Treatment such as carotid sinus massage and Valsalva manoeuver cause vagal stimulation, inhibit conduction in AV node, and help resolve paroxysmal SVT. Pharmacological treatment is best reserved for those with haemodynamic changes, severe symptoms, or sustained arrhythmias. Adenosine is the drug of choice. The ACC/AHA/ESC guidelines recommend the use of IV propranolol or metoprolol if adenosine fails, and if arrhythmia still persists it recommends the use of verapamil. In case of resistance to pharmacotherapy or maternal instability, aggressive management strategies such as electrical cardioversion should be considered. Cardioversion has been found to be safe in all stages of pregnancy as negligible current reaches the foetus. However, transient foetal dysrhythmia may occur which warrants foetal heart rate monitoring.

We conclude that regional anaesthesia is a feasible, safe option for caesarean section in patients with structural cardiac lesion with SVT. The success depends on thorough knowledge, meticulous planning, and effective collaboration between the gynaecologist, cardiologist, and anaesthesiologist.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and anonymity cannot be guaranteed.

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Conflicts of interest
There are no conflicts of interest.

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to its popularity in procedures requiring cerebral oxygenation monitoring. However, NIRS may be associated with poor specificity in certain situations. We describe one such case where NIRS monitoring using cerebral INVOS™ (Somanetics Corp. Troy, MI) revealed persistent cerebral oxygen desaturation but was not associated with any postoperative neurological deficit in an elderly gentleman undergoing repair for type B aortic dissection.

An elderly gentleman, with no history of neurological disorder, presented with type B aortic dissection and underwent a hybrid procedure with a carotid-carotid bypass and an endovascular stent to treat the condition. Neurological monitoring was performed with INVOS™ (Somanetics Corp. Troy, MI) cerebral oximeter. Fresh sensors were applied and reliable recording quality was confirmed by signal strength index. Baseline bilateral cerebral oxygen saturation (rSO2) was low (Channel 1: 47%; Channel 2: 41%) [Figure 1]. The rSO2 did not reach 50% at any point during the procedure. A systematic approach to this unexplained cerebral desaturation was performed based on the algorithm proposed by Denault et al.[1] The rSO2 remained persistently and critically low with a mean of 38% in Channel 1 and 35% in Channel 2 during entire procedure. The patient did not have any major or minor neurological event during the hospital stay or at 6 months follow-up.

NIRS with the INVOS™ monitoring system has become increasingly popular for cerebral oxygen monitoring. It is based on the fact that oxygenated and deoxygenated haemoglobin have characteristic absorption spectra and light in the range of 650–1100 nm and has an absorbance that is proportional to the relative concentrations of these two chromophores.[2] The INVOS monitor is a saturation monitor that measures the ratio of haemoglobin and oxyhaemoglobin by using a single light-emitting diode and displays a single unitless value defined as “regional haemoglobin oxygen saturation (rSO2)”.[3] The typical acceptable range of rSO2 is 55–80%. It has been reported that rSO2 values below 59 provide 100% sensitivity and 47% specificity.[4] As seen in our case where the mean values were significantly lower with no postoperative neurological impairment, it is the specificity that is of major concern. Interestingly, in our case the rSO2 remained low throughout, and no considerable drop was seen during the procedure phase compared to the preprocedure evaluation. To address similar situation, it has been proposed that along with absolute numbers, perhaps a percentage decrease from baseline might be a useful criterion for cerebral ischaemia. A proposed cutoff value of 20% was reported to have 83% sensitivity and 83% specificity for cerebral ischaemia.[4]

Artificially low rSO2 values may be attributable to cranial bone anomaly or frontal sinus inflammation, presence of infrared-absorbing intracranial or intravascular pigments or dyes, optode positioning over an intracranial photon sink (i.e., intracranial venous sinus or haematoma), excessive photon scattering (i.e., hair or hair follicles), or dyshaemoglobinemias.[5] In this particular patient, despite critical evaluation, we were unable to find a definite cause for the low rSO2 values. However, it has been shown that during hypothermic cardiopulmonary bypass, as needed in this case, the rSO2 values can be paradoxically low and may have been the cause for the persistent low rSO2 value seen.[6]

NIRS may produce not only artifactually low rSO2 values but can also fail to detect cerebral ischaemia in certain cases. It is possible that the placement of sensors on the forehead may not be able to detect ischaemia of parietal or deeper lobes without reflecting any changes in the monitored frontal lobe. Infarct of the middle cerebral artery with an entirely satisfactory rSO2 values have been reported in the literature. Moreover, as NIRS reflects the balance between regional oxygen supply and demand, saturation may be near-normal in infarcted nonmetabolizing brain.

The problem with NIRS or any other monitoring system for cerebral ischaemia is absence of an objective gold standard with no single monitoring method providing perfect sensitivity and specificity.

Transcranial Doppler sonography and carotid artery stump pressure are the other monitoring techniques currently available; however, no single method has been proved to be superior to others. Considering that NIRS is easy to carry out and provides comparable accuracy, this should perhaps continue to be the technique of choice. However, rather than relying only on absolute rSO2 values of 50%, a 20% drop from baseline rSO2 values should also be incorporated in

| Channel | rSO2 Range | Baseline | Avg rSO2 |
|---------|------------|----------|----------|
| Ch1     | 28 - 48    | 47       | 38       |
| Ch2     | 15 - 48    | 41       | 32       |

**Figure 1:** Baseline bilateral cerebral oxygen saturation (rSO2)
order to increase the specificity of this diagnostic tool for cerebral ischaemia.

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There are no conflicts of interest.

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