Introduction
Cardiopulmonary resuscitation (CPR) is an emergency procedure for manually preserving brain functions by spontaneous circulation and breathing in a person who has suffered cardiac arrest. Though CPR alone is unlikely to restart the heart as it only restores partial flow of oxygenated blood to the brain and heart. It delays tissue death and extends the brief "window of opportunity" for a successful resuscitation without permanent brain damage.

If hemodynamics, neurological recovery and overall survival do not improve with optimal external chest compressions, consideration should be given to convert to open-chest cardiac massage whenever prolonged resuscitation is expected and wherever feasible. Open massage produces superior cardiac output, better perfusion pressure, improved neurological outcome and increased likelihood of successful resuscitation. When cardiac arrest occurs inside the hospital, outcome is better because CPR and internal cardiac massage can be started early.

Material and Methods
Patient 1
A patient aged 58 years who presented with history of exertional dyspnea was evaluated and diagnosed to have triple vessel disease with 80% lesion in the mid left anterior descending artery (LAD), first diagonal (D1) 70% lesion, first obtuse marginal (OM1) 70% lesion and right coronary artery (RCA) 100% lesion. All the pre-operative laboratory investigations were within normal limits. Echocardiogram (ECHO) revealed dilated left ventricle, ejection fraction 50%, mild hypokinesia of inter-ventricular septum, inferior and posterior wall, severe mitral regurgitation, trivial aortic regurgitation & trivial tricuspid regurgitation. The patient was on regular medications with beta blockers and sorbitrate. He was scheduled to undergo off-pump coronary artery bypass (OPCAB) and mitral valve replacement under cardiopulmonary bypass (CPB).

After placement of arterial and central venous, lines anaesthesia was induced with Inj.fentanyl 250microgram, Inj. midazolam 5mg & Inj. propofol 50mg. Trachea was intubated with no. 8.5 mm cuffed endotracheal tube using vecuronium 6mg as muscle relaxant.
Midline sternotomy was done. After harvesting internal mammary artery (LIMA) & saphenous vein (GSV) and heparinisation, LIMA was anastomosed to LAD off-pump. CPB was initiated with routine aortic & bi-caval cannulations, cooled to 28°C and cardioplegia administered. GSV was grafted to OM1, mitral valve replaced with 29 mm TTK-CHITRA mechanical tilting-disc type valve. CPB discontinued after fulfilling all criteria. Proximal grafting to aorta was completed, heparin reversed with protamine, hemostasis achieved & chest closed.

Inotropes on flow were Inj. adrenaline 0.05mcg/kg and Inj. dobutamine 5mcg/kg. Patient was shifted to the postoperative ward & electively ventilated. Hemodynamics and blood gases remained stable.

Three hours post-operatively, blood pressures (BP) started gradually reducing from 100/70 mm Hg to 60/40 mm Hg and heart rate (HR) dropped from 70 to 40 bpm. Treatment was attempted with increased infusion rate of inotropes, pacing and Inj. atropine 0.6 mg intravenous. In spite of all these measures, the patient developed cardiac arrest.

CPR was immediately commenced according to the American Heart Association (AHA) guidelines. In view of the possibility of surgical related complication, chest was opened bedside and internal cardiac massage instituted. Medications like Inj. adrenaline (1mg every 3minutes) totally 4mg, Inj. calcium gluconate (10ml of 10% solution to correct hyperkalemia) and Inj. sodium bicarbonate (to correct acidosis) according to the base deficit were administered intravenously. In addition, Inj. heparin 100mg was administered to treat coronary vessel thrombosis if any. Simultaneously causes for the asystole and subsequent cardiac arrest were sought for. Kinking of the grafts was ruled out. Ventricular fibrillation was treated with internal defibrillation along with Inj. lignocaine 100mg & subsequently with Inj. amiodarone 250mg.

Patient was successfully resuscitated after one hour of continuous internal cardiac massage as indicated by ROSC, stabilization of blood pressures at 110/70 mmHg & pulse of 100 bpm. Inotropes on-flow at that time were adrenaline 0.05mcg/kg/min, dopamine 10mcg/kg/min and dobutamine 5mcg/kg/min. Hyperglycemia was controlled with insulin infusion. Patient remained stable overnight. Patient was sedated, paralyzed & electively ventilated.

