Letters to the Editor

Surgical nurse assistants in cardiac surgery: a UK trainee’s perspective

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In the United Kingdom, the main role of the cardiac surgical nurse assistant (SNA) is to harvest the vascular conduit, a role traditionally held by the cardiothoracic Senior House Officer (SHO). This overlap in roles may deprive the cardiothoracic SHO of the chance to harvest an intact long saphenous vein (LSV) which provides an important training exercise in careful tissue handling, haemostasis and wound closure, skills all essential for a surgeon in training. Alex et al. [1] have shown that the use of SNAs does not adversely affect outcomes. This is interesting but of greater importance is their impact on surgical training. The authors have not addressed this.

We performed a telephone questionnaire survey of all cardiothoracic SHOs in accredited training posts in England and Wales between 20 and 30 July 2002 (unpublished data). The SHOs were asked to provide data on the number of operative procedures performed over the previous 6 months as recorded in the Royal College of Surgeons logbook. One hundred and ten SHOs were included in the survey and divided into two groups. The SNA group \( n = 58 \) comprised SHOs who worked with SNAs, while the non-SNA group \( n = 52 \) did not.

The two groups were similar with regards to cardiac surgery experience and possession of the diploma of membership of the Royal College of Surgeons (MRCS) or equivalent post-graduate qualification. The median number (interquartile range) of LSV dissected free in the non-SNA group was 30 (14.3–71.3) compared to 15 (3.6–35) in the SNA group \( P = 0.001 \). In the non-SNA group, the median number (interquartile range) of cases in which the SHO initiated cardiopulmonary bypass was 1 (1–2) compared to 10 (2–25) for those in the SNA group \( P = 0.004 \). 37/58 (64%) of SHOs who worked with SNA found their presence beneficial and in some cases it allowed them to perform other more advanced aspects of the operation such as median sternotomy, initiation of cardiopulmonary bypass and sternal closure.

The presence of the SNA seems to alter the nature of surgical training for SHOs. The impact of this on the quality of surgical training needs to be investigated further.

Reference


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Reply to the Letter to the Editor

Reply to Shrivastava et al.
A little of something is good for nothing

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An interesting observation from Mr V. Shrivastava and his team, but I wish to highlight certain aspects of their study from the limited data presented.

First, their data only confirms our argument. It is satisfying to show statistically significant difference between groups, but the sad fact is that the median number of LSV harvest by SHOs in the SNA group was only 15 in a 6-month period (0.58 per week) and of even more significance, is the fact that some of them had harvested less than 1 per month. The median number of ‘cannulations’ in the same group during this period was only 10 (0.38 per week). I wonder how many of these post-MRCS SHOs in this group were technically sound in either procedures. Though 64% in this group found the presence of SNAs ‘beneficial’ one wonders if the remaining 36% were the ones who lost out on basic surgical skills, failed to gain any advanced skills and ended up being delegated to the wards.

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Secondly, any telephone questionnaire is prone to responder bias when the questions relate to the respondents' skill. Unlike validated data from the PATS database or operation notes, a telephone interview questioning one's surgical skills is prone to exaggeration and personnel interpretations.

In an overtly inquisitive and critical environment where 'surgeon-specific results', 'league tables' and 'performance indicators' are the first things that cross a patient's mind I am sure Mr Shrivastava and his team would agree that there is no substitute to 'hands-on' practice.

I read with great interest the article by Wehlin and colleagues [1] in the January issue of EJCTS. They carried out a prospective, randomised study to investigate the influence of extracorporeal circulation on the biological functions of inflammatory cells and concluded that there was less complement activation in low risk OFFCB patients compared with ONCB patients. A corollary of their study was that as there was obvious inflammatory response in both groups and a lack of significant differences between groups, therefore, factors other than CPB are responsible for cellular and cytokine activation. Furthermore, they suggest that the 'dark side' of CPB may be overestimated and the surgical trauma could be the more important contributing factor to the enhanced inflammatory response during cardiac surgery. I humbly tend to disagree with the authors in this regard.

Cardiac surgery undoubtedly provokes a vigorous inflammatory response, which has important clinical implications. Although factors influencing incidence, severity, and clinical outcome of the inflammatory response are currently not well understood yet available evidence suggests that CPB specifically activates the inflammatory response via at least three distinct mechanisms. One mechanism involves direct 'contact activation' of the immune system following exposure of blood to the foreign surfaces of the CPB circuit [2]. A second mechanism involves ischemia-reperfusion injury to the brain, heart, lungs, kidney and liver as a result of aortic cross-clamping. Restoration of perfusion on release of the aortic cross-clamp is associated with activation of key indices of the inflammatory response [2]. Finally, splanchnic hyperperfusion, a common finding during and following CPB, may damage the mucosal barrier, allowing gut translocation of endotoxin. The ensuing endotoxaemia may indirectly activate the inflammatory cascade [3].

Furthermore, the composition of the priming solution, cardioplegia, presence of pulsatile or non-pulsatile perfusion, type of oxygenator and pump, type of extracorporeal circuit, temperature during CPB and its duration are all important factors influencing the inflammatory response [4]. An additional factor is the development of excessive shear stress during CPB as a result of large pressure changes across the CPB circuit, causing damage to blood constituents and activating the inflammatory response [5]. Shear stress decreases erythrocyte deformability and increases hemolysis. Leukocyte adhesiveness is increased, and mechanical disruption, with neutrophil degranulation and release of cytotoxic products, may be seen at high levels of shear stress. Excess shear stress also increases platelet activation and may contribute to endothelial injury [5].

In short, we must admit that the etiology of inflammatory response after cardiac surgery is probably a composite of unstable peribypass hemodynamics, global myocardial ischemia, suboptimal organ perfusion during CPB, and immune events related to exposure to extracorporeal circulation per se and despite all efforts to improve the design of CPB to make it friendlier the 'dark side' of CPB will always be there.

References


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