The risk factors of medical device related (MDR) pressure ulcers at the nostril in oral maxillofacial surgery.

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Abstract

Background: Medical device related (MDR) pressure ulcers have occurred postoperatively in other types of surgery\(^1\). There are few reports about MDR pressure ulcers in head and neck region. We have experienced MDR pressure ulcers at the nostril in 16 cases for these 3 years. They all occurred in prolonged cases, at least 8 hours. In this study, we investigated risk factors of MDR pressure ulcers on the nostril in oral maxillofacial surgery retrospectively.

Materials and Method: We checked the anesthesia records of patients, over 20 year-old, ASA-PS I or II, who underwent oral maxillofacial surgery from April 2010 to September 2012. We investigated patient’s background (gender, age, height, weight), airway management and duration of anesthesia, and investigated these factors again focusing on prolonged cases over 8 hours.

Results and Discussion: Seven-hundreds-twenty-six cases were suitable for this study. There were significant differences in gender and duration of anesthesia between patients with and without MDR pressure ulcers at the nostril. Sixteen of 726 cases (2.2%) had postoperative MDR pressure ulcers, and 15 of 16 cases (93.8%) were male, while 384 of 710 cases (54.1%) were male (\(p=0.0037\)). Remaining one case was female. Duration of anesthesia was 945±341 minutes in 16 cases with pressure ulcers at the nostril, while it was 345±218 minutes in 710 cases without pressure ulcers (\(p=0.0001\)). Pressure ulcers in 11 cases with nasotracheal intubation were caused by the nasotracheal tube, and pressure ulcers in 5 patients with tracheotomy were caused by the nasogastric tube.

The prolonged cases more than 8 hours were 87 cases, and 48 cases were male (55.2%). Gender still remained as a risk factor associated with MDR pressure ulcers on the nostril (\(p=0.0041\)).

Conclusion: Gender and duration of anesthesia were suggested as risk factors of medical device related (MDR) pressure ulcers at the nostril in oral maxillofacial surgery.

Diffusion of nitrous oxide through endotracheal tube cuffs

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Aim
During anesthesia with nitrous oxide, nitrous oxide enters the cuff of the endotracheal tube, which increases the pressure inside the cuff and may cause damage to the tracheal tissue. A new line of endotracheal tubes has been reported to overcome this problem. In this study, we used one of these tubes to compare the permeability of nitrous oxide through endotracheal tube cuffs with a normal tube.

Methods
Protex Blue Line Profile Soft Cuff (SPN) from Smiths Medical and Mallinckrodt™ Nasal RAE Tracheal Tube Cuffed Murphy Eye (MRN) from Mallinckrodt were prepared for this study.
Five kinds of gases were prepared and inflated into the cuff of these two cuffs respectively at the pressure of 30 cmH₂O; Air (group A), 100% oxygen (group O), 70% nitrous oxide/30% oxygen (group 70N), 50% nitrous oxide/50% oxygen (group 50N), or 30% nitrous oxide/70% oxygen (group 30N). Cuff pressure was recorded at every 5 min for 60 min. Data were compared using statistical analysis software with significance set at p<0.05.

Results and Discussion
In group A, the cuff pressures in the MRN and SPN were 5.5±1 cmH₂O at 60 min. In group 70N, however, the cuff pressures in the MRN and SPN at 60 min decreased to 3.2±0.4 cmH₂O and 5.5±1 cmH₂O, respectively. In the nitrous oxide-containing groups (70N, 50N, and 30N), the time needed for cuff pressure in the MRN to reduce to 15 cmH₂O was clearly faster than that needed in the SPN. Moreover, cuff pressure in the MRN and SPN was decreased significantly faster in group 70N after 10 and 20 min, respectively, compared with other groups. Both MRN and SPN in the nitrous oxide-containing groups showed a significant decrease in cuff pressure after 5 min compared with group A, although cuff pressure of SPN maintained higher than that of MRN. It was suggested that the decrease in cuff pressure had been caused by the diffusion of nitrous oxide to outside of the cuff.

Conclusion
Recently, some tube cuffs were developed to prevent cuff pressure change due to permeability of nitrous
oxide. However, this function may be still insufficient in spite of some improvement of permeability. We have to pay attention to cuff pressure during anesthesia using nitrous oxide.
Effect of adrenaline or noradrenaline with/without lidocaine on the contraction response in LPS-treated rat thoracic aorta

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Abstract

Background
It is well known that analgesic effect is often insufficient when a local anesthetic is administered in acute inflammatory tissues. This phenomenon has been explained by reduction in the pH of inflammatory tissues and/or vasodilation of the inflammatory area. Vasoconstrictor, therefore, is usually contained local anesthetics to prolong the duration of analgesia. Effects of vasoconstrictors (adrenaline: AD, noradrenaline: NA), however, has not been investigated sufficiently with local anesthetics on dilated vessels in inflammatory tissues. There are few reports that investigated the effect of AP and NE to a lipopolysaccharide (LPS) treated vessels in the presence of a lidocaine. In this study, we prepared the blood-vessel inflammation model using LPS, and investigated the contractile effects of AP and NE on LPS-treated vessels in combined with lidocaine.

Methods
Sixteen male Wister rats aged 6 ~ 8 weeks were used in this study. Thoracic aortas were dissected from rats and cut into about 3-4-mm-long rings. Each ring was stretched by a pair of hooks in an organ bath (5 ml) filled with Krebs-Henseleit solution (37°C, pH=7.4). After exposure of LPS (1 micro-g/ml), AD or NA were applied as a vasoconstrictors in a cumulative manner from $10^{-9}$ to $10^{-5}$ M with or without lidocaine ($10^{-4}$ M). Changes in isometric vasocontractions were recorded continuously with an amplifier system using the PCD-30A computer system.

Results and discussion
LPS treatment attenuated the concentration-dependent contraction by AD and NA in the presence of lidocaine in a time-dependent manner (Fig.A,B). In the aorta with LPS treatment, on the other hand, lidocaine enhanced the contractile responses produced by low concentrations by $10^{-9}$ to $10^{-5}$ M AD, while lidocaine has vasodilation effect. Analgesic effect, needless to say, depends on the concentration of lidocaine. These results suggested that concentration of AD is important for sufficient analgesia.
Conclusion

In our results, LPS treatment attenuated contraction response by either AD or NA. Lidocaine was not effective on it in spite of its vasodilation potential.