Patient developed fever on post-operative day (POD) 1 evening. Antibiotics were stepped up. Patient was further sedated & ventilated for 2 days maintaining stable hemodynamics. After full neurological recovery and meeting all extubation criteria, patient was extubated on POD2 evening. Vitals remained stable, inotropes were slowly tapered & drains removed on POD3. Invasive lines & pacing wires were removed on POD4 and patient was shifted to the ward. Subsequently he had an uneventful post-operative period till discharge. The cause of cardiac arrest remained unknown, though a mechanical valve dysfunction was suspected.

Patient 2
A 3 year old girl weighing 10 kg & post-operative case of surgical atrial septal defect (ASD) closure 1½ months back, presented to our center with complains of on and off fever, loss of appetite and purulent discharge from the sternal wound area since 1 month.

On examination, she was febrile. Local examination revealed infected old median sternotomy wound and exposed sternal wires, eroded manubrio-sternal junction and blood clots. Diagnosis of surgical site infection with a possibility of pseudo-aneurysm of aorta at the old aortic cannulation site was arrived at.

Laboratory reports revealed hemoglobin (Hb) of 7.3g%, raised white blood cell counts. Chest X-Ray revealed mild pleural effusion & ECHO showed mild pericardial effusion. She was posted for emergency re-exploration of the chest & wound debridement with adequate blood products available.

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Patient was shifted to the operation theatre; standard American Society of Anaesthesiologists (ASA) monitors (Electrocardiogram, non-invasive BP, pulse oximetry) were connected. Adequate venous access was secured. Arterial
Airway was secured after rapid sequence induction with Inj. propofol 20mg & Inj. succinylcholine 20mg. Patient was intubated with 6 mm cuffed oral endotracheal tube (COETT). Anaesthesia was maintained with N₂O & O₂ (FIO₂ 0.5) and 1 MAC isoflurane with Inj. atracurium boluses.

Old sternotomy incision was re-explored. Sternal wires were removed and thorough wound debridement was performed. Chest was closed after hemostasis.

After reversing the residual neuromuscular block, patient was extubated. At the time of shifting the patient out of operation room (OR), alarming bleeding was noticed from the sternal wound site. She was immediately shifted back to the OR and re-exploration was decided.

Adequate units of fresh, compatible whole blood were requisitioned for. Then she was administered general anaesthesia in the same sequence as described above.

On re-exploration, profuse bleeding from surgical site was noticed. There was an immediate fall in blood pressures to 30 mm Hg systolic after re-opening the chest. 1 unit of whole blood was transfused. Bleeding point at the old aortic cannulation site was identified and controlled simultaneously. A rent measuring about 2 cm over the old aortic cannulation site on the ascending aorta was sutured in 2 layers. Blood pressures were sustained with blood transfusion & phenylephrine boluses of 20 mcg.

Patient fibrillated & developed cardiac arrest after 5 minutes of initiation of surgery. She was ventilated with 100 % oxygen; CPR was initiated with internal cardiac massage. Defibrillation was attempted with 5J shock increasing in strength up to 20J after every 3 minutes of internal cardiac massage. Patient was administered Inj. adrenaline (0.1 mg/kg of 1:10000 dilution) every 3 minutes according to the AHA CPR protocols. Cardiac massage was continued. Inj. xylocard 100mg and Inj. amiodarone 250mg bolus in 20 minutes were administered. Cerebral protection was achieved with Inj. thiopentone 50mg bolus & ice packs. Various causes of ventricular fibrillation were considered. CPR was continued for 45 minutes without interruptions.

At the 46th minute, cardiac activity was noticed & heart maintained normal sinus rhythm with good ejections. All the bleeding points were sought for thoroughly and hemostasis achieved. Sternum was subsequently closed.

Stable hemodynamics was achieved with inotropes infusions of adrenaline 0.05mcg/kg/min and dobutamine 5 mcg/kg/min. Patient was shifted to the postoperative ward, electively ventilated overnight and was extubated next day after fulfilling standard extubation criteria & normal blood gases.

**Results**

In both these scenarios, patients survived because of early recognition of the adverse cardiac events, early CPR and prompt institution of internal cardiac massage. Patients did not show any neurological deficits and were haemodynamically stable post resuscitation and at discharge. This was possible due to the occurrence of cardiac arrest in the hospital setting & specifically so, in the cardiac Operation Room (OR) and cardiac ICU. These areas have advanced monitoring facilities were well equipped & adequately staffed to handle such emergencies. Rapid institution of internal cardiac massage & prolonged CPR duration contributed to the return of cardiac activity.

**Discussion**

Cardiac arrest is multi-factorial in aetiology and the outcome depends on timely and appropriate interventions. CPR is used on patients in cardiac arrest in order to oxygenate blood and maintain a cardiac output so that vital organs survive. The brain may sustain damage without oxygenated blood for four minutes and suffers irreversible damage after about seven minutes. Therefore, CPR is effective only if performed within seven minutes of the cessation of blood flow.

