Mallampati Classification, an Estimate of Upper Airway Anatomical Balance, Can Change Rapidly during Labor

THE Mallampati classification is a rough estimate of the tongue size relative to the oral cavity. Although the single usage of the Mallampati classification has limited discriminative power for difficult tracheal intubation, it is a simple, reproducible, and reliable preanesthetic airway assessment method when performed properly. In addition to difficult tracheal intubation, Mallampati class 3 or 4 is an independent predictor for difficulty of mask ventilation during anesthesia induction and presence of obstructive sleep apnea. Increased tongue volume during normal pregnancy increases even during normal pregnancy (7–17%), suggesting that tongue volume increases even during normal pregnancy as previously reported. Increased tongue volume is presumably due to fluid retention during pregnancy may be partly responsible for increasing both prevalence of obstructive sleep-disordered breathing in pregnant women and incidence of difficult tracheal intubation in obese subjects. The risk of emergency cesarean delivery and surgery for postpartum hemorrhage is particularly high in obese parturients, presumably because of their higher incidence of maternal complications and fetal growth retardation. Noticeably, there were no deaths during anesthesia induction, and five of eight anesthesia-related deaths resulted from hypoventilation or airway obstruction during emergence, endotracheal extubation, or recovery. Although safety of airway management during anesthesia induction seems to have greatly improved as a result of development of the airway algorithm and various intubation devices, an unsolved and significant problem in obstetric anesthesia is how to assess and manage the upper airway upon emergence and endotracheal extubation. The data of Kodali et al. suggest the labor is a potential risk factor for perioperative airway catastrophe in parturients in addition to obesity, craniofacial abnormalities, and sleep-disordered breathing. Pregnancy and labor are inevitable and physiologic processes for human beings that significantly burden the respiratory system by decreasing lung volume and thoracic compliance and narrowing the upper airway. Labor potentially makes some parturients more susceptible to pathologic upper airway narrowing.

Clinical Significance of Upper Airway Changes during Pregnancy and Labor

Kodali et al. did not directly test the clinical significance of the increased Mallampati class because none of the women underwent general anesthesia; however, careful interpretation of their data reveals noticeable features of the upper airway structures in pregnant women. First, Mallampati class 3 and 4 seem to be more prevalent in parturients at the beginning of labor (28%) than in the general adult population (7–17%), suggesting that tongue volume increases even during normal pregnancy as previously reported. Increased tongue volume presumably due to fluid retention during pregnancy may be partly responsible for increasing both prevalence of obstructive sleep-disordered breathing in pregnant women and incidence of difficult tracheal intubation in obstetric anesthesia.

More surprisingly and significantly, half of the parturients demonstrated Mallampati class 3 or 4 by the end of labor, predicting a further increase in difficulty of airway management during labor or immediately after delivery upon medical interventions such as general anesthesia.

Upper Airway Anatomical Imbalance in Parturients

The pharyngeal airway is a collapsible tube whose patency is precisely regulated by upper airway dilating muscles such as the genioglossus. Increase in the dilating muscle activity acts to maintain the narrowed pharyngeal airway during wakefulness in patients with obstructive
sleep apnea. Similar neural mechanisms presumably compensate the progressive upper airway narrowing in parturients. Preservation of these neural regulatory mechanisms is, therefore, crucial for parturients with a high Mallampati class to maintain their breathing. Regional anesthetic techniques have only minimal influence on the neural mechanisms; however, the neural compensatory mechanisms become weaker during general anesthesia, sedation, and sleep with residual anesthetics. The pharyngeal airway patency entirely depends on its structural stability in parturients undergoing emergency cesarean delivery during general anesthesia.

Structurally, the pharyngeal airway is surrounded by soft tissues such as the tongue and soft palate, which are enclosed by bony structures such as the mandible and spine. Size of the airway space is determined by the balance between the bony enclosure size and soft tissue volume (anatomical balance) when pharyngeal muscles are inactivated by general anesthetics and muscle relaxants. Pharyngeal edema, presumably due to fluid retention during pregnancy, and pharyngeal swelling acutely developed during labor increase the soft tissue volume surrounding the airway, narrowing the pharyngeal airway in parturients. Recent extensive research on the pathophysiology of upper airway obstruction revealed a significant role of the lung volume reduction in pharyngeal narrowing. Tagaito et al. demonstrated that lung volume dependence of pharyngeal airway patency is more pronounced in obese patients. Accordingly, obese parturients, a high-risk group for perioperative airway catastrophe, are prone to develop progressively narrower pharyngeal airways due to increase of soft tissue volume surrounding the pharyngeal airway and decrease of lung volume during pregnancy. Lung volume reduction during general anesthesia is known to be more prominent and prolonged in obese patients. General anesthesia for emergency cesarean delivery in obese parturients during or immediately after labor may tend to exaggerate upper airway swelling and lung volume dependence, in addition to impairment of neural compensatory mechanisms, and is, therefore, a worst-case scenario for upper airway maintenance. Application of positive end expiratory pressure during anesthesia and full consciousness at endotracheal intubation are strongly recommended for these patients.

