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Case Report

Failure to restore respiratory effort after reversing neuromuscular blocking agents following general anesthesia

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Abstract

A male patient of 25 years was admitted with the complaint of Deviated Nasal Septum (DNS) Who was scheduled for submucous resection of septum (SMR) operation under General Anaesthesia. All routine Investigation reports time were within normal limit. Patient's respiratory effort was appeared after 13 hours following reversal with anticholine esterase. It is expected that normal respiratory effort returns within 5-10 minutes after reversing the neuromuscular blocking agents. It was a rare case of probably acetylcholine esterase enzyme abnormalities and it is anxious and distressing to an anaesthetist Complete recovery outcome was delayed. Some measures were taken including blood transfusion but ultimate patient's outcome was good.

Introduction

It is expected that normal respiratory effort returns within 5-10 minutes after reversing the neuromuscular blocking agents with Anticholine esterase (Neostigmin) at the end of operation. Unable to return respiratory effort after 30 minutes following administration of Anticholine esterase (Neostigmine) is worried to an Anaesthetist. It requires relevant investigations unless an obvious preoperative or perioperative cause is apparent^{1,3}. Restoration of respiratory effort at the end of operation may appear in one of two ways, first one in spite of the administration of an adequate dose of anticholine esterase, there is no respiratory effort and second one following reversal with anticholine esterase, respiratory effort appear but tidal volume is inadequate^{4,5}. However an anaesthetist have to handle with care this condition.

Case report

The patient was admitted in North Bengal Medical college hospital, Sirajganj with deviated nasal septum (DNS) and he was prepared for submucous

resection of the septum (SMR) operation. He was sent for pre-anesthetic check up. All his clinical investigations were normal except ESR which was slightly elevated. He was given anesthetic fitness as usual. In the operation theatre he was also instructed that he would have to take breath by mouth as both of his noses would be blocked with tight pack. He was given induction with thiopental sodium at a dose of 7 mg/kg body weight, and Intubation was done after administration of depolarizing short acting muscle relaxant (Suxamethonium) 1.5 ml/ kg. Anesthesia was maintained with oxygen, nitrous oxide and Halothane and long acting muscle relaxant (vecuronium bromide). Pethidine-50 mg was used in between operation. Towards the end of operation all the anesthetics were omitted and the patient was given reversal (Neostigmine) but there was no respiratory effort. Manual ventilation was maintained for about 1 hr but no signs of respiratory effort was observed. So ventilation was maintained manually and all investigations including water and electrolyte balance and blood gas analysis was repeated. The reports revealed elevation of K⁺ and slight depletion of sodium.

Sample of blood was taken and transfusion of whole blood started. Proper nursing care was continued. Other supportive treatment was carried out. Injection Naloxone, Injection 10% dextrose, Injection Frusemide all were given. On the next day at 8 am (after 11-hrs) signs of respiration was found to return very slowly and after 13 hours the patient started breathing deeply and rhythmically. Thirteen hours of anxiety and tension thus came to an end.

Discussion

The automaticity of the respiratory centre is affected by afferent impulses from the carotid body (Chemoreceptor), by the pH of the cerebrospinal fluid and $p\text{CO}_2$ of the perfusing blood, and by higher centres.³ All of these afferent mechanisms as well as respiratory centre itself may be affected by anesthetic drugs.⁴ The efferent pathway via the phrenic nerve to the diaphragm may be blocked at the neuromuscular junction. Mechanical impairment of ventilation due to obstruction of the air passage or diaphragm must be excluded as a cause. Failure to make any respiratory effort is usually due to central depression of the respiratory centre by drugs or hypocarbia^{4,5}. In patients with severe airways disease and with significant preoperative hypoxic drive, a normal or raised $p_{\text{a}}\text{O}_2$ may depress reflex respiratory drive, making the re-establishment of normal respiratory effort difficult to achieve postoperatively^{5,7}. The causes of hypoventilation include those affecting the respiratory centre such as drugs, hypocarbia, an anoxic insult to the respiratory centre, acidosis, loss of hypoxic drive due to high $p_{\text{a}}\text{O}_2$ and failure to compete the reversal of the neuromuscular block⁸. In the absence of adequate gas exchange due to inadequate tidal volume, artificial ventilation must be maintained. Artificial ventilation should be continued whilst an effort is made to correct any metabolic or respiratory abnormality and whilst any residual drug effect has time to be dissipated. It is often

preferable to accept the necessity for intermittent positive pressure ventilation for 12 hours or more rather than to resort to compounding any pharmacological problem by the use of antidotes and analeptics. The ability of a patient to raise his or her head off the pillow for 30s and to sustain a cough is considered to be evidence of adequate return of neuromuscular function^{5,6}. If a peripheral nerve stimulator is available, the restoration of the fourth-to-the first ratio of the train-of-four to above 70 per cent is convincing confirmatory evidence⁹. It is first necessary to demonstrate that the cause of failure to restart normal respiration is not due to a cause other than residual neuromuscular block. To determine whether there is depression of the respiratory centre's response to carbon dioxide by opiate drugs, naloxone should be administered. If the patient had preoperative pulmonary disease, the possibility of reduced hypoxic drive causing respiratory depression can be tested by cautiously reducing the inspired oxygen content to 21 per cent. Adequate oxygenation during this maneuver should be confirmed by pulse oxymetry or blood gas analysis. If there is any evidence of residual block it is reasonable to administer a further 2.5 mg of neostigmine with atropine⁸. If the patient is suspected of having a dual block following the use of large amounts of suxamethonium, this may cause a short-lived partial reversal when 10 mg edrophonium is administered, the patient should be electively ventilated for 2-4 hours to allow the drug to be excreted, mannitol is often helpful in this condition.^{3,7,8} In this case all drugs were used. No drug overdose done. But antidote of narcotic analgesic (pethidine) was not used due to non availability of this drug. During recovery the patient was able to raise his head off the pillow for about 10s and he was able to catch anaesthetist fingers, and also tried to open his eyes. All sorts of stimulation were given to the patients but there was no response from the patient. But afterwards the patient responded well. Transfusion of fresh blood helped in this case as it raised serum cholinesterase which was also helpful to return respiration

quickly^{2,8}. During discharge the patient was issued a red card and was advised to preserve that card for ever for further anaesthesia and operation when necessary⁹

Conclusion

Although this type of cases are distressing, awesome and painstaking for an anesthetist, one should be alert during this situation. An anesthetist should be cool, calm and quite to handle this abnormal types of cases and apply his skill and knowledge properly. Co-ordination from the surgeon and other OT staff is absolutely essential. Residual neuromuscular blockade should never jeopardize a patient's life if adequate ventilatory support is maintained. Injudicious use of drugs to reverse the condition may cause complications and compromise ultimate recovery.

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