Title: Cough CPR for bradycardia management: Myth or Reality?

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Sir,

We read an interesting report by Karippacheril et al., regarding a patient who developed severe bradycardia/asystole during a total hip arthroplasty (THR) that responded to the ‘cough cardio-pulmonary resuscitation (CPR)’ [1]. We are not fully convinced by the author's conclusions and have some concerns regarding the same.

Bradycardia (<50 bpm) is common after spinal anesthesia and may in occur in up to 10% of the patients [2]. The causes of bradycardia after spinal anesthesia are not very clear and some of the reasons include blockade of T1-T4 cardiac sympathetic fibers, vasodilatation and decrease venous return to the right atrium. Also, bradycardia generally occurs within the first 60 minutes of spinal anesthesia, when the cardiovascular effects of the spinal anesthesia and positioning are maximum. Bezold-Jarisch reflex is a paradoxical increase in inhibitory neural activity to the heart due to poorly filled hypercontractile left ventricle and has also been reported to be a cause of severe bradycardia. It is most likely to occur due to sudden absolute/relative volume depletion that may occur when the effect of spinal anesthesia is maximum during initial phases, during extreme positional changes or sudden loss of blood during the procedure.

In the case described by Karippacheril et al., the patient was a healthy young adult who had severe bradycardia/asystole 90 minutes after spinal anesthesia during THR. They attributed the mechanism to Bezold-Jarisch reflex. But there was no sudden posture change or blood loss during that time that may have led to the situation. Moreover, if Bezold-Jarisch reflex
was suspected to be the cause, then the fluid bolus should have been given which was not done.

Management of symptomatic bradycardia leading to hypoperfusion to heart and brain is atropine and epinephrine. Transcutaneous pacing and dopamine are considered in severe cases when first-line drugs are not effective. As a standard practice, we routinely load a syringe with atropine and epinephrine in a patient undergoing surgery under spinal anesthesia. The patient reported here had severe symptomatic bradycardia with probably transient asystole. As a management option ‘cough CPR’ was considered initially. The sequence of events of loss of QRS complexes in electrocardiogram (ECG) while the pulse rate tracing and pulse oximeter readings were present are a bit confusing. Had the corresponding ECG tracings and screenshots from monitor were available, the picture could have been clearer. At that critical time, while the patient was being resuscitated, they might not have been able to keep a mental track of the events and use of screenshots and trends in monitor memory would have been better. Also, the flat line protocol was not followed. It is possible that some interference due to the use of electrocautery led to the loss of ECG trace.

A forceful cough may maintain enough blood flow to the brain due to increased intrathoracic pressure and may help the victim to maintain consciousness for a few seconds until the arrhythmia is treated. A forced cough may pump up to 700ml of blood from extremities if a healthy volunteer coughs forcibly from total lung capacity [3].

But in a patient with spinal anesthesia undergoing THR that too in lateral position the effectiveness of cough would be substantially reduced [4]. Also, severe bradycardia/asystole is an emergency and it would have been difficult to make the patient understand the intricacies of ‘cough’ maneuver to be effective for CPR. The American Heart Association have described it to be useful only transiently, for in-hospital situation in a controlled
environment in a conscious and responsive person [5]. However, its usefulness in the present scenario is doubtful as the authors have mentioned that the patient was ‘partially responsive’ at the time of the event.

We are surprised that the authors resorted to ‘cough CPR’ in their patient as a first choice instead of giving atropine or epinephrine which are usually loaded for all elective cases under spinal anesthesia for any eventuality. Also, there was no mention of the level of the spinal blockade at that time. We feel that the level of spinal anesthesia may have been higher and may have caused extensive sympathetic blockade and led to bradycardia. This is also evident from the fact that ‘cough CPR’ did not improve the condition of the patient remarkably. Authors lost the valuable time during this grave situation and delayed the use of atropine and epinephrine. The patient condition eventually improved after atropine and epinephrine. A further delay in the institution of the recommended therapy may have been catastrophic. We believe that priority in such a case should be immediately stopping the surgery, turning the patient supine, administration of 100% oxygen and atropine. The said patient remained lateral for some time while cough CPR was being tried.

The effectiveness of ‘cough CPR ’ for symptomatic bradycardia in a patient undergoing THR in the lateral position with suboptimal pulmonary function due to spinal anesthesia is doubtful. We do not confer to authors suggestion that ‘cough CPR’ should be attempted routinely in cases of bradycardia under spinal anesthesia.
References


