intracranial hypotension, but other possibility of these features being secondary to mass effect from subdural collections was cannot be entirely ruled out. Keeping in mind subdural hematomas may be because of benign intracranial hypotension we adviced MR myelogram of spine to look for CSF leaks. MR myelogram showed thin layer of epidural fluid in lumbar spine extending from L1 to L5. Which was indirect evidence of CSF leak and collection of CSF into epidural space but was not confirmatory. As patient condition was deteriorating we planned for evacuation of SDH under GA. But before that we planned to put epidural blood. Before inducing the patient under fluoroscopy guidance in lateral position 25 cc of autologous blood was injected in T11-T12 epidural space. Then under standard general anaesthesia only left sided SDH was evacuated which was causing mass effect. Postoperatively patient nursed in supine position for 5 days and gradually reverse trendelburg position was given. Again at 45 degree propped up position patient started having headache, vomiting. Repeat epidural blood patch was given at T6-T8 position. Patient recovered well post procedure and there were no symptoms after 3 days even at erect posture. Conclusion: Benign intracranial hypotension can present with wide variety of symptoms and always have to be kept in mind. High index of suspicion, proper history and investigations, sealing of CSF leak with epidural blood patch will help the patient.

## ISNACC-S-09

ECG and echocardiographic abnormalities in head injury patients undergoing emergency surgical decompression

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**Background:** Myocardial dysfunction leading to circulatory instability (hypotension) during the perioperative period in traumatic brain injury (TBI). We intended to study myocardial dysfunction in TBI patients undergoing emergency surgical decompression and its association with neurological outcome. Methods: We recruited adult head injury patients undergoing surgery within 48 hours of insult. Preoperatively at bedside, ECG and Echocardiography were done. Postoperatively, patient was followed up for 48 hours with an ECG and an echocardiography. ECG was analyzed for heart rate, intervals (PR, QRS and QTc), morphologic end repolarization abnormalities (MERA), ST segment and T wave changes. Echocardiographic measurements collected were left ventricular ejection fraction (LVEF) and regional wall motion abnormalities (RWMA). GCS status at discharge was recorded. Results: Of 110 patients recruited before surgery: ECG abnormalities were sinus tachycardia (15%), prolonged QTc interval (42%), T wave abnormalities (42%), ST segment abnormalities (11%) and MERA (47%). Echocardiography showed LVEF <50% in 10% and RWMA in 10.8%. After surgery, ECG showed significant increase in sinus tachycardia and T-wave abnormalities, but reduction inprolonged QTc interval and MERA. Echocardiography showed significant decrease in LVEF <50% and RWMA. Presence of LV dysfunction were associated with lower GCS score at discharge. Independent predictors of LV dysfunction were poor GCS motor score and prolonged QT\_interval. **Conclusion:** Left ventricular dysfunction improved following surgical decompression. Poor LV function was associated with poor admission GCS and prolonged QT interval. Patients with poor LV function had lower GCS at discharge.

## **ISNACC-S-10**

Time is brain

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**Introduction:** Acute ischaemic stroke is a neurological emergency that can be treated with time sensitive interventions including IV TPA and endovascular approaches. Chief criteria to select patients for vascular reperfusion treatment is duration of stroke symptoms. These patients are usually agitated. General anaesthesia keeps them comfortable and motionless during endovascular reperfusion treatment. However retrospective studies show poorer outcomes with general anaesthesia due to haemodynamic instability, delay in treatment and prolonged intubation. We present two cases of stroke in young patients with timely intervention under general anaesthesia. Case Summary: Case 1: 34 year old male patient, with no comorbidities, brought to casualty within 2 hours 10 minutes of onset of right sided hemiparesis and inability to talk. Code GOLD activated. CT normal. MRI showed left basal ganglia infarct with totally occluded left ICA. IV TPA started. Patient taken for DSA within 3 hours of onset of symptoms where left MCA recanalization and thrombus aspiration with near total recanalization of cervical ICA done under general anaesthesia. Patient shifted to ICU on mechanical ventilation. Extubated after 24 hours. Post operatively no neurological deficit. Case 2: 19 year old female patient, known case of epilepsy brought to casualty within one hour 15 minutes of onset of left sided weakness and slurred speech. Code GOLD activated. CT normal. MRI revealed right internal capsule posterior limb acute infarct with right ICA not visualized. Patient taken