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Myocardial Ischemia and Central-mixed Venous Oxygen Saturation Gradient.

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Myocardial Ischemia and Central-mixed Venous Oxygen Saturation Gradient.

Author(s): Faydhi A A, Salim S , Nuzhat S

Abstract

Objective

Detection of Myocardial ischemia in post coronary artery bypass grafting patients.

Design

Prospectively simultaneous central venous Oxygen (ScvO2) & mixed venous oxygen (SVO2) saturation on hourly interval will be analysed from patients coming to ICU after coronary artery bypass surgery.

Setting

Cardiac surgery Intensive Care unit, King Abdul Aziz University Hospital Jeddah. Saudi Arabia.

Patients

All post CABG patients with a Pulmonary artery catheter (PAC) were included in study.

Main results

30 patients were enrolled in the study; simultaneous ScvO2 & SVO2 on hourly basis for the first 6 hours were done in post CABG patients via a PAC. 6/ 30 patients had a peak troponin > 40 ng/ml (1) while 24/30 had a troponin surge < 40 ng/ml. Patient with high Troponin surge (> 40 ng/ml) were generally older, higher incidence of on going smoking, diabetes, stroke and Peripheral vascular disease had a significant wider and a positive ScvO2-SVO2 gradient as compared to patient with lower troponin surge(8.18±4.12 vs. 3.75±6.12 p=0.00673). Other parameters i.e. bypass time, cross clamp time, time to extubation, ICU stay, Cardiac index, PAOP no significant difference was found.

Conclusion

Persistently positive and wide ScvO2 - SVO2 Gradient can be used as direct evidence of myocardial ischemia in post CABG patients.

Introduction

Many studies, both recent and remote, failed to find a fixed relationship between mixed and central venous oxygen saturation. In others words ScvO2 cannot be substituted for SVO2. The venous return in jugular veins, inferior vena cava, superior vena cava and coronary sinus are the main contributors to the blood flowing in main pulmonary artery. A change in oxygen saturation in any one of these, without a change in others, may change the net SVO2.

In a proper clinical setting, a reduction in SVO2 as compared to ScvO2 can be taken as a direct evidence of ischemia in a particular vascular bed. Coronary artery bypass surgery is carried out to relieve coronary ischemia. The surgery is not without risk and complications. Myocardial ischemia can occur in post coronary artery bypass surgery patient, the causes are numerous. Post operatively, ischemic chest pain can not be differentiated from surgical incision pain. Clinical examination is also not helpful. High troponin is marker of infarction rather than ischemia.

In absence of other pathologies, a markedly deoxygenated blood from coronary sinus may reduce SVO2 saturation when compared to ScvO2. We set forth to investigate whether a wide difference between ScvO2 and SVO2 can be used as a marker of myocardial ischemia in patients who have under gone a coronary artery bypass grafting surgery.

Methods

After approval by the hospital ethical committee & consent from the patients, all post CABG patients admitted to our unit between June 2010 till Febuary 2011 were included in study.

Patients without PAC were excluded. Moreover, incomplete or unpaired data were excluded from the study. Unpaired data means samples with time interval between sampling of central and mixed venous > 5 minutes.

Patients were transferred to ICU after CABG, intubated and sedated. All patients studied had on-pump CABG procedure, PAC (Arrow ™) was inserted as a pre-operatively. In addition to basic lab work a baseline echocardiography and Troponin were also done preoperatively.

