
Motor-evoked potential (MEP) monitoring is commonly performed during neurosurgery to monitor the integrity of the motor pathways. While muscle relaxation is not desirable for intra-operative MEP monitoring during neurosurgery, some surgeons, neurophysiologists and anaesthesiologists still prefer to use the continuous infusion of neuromuscular blocking agents to maintain partial neuromuscular blockade.

Van Dongen et al.,[1] suggested that stable neuromuscular blockade aimed at 45-55% of baseline can provide reliable responses during intra-operative myogenic MEPs. However, there have been no evidence-based comparisons of MEP monitoring with no and partial neuromuscular block (NMB).

In the April 2013 issue of British Journal of Anaesthesia, a study was published to compare the effects of different levels of NMB including no NMB on MEP parameters.[2] This study was approved by the Samsung Medical Centre Institutional Review Board (2011-04-010) and registered at www.clinicaltrials.gov. Between June 2011 and February 2012, 120 patients were enrolled in this prospective randomised study if they were receiving MEP monitoring craniotomies for tumour or aneurysm and spinal laminectomies. Patients having ASA physical status classification of III or greater and who could not undergo MEP monitoring due to central or peripheral neuromuscular diseases such as cerebral palsy, myasthenia gravis, acute spinal injury or neurologic shock were excluded from the study. Anaesthesia was induced by i.v. propofol with remifentanil through a target-controlled infusion pump and tracheal intubation was facilitated with rocuronium. Before rocuronium administration, the baseline twitch response was established with a neuromuscular transmission module.

The maximum electromyographic amplitude of T1 before rocuronium administration was considered to be the control response (Tc). Anaesthesia was maintained with propofol and remifentanil infusions. Subjects were randomly allocated into one of the four groups and were given doses of the neuromuscular blocking agent vecuronium adjusted every 15 min according to the group’s NMB target. Group A was to maintain two train of four TOF counts; Group B was to maintain a T1/Tc of 0.5; Group C was to maintain a T2/Tc of 0.5 (T1,2, first or second twitch height of TOF; Tc, control twitch height); Group D did not maintain NMB. The primary outcome measurement of the present study was the MEP amplitude, and also the co-efficient of variation (CV%) of all measured MEP amplitudes. Other variables measured and compared among the groups during surgery were (i) the incidence of patient spontaneous movements or respiration during MEP monitoring, (ii) any positive MEP changes during the surgery, (iii) the new onset of post-operative neurological dysfunction, (iv) the doses of anaesthetics administered and (v) the continuous end-tidal CO2 measurements.

All patient characteristics and perioperative clinical variables were similar between the four groups except for the patients’ height in group C, remifentanil infusion dose in group D (higher than group A or C) and mean infusion dose of vecuronium in C (lower than A or B). The mean MEP amplitudes of the left arm and both legs were significantly higher in group D than groups A, B or C. The mean amplitude of the left arm and right leg was significantly higher in group C than groups A or B. The CVs of the four limbs were significantly smaller in group D compared with group A, B or C. Although the mean arterial pressure was significantly lowest in group D, there was no difference in incidence of hypotension, bradycardia and use of vasopressors between the groups. There were
Previously Yamamoto et al. devised a new technique of post-tetanic MEP (p-MEP) and found p-MEPs could be recorded at a T1 of 1 mV or %T1 of 10% with no or mild patient movement in response to transcranial stimulation. These strategies can be used as alternatives for improved surgery and patient monitoring.

**REFERENCES**


The dynamic indicators of fluid responsiveness that are based on cardiopulmonary interactions in patients ventilated mechanically, such as respiratory variations in aortic blood flow peak velocity (DVpeak), respiratory variations in inferior vena cava diameter (∆IVCD), systolic pressure variation (SPV), pulse pressure variation (PPV), difference between SPref and SPmin (Δdown), difference between SPmax and SPref (Δup) and pleth variability index (PVI), have been shown to be predictive for fluid responsiveness. There are insufficient data on the efficacy of these dynamic variables for the prediction of fluid responsiveness in children. Children differ from adults in terms of arterial compliance, chest wall rigidity and lung compliance, and therefore, indicators based on pressure, such as PPV and SPV may not be as reliable in children.

The purpose of this study was to evaluate the predictive values of central venous pressure CVP, SPV, PPV, Δup, Δdown, ΔPeak, ΔIVCD and PVI for the determination of fluid responsiveness in paediatric patients during general anaesthesia. This study was approved by the appropriate institutional review boards and written informed consent obtained from parents of the children. Children aged 6 months to 9 year of age undergoing elective neurosurgery under general anaesthesia were enrolled in this study.

Patients were excluded if they had congenital heart disease, cardiac arrhythmia, ventricular dysfunction, unstable perfusion index (PI) (defined as a variation exceeding 30% over a 1 min period), pneumonia, atelectasis, upper respiratory infection symptoms or vasoactive and/or inotropic support. Anaesthesia was induced with thiopental (5-6 mg/kg), remifentanil (0.3-1.0 mcg/kg and inhaled sevoflurane. Rocuronium (0.6 mg/kg) was administered to facilitate tracheal intubation. Mechanical ventilation was instituted in a pressure-controlled mode adjusted to obtain a PaCO2 of 4.7-5.3 kPa during surgery. PEEP was not applied. Central venous catheter was inserted in right subclavian vein and catheter tip confirmed with ultrasound. An arterial catheter was placed in right radial artery and oxygen saturation measured continuously using Masimo rainbow SET monitoring system. Peak inspiratory pressure (PIP) was recorded. In addition, heart rate (HR), arterial pressure, CVP and end-tidal carbon dioxide (PECO2) were recorded.

Maximal pulse pressure (PPmax), minimal pulse pressure (PPmin), maximal systolic pressure (SPmax), minimal systolic pressure (SPmin) and reference systolic pressure at the end expiratory pause (SPref) at the end-expiratory pause were manually measured. SPV, PPV, Δdown and Δ up were calculated as follows: SPV(%) = 100 × (SPmax−SPmin)/(SPmax + SPmin)/2], PPV(%) = 100 × (PPmax−PPmin)/(PPmax + PPmin)/2] , Δdown = SP ref − SP min, and Δ up = SP max − SP ref. PVI was calculated using formula PVI = 100 × (Pmax-Pmin)/Pmax. Stroke volume index, ∆VMAX and ∆IVCD were measured using transthoracic echocardiography TTE.

After obtaining an expiratory tidal volume of 10 ml/kg, all variables were measured before volume loading and re-measured after fluid loading. A total number of 33 patients were included in the cohort study. There were no differences between the responders and non-responders in terms of clinical characteristics, PIP, PECO2, end-tidal sevoflurane concentration, temperature and haemodynamic variables. Fluid loading changed CVP, SPV, PPV and Dup in both responders and non-responders. However, ΔPeak, PVI, and SVI were changed by volume expansion in the responders only. Only ΔPeak (r = 0.516, P = 0.004) and...