Correspondence

Increasing tidal volumes and PEEP is an effective method of alveolar recruitment

To the Editor:
Atelectasis following general anesthesia (GA) is a regular finding caused by reduction in the functional residual capacity (FRC).[1] Atelectasis decreases partial pressures of oxygen in arterial blood (PaO_2). Reduction in FRC correlates well with an increase in alveolar to arterial differences of partial pressures for oxygen (PAO_2–PaO_2).[2] This difference in PAO_2–PaO_2 changes with variation in the FIO_2; however, ratio of alveolar to arterial oxygen partial pressures (PaO_2/FIO_2) is less dependent on the FIO_2.[3]

In this study we observed the effect of alveolar recruitment by increasing tidal volume from 8–18 $mL{\cdot}kg^{-1}$ (for ten breaths) along with a positive end-expiratory pressure (PEEP) of 5 cm of H_2O on oxygenation during GA. Seventeen adults undergoing elective surgery, and operated in the supine position were included. Initial ventilator settings included a PEEP of 5 cm H_2O. Arterial blood gas analysis (ABG) was performed at 30 (basal), 40, 60, 90 and 150 min following GA. Alveolar recruitment was applied after the basal ABG. Statistical analysis was performed using ANOVA. Mean peak inspiratory pressure and plateau pressure observed at the time of alveolar recruitment were 30.00 ± 9.55 cm H_2O and 24.90 ± 7.48 cm H_2O respectively. Postoperative chest *x-ray* revealed no evidence of barotrauma.

Alveolar recruitment resulted in a significant increase in the PaO_2, PaO_2/FIO_2 ratio and compliance along with a significant decrease in the PAO_2–PaO_2 during the study period (Table). Alveolar recruitment is an effective way of maintaining a desirable PaO_2 during GA. Increasing the tidal volume from 8–18 $mL{\cdot}kg^{-1}$ (+ PEEP 5 cm H_2O) was not associated with adverse events. Alveolar recruitment also results in better lung compliance - a prophylactic strategy for the prevention of lung injuries observed during mechanical ventilation.

P.K. Singh MD
A. Agarwal MD
A. Gaur MD
D.A. Deepali DNB
C.K. Pandey MD
U. Singh PhD
Lucknow, India

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Burst suppression ratio is the only determinant for BIS values below 30

To the Editor:
I read the article by Mérat *et al.* about bispectral index (BIS) monitoring and severe cerebral ischemia[1] with interest.

TABLE PaO_2, compliance ($mL{\cdot}cm^{-1}$ H_2O), PAO_2 – PaO_2, PaO_2/FiO_2 values (mean ± SD) at different time points. * Significant difference from basal value $P < 0.05$.

Variables	*Time in minutes following general anesthesia*				
	Basal (30)	*40*	*60*	*90*	*150*
PaO_2	204.7 ± 39	285.8 ± 45.3*	281.1 ± 39.7*	275.5 ± 43.3*	280.8 ± 37.2*
PAO_2–PaO_2	135.3 ± 39	54.2 ± 45.3*	58.9 ± 39.7*	64.5 ± 43.3*	59.2 ± 37.2*
PaO_2/FIO_2	409.4 ± 117	571.6 ± 135.9*	562.2 ± 119.1*	551 ± 129.9*	561.6 ± 111.6*
Compliance	27.8 ± 7.3	33.5 ± 6.1*	35.7 ± 7.4*	39.26 ± 0.5*	41.2 ± 4.4*

The BIS monitor is still a kind of "black box". Descriptions of the algorithm are sparse and, in their details, contradictory.[2,3] Recently a part of the BIS algorithm was made public:[4] burst suppression ratios > 40% are invariably and linearly correlated with the BIS (r = -1), according to the equation: BIS = 50 – burst suppression ratio / 2.

Conversely, BIS values below 30 are linearly correlated with the burst suppression ratio. Therefore, the reported BIS value of 8 can be directly translated into a burst suppression ratio of 84% according to the above equation.

A BIS value of 8 is not related to phase coupling, nor to bispectral analysis, but is just an effect of the burst suppression ratio.

Thus, the observation of Mérat *et al.* is not BIS-specific but merely secondary to the occurrence of a burst suppression pattern associated with cerebral ischemia. In principle, such extensive burst suppression pattern can easily be identified by visual inspection of the electroencephalography and does not require processed monitoring like the BIS.

Jörgen Bruhn MD
Bonn, Germany

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Reply:

We agree with Dr. J. Bruhn concerning the poor interest of the bispectral index (BIS) for values below 30. However our purpose wasn't to say that BIS always reflects the depth of anesthesia. We solely think that an unexpected modification of the BIS value, without modifications of anesthesia, is abnormal. In such a case, when the BIS decrease is unrelated to anesthesia, we suggest that the BIS may be useful to detect severe cerebral ischemia, whatever the BIS value.

Stéphane Mérat MD
Paris, France

Injury to the liver and spleen after diagnostic ERCP

To the Editor:

Endoscopic retrograde cholangiopancreatography (ERCP) is an invasive procedure performed to diagnose and treat pancreatic and biliary disease. In approximately 5%–10% of cases, the procedure itself causes adverse events.[1] Splenic injury is a relatively rare, but increasingly reported complication of endoscopic procedures.

A 42-yr-old man was referred for diagnostic ERCP because of intermittent epigastric pain. His past medical history was unremarkable. Abdominal sonography revealed cholecystolithiasis with a markedly dilated common bile duct. ERCP was performed with relative ease. The cholangiogram showed cholecystolithiasis, a distal common duct stricture, and several stones within the dilated prestenotic portion of the duct. The patient complained of diffuse abdominal pain soon after the procedure. Vital signs and physical examination were unremarkable.

Twenty minutes after, the patient was hypotensive (systolic blood pressure 70 mmHg), but was otherwise well. Intravenous saline was administered, and the blood pressure returned to normal. One hour later, hypotension recurred, and the patient's hematocrit was found to be 18%. After resuscitation with blood and crystalloid, a hemoperitoneum was found upon opening the abdominal cavity (2.0 L). Exploration revealed a splenic laceration as the source of bleeding. Other organs were normal. Conservative surgery was performed and the postoperative course was uneventful.

Several cases of splenic injury have been described after colonoscopy, and rare cases of splenic rupture after ERCP have been published. Splenic rupture during routine ERCP was reported in 1988.[2] A possible mechanism is the avulsion of the splenic vessels secondary to bowing of the endoscope in the stomach during attempts to pass the large endoscopes through the narrowed duodenum or while attempting to cannulate the papilla while in the "long" position.[3]

Splenic injury during endoscopy is a real possibility and may occur even when the procedure is not technically difficult. Delayed diagnosis is a characteristic feature in many cases. Although the signs and symptoms are the same as for splenic rupture from non-endoscopic causes, splenic injury needs to be considered if sudden abdominal pain, hypotension, or drop in hematocrit value occur after diagnostic or therapeutic ERCP. The diagnosis requires a high