Once in the ICU, basic lab works were sent including Complete blood count, electrolytes, Chest X ray (CXR), EKG, arterial blood gas analysis (ABG) and CPK, CKMB & Troponins. Serum Troponin was done on a daily basis. The CXR is viewed at the spot (digital Imaging) to confirm correct position of PAC(to keep the tip of PAC in main pulmonary artery) and to view the position of endotracheal tube, chest tubes and

assess lung regarding atelectasis & pneumothorax. 2 venous blood gas (VBG) sample were withdrawn and analysed (Cobas B21). Meticulous care was exercised to withdraw blood samples at slowly as rapid withdrawal of blood creates a vacuum inside the syringe may falsely reduce the oxygen in the sample. Initial 1 ml of blood was kept in a separate syringe so as to remove the fluid and blood from within the lumen of PAC, this blood was later returned, than the sample was withdrawn for analysis. First VBG sample was taken from the proximal port of PAC i.e. right atrium representing ScvO2 & the second sample from the distal tip of PAC main Pulmonary artery representing SVO2. The proximal port is approximately 30 cm proximal to the tip. The tip was positioned in main pulmonary artery, which was confirmed radiologically. If there is no coil in the catheter, then 30 cm proximal will position the proximal port somewhere in the vicinity of junction of Superior vena cava and Right atrium. This position is ideal for sampling for ScvO2 and is well proximal to the influx of coronary sinus flow. The treating physicians were unaware of the sampling. The samples were withdrawn by the in charge nurse of the patient. Analysis was simultaneously performed on the same blood gas analyser machine situated within the unit. The VBG samples were withdrawn hourly for six hours. Patient usually arrive hypothermic, patient is warmed externally. Patient is monitored for bleeding, inotrope (if started) are tapered of keeping the MAP > 65 (may be higher if the patient is hypertensive). If drains, intercostal & mediastinal, were minimal for > 24 hours, these drain were removed, clamping the drains prior to removal is no more practiced. If the patient remains hemodynamically stable (preferably without or low dose of inotropes) and regains full awareness patient was weaned for mechanical ventilator. A brief spontaneous breathing trial (SBT), of 15 to 30 minutes, was initiated and if patient passed the trial, patient was extubated. The time to extubation was recorded. Upon resumption of regular walking & free of any symptoms, other than mild pain at the surgical site, patient was transferred out of ICU and time to transferring out was recorded. Echocardiography was performed after a month as immediate echocardiography in presence of inotropes & vasopressor may not detect a new segmental dysfunction. The echocardiographist was unaware of the on going study.

Results

30 post CABG were included in study, 1 of the patients data were recorded wrongly & was not complete so was excluded from the analysis.

Post CABG a rise in Troponin is observed which peak on the second day. The patients who develop an acute myocardial infarction the peak is much higher. The cut-off value for these patient is not clearly defined. In a recent meta-analysis (1) patients with Troponin level higher than 40 ng/ml had a more complicated course as compared to patient with40 ng/ml and the other < 40 ng/ml.

36 and 150 paired samples were analysed from 6/30(high Troponin surge) and 24/30 (low Troponin surge) patients respectively. The ScvO2-SVO2 Gradient was wider in patients with high surge troponin then with low surge troponins (8.18±4.12 vs. 3.75±4.52 p=0.00673). Serum lactate measured at the same time showed no difference in the central or mixed venous blood (0.551±1.42 vs 0.79 ±1.71 p=0.258).

The observed spike on the Figure 1 are the result of borderline value i.e. trop surge 30-40, which by definition are in low surge group. Perhaps if the cut-off value should be reduced to 30 ng/ml. For this reason a larger trial perhaps will clarify the situation. In low surge group the gradient at times goes in the negative territory i.e. SVO2 which is more than ScvO2 so the ScvO2-SVO2 is a negative, this situation never occurred in high surge group.

There was some difference in other parameters as seen on the table 1, but because of small number a definite conclusion cannot be drawn. Other variables which were looked at included duration of symptom prior to surgery, time to extubation, cross clamp time, bypass time and ICU stay none of these showed statistical difference between groups.