Chest compressions should be immediately initiated in a pulseless patient. With a straight downward thrust, the sternum is depressed 1½–2 inches (4–5 cm) in adults, 1–1½ inches (2–4 cm) in children, and then allowed complete recoil. For an infant, compression depth is ½–1 inches.
(1½–2½ cm). Compression and release times should be equal.

Chest compressions force blood to flow either by increasing intra-thoracic pressure (thoracic pump) or by directly compressing the heart (cardiac pump). During CPR of short duration, blood flow is created more by the cardiac pump mechanism; as CPR continues, the heart becomes less compliant and the thoracic pump mechanism becomes more important.

In-hospital cardiac arrest (IHCA) has not received the same level of focused research as out of hospital cardiac arrest (OHCA). The chances of survival were better when cardiac arrests occurred in the well monitored areas like OR, ICU and cath suites. This may be due to immediate resuscitation and internal cardiac massage possible in the cardiac ORs.

Yanagidani T et al took a total of 100 minutes to successfully resuscitate a patient without any resulting neurological damage. This was attributed to appropriate open chest massage. They opined that open chest massage should be considered if a case of cardiac arrest due to any coronary artery disease does not respond to the usual cardiopulmonary resuscitation.

Darragh Twomey et al in their study concluded that internal massage was superior to external massage for patients suffering a cardiac arrest after cardiac surgery. They have shown that closed chest massage generates a cardiac index of around 0.6 l/min/m² which rises to 1.3 l/min/m² or more with open-chest-CPR, accompanied by even bigger improvements in coronary perfusion pressure. Based on International Liaison Committee on Resuscitation (ILCOR) guidelines, they recommend early conversion to open-chest-cardiac massage in such patients in order to significantly improve the quality of cardiopulmonary resuscitation.

Animal studies done by Sanders et al showed that open-chest-CPR resulted in 4/5 dog survivors compared to none in their closed-chest group. Similarly, Kern et al in their study on 29 dogs examined whether open-chest cardiac massage could improve 7 day survival and neurologic function when instituted after the failure of standard closed-chest compression CPR. There was more success in dogs receiving open-chest cardiac massage, (14/14 vs 5/14; p less than .005), 24 hr. survival (12/14 vs 4/14; p less than .005), and 7 day survival (11/14 vs 4/14; p less than .02) than in those receiving continued closed-chest compressions. They concluded that resuscitation significantly improved if chest opening is instituted sooner.

Fialka et al in their study observed that patients with blunt trunk trauma and cardiac arrest after hemorrhagic shock may benefit from open-chest CPR. They stated that internal cardiac massage is very effective if started at least within 20 minutes after initiation of traditional CPR methods.

In the modern era, however, thoracotomy and open-chest cardiac massage are not part of routine CPR because of the high incidence of severe complications. Nonetheless, these invasive techniques can be helpful in specific life-threatening clinical situations that preclude effective closed-chest massage. Possible indications include cardiac arrest associated with penetrating or blunt chest trauma, penetrating abdominal trauma, severe chest deformity, pericardial tamponade and post cardiac surgical patients.

Lim GB in his study observed that many hospitalized children survive after prolonged resuscitation (more than 35 minutes) for cardiac arrest. Over half of survivors of prolonged resuscitation had no neurological sequelae. Longer duration of CPR may save the lives of children who otherwise would have died.

The 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care acknowledge that there is not yet enough scientific evidence to guide when further efforts of CPR would be futile for an individual patient and hence do not currently recommend a specific duration for resuscitation attempt.

Goldberger et al in their observational study evaluated a
potential association between duration of CPR and survival after in-hospital cardiac arrest. The evidence remains insufficient to recommend a minimum duration for an in-hospital resuscitation attempt. Instead, the duration of CPR should be determined on a case-by-case basis, delivery of higher-quality CPR and coordinated & fruitful teamwork. 18

Conclusion
As we strive & struggle to realize the goal of full neurological recovery after cardiac arrest, future studies should examine the strategy of initiating open cardiac massage by trained individuals if closed-chest CPR for \( \approx 15 \) minutes (or less) fails to resuscitate victims in-hospital settings. Prolonged CPR can result in high-quality survival if the patient has a potentially reversible cause for cardiac arrest. In all durations of CPR, IHCA, especially cardiac surgery patients have more odds for survival and favorable neurologic outcomes than OHCA.

References