What's New in MCS

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Articles:

Journal of Cardiac Surgery March, 2014
*1.Hysi I, Fabre O, Renaut C, Guesnier L. *Extracorporeal Membrane Oxygenation with Direct Axillary Artery Perfusion*. J Card Surg 268-269
http://www.ncbi.nlm.nih.gov/pubmed/24131078

European Journal of Cardio-Thoracic Surgery March, 2014
*1.Floerchinger B, Philipp A, Foltan M, Keyser A, et al. *Neuron-specific enolase serum levels predict severe neuronal injury after extracorporeal life support in resuscitation*. European J of Cardio-Thor Surgery 496-501
http://www.ncbi.nlm.nih.gov/pubmed/23878016

The Journal of Heart and Lung Transplantation March, 2014

*1.Cantillon D, Salida W, Wazni O, Kanj M, et al. *Low cardiac output associated with ventricular tachyarrhythmias in continuous-flow LVAD recipients with a concomitant ICD (LoCo VT Study)*. JHLT 318-320
http://www.ncbi.nlm.nih.gov/pubmed/24559946

2.Kamdar F, Eckman P, John R. *Safety of discontinuation of anti-coagulation in patients with continuous-flow left ventricular assist devices*. JHLT 316-318
http://www.ncbi.nlm.nih.gov/pubmed/24462558

Circulation March, 2014
1.Burke M, Givertz M. *Assessment and Management of Heart Failure after Left Ventricular Assist Device Implantation*. Circulation 1161-1166
http://www.ncbi.nlm.nih.gov/pubmed/24615964

Annals of Thoracic Surgery March, 2014

1.Thomas S, Smallwood J, Smith C, Griffin L et al. *Discharge Outcomes in Patients with Paracorporeal Biventricular Assist Devices*. Annals of Thoracic Surgery 894-900
http://www.ncbi.nlm.nih.gov/pubmed/24280185

2.Bruce C, Bhimaraj A, Smith M. *Revisting Surrogate Consent for Ventricular Assist Device Placement*. 747-749
http://www.ncbi.nlm.nih.gov/pubmed/24580897

Reviews:

Extracorporeal Membrane Oxygenation with Direct Axillary Artery Perfusion. J Card Surg 268-269

This is a descriptive single-center retrospective review of sixteen patients who underwent direct axillary artery cannulation for ECMO support rather than the interposition of a Dacron graft for arterial access. Results were reviewed from January 2009-April 2013. Cannulation was on the right-side in fourteen of the sixteen patients with a mean cannula size of 17.5 +/- 1.5 French. No patient was undergoing cardiopulmonary resuscitation (CPR) at the time of ECMO placement.

There was no documented failure of arterial cannulation. 30 day and 90 day mortality rates were 56.2% and 68.7%, highlighting the severity of the underlying illness in this cohort. No complications affecting the ipsilateral upper extremity limbs were noted including no injuries to the brachial plexus. Two patients required reoperation for subcutaneous bleeding but no bleeding was found at the level of the axillary artery cannulation site.

The authors conclude in their retrospective analysis that direct axillary artery cannulation for ECMO support has a low rate of hemorrhagic and ischemic complications. The ability to place a large diameter cannula also theoretically allows a higher mean outflow of blood. Unfortunately, this procedure's use is limited to stable patients who can go to the operating room.

Neuron-specific enolase serum levels predict severe neuronal injury after extracorporeal life support in resuscitation. European J of Cardio-Thor Surgery 496-501

Biomarkers are used to assess prognosis after cardiac arrest. Since neurological outcomes of ECMO supported patients can be especially poor, this study examined a routinely monitored biomarker, neuron-specific enolase. This retrospective study included a cohort of 31 adult patients who had VA ECMO initiated during CPR. Serum neuron–specific enolase peaks were monitored and correlated with neurological recovery and in-hospital mortality. A comparator group of fourteen patients on ECMO support who did not receive CPR was included in the study as a reference for neuron-specific enolase levels during time spent on ECMO support. All controls had CT imaging to exclude elevated neuron-specific enolase levels as a device-related effect. Patients were then divided into two groups with mild-moderate and with high neuron-specific enolase levels with a cut off value of 100ug/l. Neuron-specific enolase levels were measured within 12-24 hours of ECMO cannulation and were repeatedly monitored within a 24-hour time frame of initiation of circulatory support.