Discussion

It's inappropriate to substitute ScvO2 for SVO2, as confirmed in many studies (4). There are 3 main contributors to venous return i.e. SVC, IVC & CS. Changes in any one of these can change the final SVO2. A change in SVO2 will only occur if a) any of IVC, SVC or CS is markedly deoxygenated(5) or b) if the degree deoxygenation is mild but the blood flow is large as in case of IVC. If patient has a grand mal convulsion the oxygen is consumed during the muscular contraction, causing a significant reduction in venous saturation in IVC- thus reducing SVO2. On the other hand ischemia of a smaller gland e.g. adrenal will not really change the SVO2. Myocardial ischemia will only change SVO2 if the ischemia is marked. Normal Coronary blood flow is 250 ml/min which is only 5% of the total cardiac output, but myocardial oxygen extraction is 70-80%- this markedly

deoxygenated blood and is diluted in IVC & SVC venous return, the net change is little under normal circumstances. If with myocardial ischemia further deoxygenation occur a change is expected in SVO2.
Can not be explaned by the method of cardioplegia
During post CABG period diagnosis of acute myocardial infarction (AMI) is difficult. Patients are under the effect of analgesia (narcotic being the predominant); patient is unable to discriminate between the pain due to sternotomy and myocardial ischemia, at times both may be contributing. Clinical deterioration i.e. signs of left heart failure may develop late (depending upon the cardiac reserve). Ventricular tachyarrhythmia do develop early in infarct but during immediate post CABG other processes may also induce arrhythmia.
ECG change due to pericardial reaction makes the ECG diagnosis of AMI inaccurate. Echocardiography also become less sensitive immediate postop in detecting segmental wall motion abnormality- due to iontropes & vasopressor on going, which may attenuate the wall motion dysynchrony.
PAC is inserted to measure the pressure & cardiac output changes. The magnitude of these changes will depend upon the size of infarct & the pre-existing cardiac reserve. Inotropes & pressor may increase the Cardiac Output in spite of an on-going infarct & may attenuate segmental wall motion abnormality so echocardiogram may not be reliable. The pressure changes detected by the PAC occurred late after the onset of ischemia- only if the ischemia is profound & prolonged. Add to these other factors e.g. the patient temperature, air bubbles etc. which can be a cause of inaccurate reading.
Many studies are now demonstrating an increase in mortality associated with PAC. This excess mortality is more than the procedural mortality; actually it's the misinterpretation of the data which results in an inappropriate clinical decision.
Monitoring the ScvO2-SVO2 with conventional PAC gives an indirect evidence of myocardial ischemia, after excluding other causes of ischemia in lower body. A high risk value (high & persistently + ScvO2-SVO2 Gradient) should alert us for more definitive investigation. Another use we found was to diagnose cause of hypotension, i.e. if the hypotension is accompanied by wide Gradient and low ScvO2 then probably a systemic cause has resulted in myocardial ischemia, while if the Scvo2 remained well maintained but the SVO2 is low mean a primary myocardial dysfunction. But a persistent difference is more important than a single reading.
Other situations where a PAC may be inserted is septic shock patients. For the reasons said above relying solely on ScvO2 or SVO2 does not give a clear picture of tissue oxygenation. So both ScvO2 and SVO2 should be analysed. Myocardial ischemia developing during the course of treatment of septic shock may goes unnoticed till myocardial reserve is overwhelmed and a clinical event occurs, often manifested by a sudden cardiovascular collapse or a cardiac arrest-by this time it may be too late. Myocardial ischemia is likely to occur in patient with septic shock a on high dose vasopressors. The norepinephrine, due to its predominant alpha stimulation, may cause myocardial small vessel constriction & a global myocardial ischemia. Detecting myocardial ischemia early & intervene by alteration in medication i.e. vasopressors may prevent a clinical event. We do suggest that if a PAC is inserted both the ScvO2 and SVO2 be monitored & a positive Gradient (ScvO2-SVO2 of a positive value) should be taken seriously & appropriate adjustment in treatment be made.
Whether this measurement can be used for an intervention at that stage needs to be studied more selectively. Although it was not the aim of our study, but we do feel the cut-off value of troponin post CABG should be reduced to 30 ng/ml. The study quoted earlier, correlate events with the troponin. At times AMI may occur without producing a change in clinical status.

Conclusion(s)

Both central and mixed venous oxygen should be measured, as a positive gradient is an evidence of myocardial ischemia.

