpatient, skilled nursing, and home health costs was examined in the Medicare subset. Overall, 17% of patients were discharged early, with increasing prevalence over time. The tendency to discharge early varied widely among hospitals (2% to 42% of patients). No association between hospitals' tendencies for early discharge and adverse outcomes was found. Lower post-discharge costs among patients discharged early (mean of \$3,491 versus \$5,246 for typical stays) resulted in average cumulative savings of \$6,309. These investigators conclude that patients selected for earlier discharge after CABG did not have increased adverse event rates or higher costs. Variation among hospitals in early discharge suggests that more efficient patient management could be achieved at some hospitals.

Reviewer's Comments

In the 1990s, postoperative stays following CABG decreased substantially with the adoption of aggressive care protocols that facilitated earlier extubation and mobilization of patients. The potential clinical advantages of shorter postoperative stays include improved respiratory outcomes, reduced nosocomial infection rates, and the opportunity to recover at home. However, premature hospital discharge of inappropriately targeted patients could increase postoperative complications, subsequent readmissions, and mortality. Furthermore, potential savings associated with shorter stays may be transitory if readmissions increase or care shifts to outpatient or nonacute institutional settings. This topic is difficult to study because healthier patients are more likely to have shorter hospital stays and fewer complications after discharge. Also, what

constitutes early discharge for a high risk patient may be a typical length of stay for a healthy patient. In order to account for this, these investigators assessed the effect of early discharge defined relative to individual patient risk. They found no evidence that patients discharged early were more likely to die or be readmitted within sixty days of discharge. Also, while substantial variation was found among hospitals in the prevalence of early discharge, a hospital's tendency to discharge early had no effect on its likelihood of adverse events. The strengths of this study include the large sample size, comprehensive ascertainment of post-discharge adverse events, and the use of clinical baseline data. Limitations of this study include the observational design, somewhat imperfect risk adjustment, unreliable cost data, and the fact that the study is somewhat dated (ending in 1998). Furthermore, the Invited Commentary associated with this article postulates that the "hospital savings demonstrated" is actually "cost shifting" (and workload shifting) from hospitals to individualized clinical practices. Despite such reservations, this study indicates that patients appropriately selected for earlier discharge following CABG surgery appear not have increased rates of adverse clinical events or higher health care costs and more efficient patient management could be achieved at some hospitals without comprising quality of care.



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IMPORTANT NOTICE: 2008 ANNUAL MEETING DATE CHANGE

The Board of Directors of the SCA wishes to extend an apology to all SCA members regarding a need to change the date of the 2008 Annual Meeting. The meeting was inadvertently scheduled to overlap with Passover, and this was recognized only a few weeks ago. The Board has approved a new date for the 2008 Annual meeting: June 18 – 22. The meeting will be in Vancouver Canada, as originally planned.

Society of Cardiovascular Anesthesiologists

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