Patient age was comparable in the study and control groups (55 +/- 17 years vs 52 +/- 13 years, p=NS). Time of resuscitation prior to ECMO support was 48 +/- 32 minutes. Duration of CPR was comparable in patients with mild-moderate and high neuron-specific enolase levels (46 +/- 34 vs 54 +/- 26 min, p=NS). In seven of the patients, neuron-specific enolase levels were greater than 100 ug/l (mean 218 +/- 155 ug/l). The remaining twenty four patients had a neuron-specific enolsae level <100 ug/l (mean 50 +/- 23 ug/l).

Neurological outcome appeared inferior in the high neuron-specific enolase group compared with the mild-moderate neuron-specific ensolase group. The sensitivity, specificity, and positive predictive value of peak neuron-specific enolase levels for severe neurological events (defined as multiple lesions or cerebral edema causing herniation) in this study were calculated as 100%, 85%, and 83% respectively. Peak neuron-specific enolase levels for predicting in-hospital mortality was only 64% sensitive and 7% specific, with a positive predictive value of only 45%.

Although this study was retrospective and not powered to recommend changing the current practice of maintaining or removing extracorporeal circulatory support, this study has shown that neuron-specific enolase, a noninvasive and easily available blood test, is able to detect early neuronal damage which does seem to correlate with worse neurologic outcomes. This study has several limitations, chief

among them is that this is a very small study and it is retrospective in nature. In addition, the cut-off value of 100ug/l for neuron-specific enolase levels was based on previous evidence on patients treated with therapeutic hypothermia after CPR and not patients undergoing mechanical circulatory support of some kind.

Low cardiac output associated with ventricular tachyarrhythmias in continuous-flow LVAD recipients with a concomitant ICD (LoCo VT Study). JHLT 318-320

This is a prospective study of ten patients on HeartMate II CF-LVAD support at a single-center whose purpose was to determine the effect of ventricular fibrillation (VF) on cardiac output. Patients undergoing defibrillation threshold (DFT) testing by inducing VF had their LVAD flow measured from the power base unit console while at baseline as well as during and after induced VF. Patients were included in this study if they were undergoing de novo ICD implant or generator exchange. Each patient served as their own control. Eight patients were de-novo ICD implants and two underwent pulse generator exchange.

There were twenty-one defibrillation tests performed among ten patients. The patients' mean age was 54.2 years with an average ejection fraction of 14%. The mean baseline speed was 8,861+/-369 RPMs, flow was 4.9 +/- 1.4 LPM, and PI was 4.9 +/- 0.6. The mean cycle length for induced VF was 230 milliseconds with time in VF 5.5 +/- 2.2 seconds. During time in VF, the flow decreased in all ten study patients by a mean of 31.8% to a nadir of 3.39 +/- 1.1 LPM. The mean time to return to baseline LVAD output after successful defibrillation was 18 +/- 4 seconds. All patients recovered to their baseline flow after successful defibrillation within 30 seconds.

This study concludes that VF in CF-LVAD patients causes a significant decrease in flow of 31.8% that returns to baseline by ICD therapy within 30 seconds in all patients. Although this is a small study, it is a useful physiologic study. It implies that in all LVAD supported patients, a fibrillating right ventricle supplies less preload across the pulmonary circulation to the LVAD. It would be useful to know what the pulmonary vascular resistance (PVR) of the study patients was. For example, if the PVR was low, would the fibrillating RV be better able to serve as a passive conduit and LVAD flow be more preserved? If the PVR was higher, would the LVAD flow in VF be lower? This study is an important one as there is debate in the literature about the clinical importance of having an active ICD to rapidly defibrillate a LVAD supported patient who is in a ventricular tachyarrhythmia.