Acknowledgement(s)

The authors are in debt to all the bed side nurses and the respiratory therapists for their cooperation in conducting this study- as it involves some extra work with meticulous care to the method of blood withdrawal and minimize the time interval between sample withdrawal and analysis.

References

1. Determination of troponin I release after CABG surgery. Noora et al. J Card Surg. 2005 Mar-Apr;20(2):129-35.
2. Controverversies in Intensive care medicine. Medizinisch Wissenschaftliche Verlagsgesellschaft.

2008: 77.
3. Usefulness of Cardiac Troponin I in Patients Undergoing Open Heart Surgery. Greenson et al. American Heart Journal. 2001;141(3).
4. Continuous mixed venous and central venous oxygen saturation in cardiac surgery with cardiopulmonary bypass. Lequeux et al. Eur J Anaesthesiol. 2010 Mar;27(3):295-9.
5. Venous oxygen saturation and lactate gradient from superior vena cava to pulmonary artery in patients with septic shock. Kopterides et al. Shock 2009 Jun;31(6):561-7.

Illustrations

Illustration 1

JPG

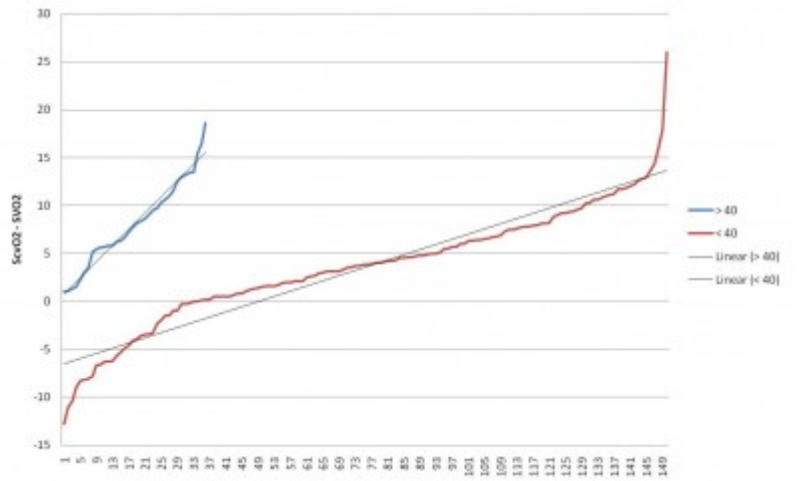

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Illustration 2

DOC formats

Parameters	Peak Troponin Surge <40 ng/ml	Peak Troponin surge >40 ng/ml	Significance
Total number of patients	24	6	
Male to female	19:3	5:1	*
Age	**51±4**	**58±8**	
Death	1	Nil	*
Diabetes Mellitus	**13/24(54.16%)**	**4/6(66.66%)**	
Hypertension	17/24(70.83%)	4/6(66.67%)	
Previous Myocardial infarction	18/24(75%)	4/6(66.67%)	*
Current Smoking	**1/24(4.16%)**	**2/6(33.33%)**	*
Hypercholesterolemia	1(0.09%)	0	*
TIA, CVA	**2/24(8.13%)**	**3/6(50%)**	*
Intermittent Claudication	**3/24(12.5%)**	**2/6(33.33%)**	*
Cross Clamp time (minutes)	72.3±24.57	96.6±40.2	*
Bypass time (minutes)	132.3±8.30	132.8±91.77	NS
Number of observations	**150**	**36**	
ScvO2-SVO2 Gradient	**3.75±6.12**	**8.18±4.52**	
Lactate difference (mg/dl)	0.551±1.41	0.79±1.71	
ICU Stay (days)	4.8±2.8	3.5 ±2.06	
Time to extubation (Hours)	39±45.88	26.25±30.24	
New RBBB Post op	1(9.09%)	1(20%)	*
New LBBB post op	1(9.09%)	0	*
Post op arrhythmia: AF	0	1(20%)	*

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