Mallampati Classification for Assessment of Upper Airway Anatomical Balance

Kodali et al. demonstrated a decrease in upper airway volume of approximately 10 ml during labor and delivery. Although they did not simultaneously assess changes in Mallampati class in this group of parturients, it is of interest how much reduction of the upper airway volume, i.e., how much increases in the tongue volume, leads to a 1-point increase in Mallampati class. Assuming similar changes of the Mallampati class in both study groups, e.g., a 26-point increase of the Mallampati class in 61 subjects leads to a 10-ml reduction of upper airway volume on average, it can be roughly estimated that a 1-point increase of the Mallampati class approximately corresponds to a 20-ml increase of the tongue volume in women with Mallampati class 3 or 4 before labor. Upper airway volume differed between patients with and without difficult tracheal intubation by 30–40 ml. Tongue volume was significantly larger in patients with obstructive sleep apnea, by approximately 20–25 ml, than in non-apneic persons. For every 1-point increase of the Mallampati class, the relative risk of obstructive sleep apnea doubles and apnea hypopnea index increases by 5 h−1. Accordingly, a 20-ml increase of the tongue volume during labor potentially results in difficult tracheal intubation and upper airway obstruction under influence of general anesthetics and sedatives.

The Mallampati classification allows us to instantaneously identify such small but significant increases in the tongue volume at the bedside without using sophisticated apparatuses. The Mallampati classification originated in our specialty, and recently, clinicians and researchers in other specialties have recognized its usefulness for assessment of upper airway anatomical balance. We anesthesiologists should be proud of the Mallampati classification and are encouraged to use this classification to assess the upper airway anatomical balance with it before every general anesthesia induction. The article by Kodali et al. reminds us that the Mallampati classification is not static, but can change over hours with processes such as labor, and we should assess it just before instrumentation, rather than relying on an assessment even a few hours earlier.

Shiroh Isono, M.D., Department of Anesthesiology (B1), Graduate School of Medicine, Chiba University, Chiba, Japan. shirohisono@yahoo.co.jp

References
Impact of Analgesia on Bone Fracture Healing

BONE fractures are painful and, in general, temporarily disabling injuries that occur after trauma or as an end result of various pathologic conditions, such as osteoporosis or bone invading cancer (pathologic fracture). It is common practice for acute pain services to be heavily involved in the treatment of orthopedic patients, and although it is usually understood that fractures are very painful, few studies have directly attempted to measure or define the nature of the pain after a fracture. In addition, controversy exists regarding the effects of analgesics, including opioids and nonsteroidal antiinflammatory drugs, on skeletal tissue healing. In this issue of Anesthesiology, reports from Freeman et al.1 and Minville et al.2 show that commonly used fracture healing models also can be used to assess pain quantitatively and therefore to assess analgesic efficacy. Because bone is a highly innervated tissue, these models also can be applied to define the mechanism of pain transmission after fracture. Furthermore, and of even greater clinical importance, these fracture pain models lend themselves to studying the effects of pharmacologic interventions on bone healing.

Bone fractures are treated by restoring the anatomy of the broken bone (reduction) and immobilizing the bone pieces (fixation) while regeneration proceeds. Commonly, fracture fixation is done by casting the broken bone. This also can be achieved surgically by use of intramedullary rods or external fixators that use percutaneous pins or rods to hold the bone fragments in correct anatomical alignment. In these cases, the fracture site is not significantly disturbed and the fractures heal by bone regeneration. Initially, the fracture causes localized tissue hypoxia and hematoma formation and is soon followed by a robust inflammatory response. Next, mesenchymal cells migrate and proliferate at the fracture site to form a callus. Concomitantly, osteoblasts in the periosteum near the fracture site begin to proliferate. At the interface with the periosteal osteoblasts, the mesenchymal cells differentiate into chondrocytes and elaborate a cartilage matrix. Eventually, the chondrocytes undergo hypertrophy and mineralize the cartilage matrix, which then acts as a substratum for osteoblast bone formation. This process is reiterated from the periphery of the fracture site toward the center until the fracture is bridged with newly formed bone and is dependent on angiogenesis. Subsequently, the bony callus is remodeled to restore the mechanical properties of the bone. This is the normal endochondral ossification pathway of fracture healing and is often referred to as secondary fracture healing. In contrast, primary fracture healing occurs only after surgical fixation of the fracture in which the fracture callus is removed, and the bone ends are closely abutted and rigidly fixed in place, usually with a plate. In this case, the fractures heal slowly via normal bone remodeling mechanisms while the metal plate stabilizes the fracture and provides for any weight-bearing functions.

Pharmacologic treatments that can affect the molecular and cellular processes of bone regeneration can have a significant impact on healing. For example, fracture healing is severely impaired in rats treated with TNP-470, an antiangiogenic compound.3 More important in terms of pain management, nonsteroidal antiinflammatory drugs and cyclooxygenase-2 inhibitors, such as celecoxib, impair fracture healing in animal models.4,5 Eight weeks after fracture, twice the normal bridging time in rats, femur fracture healing had failed in approximately one third of female rats treated with celecoxib (4 mg/kg daily) for 5 days after fracture. However, celecoxib therapy before fracture or celecoxib therapy initiated 2 weeks after fracture had no significant effect on healing in rats. Limited retrospective data also indicate that these effects may translate to humans.6 Among acetabular fracture patients treated with indomethacin or localized radiation to prevent heterotopic ossification, 29% of those patients treated with indomethacin experienced a non-

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