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Original Article

In high Spinal Block of apnoea is secondary to hypotension but not primary for respiratory muscle paralysis Experience in Rajshahi Medical Collage Hospital

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Abstract

This experience was based on ten cases of caesarean section. After spinal anesthesia within few minutes-these patients become cyanosed and there was profound bradycardia and hypotension.

Ventilatory support was given by IPPV and I/V bolus vasopressor was given. The BP was improved spontaneous respiration returns. The artificial ventilatory support was with drawn again. So artificial ventilation was reinstituted and vasopressor was given both intermittently and as infusion. The patient began to take spontaneous respiration and the blood pressure became static. There was no further fall of BP and no further deterioration of respiration.

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Introduction

In anesthesia specially spinal anesthesia for C/S frequently high block occurs. In such alarming situation the anesthesiologists are tensed. The cardiopulmonary resuscitation is done by IPPV with-face mask (Endotracheal tube) and vesopressor. If cardiopulmonary resuscitation is applied in due time and effectively good result comes. Sometime ventilation is supported wrongly for long time, but it is not essential as this apnoea is secondary to hypotension induced by spinal anaesthesia and ischaemia to respiratory centre which ceases motor impulse to the diaphragm by the phrenic nerve.

So if hypotension is corrected and the temporary respiratory support is given, apnoea is also corrected due to improved circulatory status to respiratory centre.

Material and Methods

Vasopressor- Ephedrine
Dopamine
Adrenaline
Dobutamine

Patients of A.S. grade I & II

All resuscitating equipments
Facilities for G.A
Defibrillator
E.C.G Monitor
Pulse oxymeter etc.

All the patients were given spinal anesthesia in sitting position after proper aseptic preparation and the patient was rapidly repositioned to lying position. The dose was calculated as per body weight. The patients when they were in apnoic situation temporary artificial ventilation was given

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and Inj. Ephedrine was first used but when improvement of cardiovascular status was observed the Inj. adrenaline was pushed.

Table-I

Age in years	Weight in kg.	Height in fit & Inch
25	75	5′-1″
24	65	5′-1″
30	76	5′-0″
28	80	5′-0″
19	60	5′-2″
19	65	5′-0″
22	70	5′-1″
30	60	5′-4″

Table-II

Dose of Ephedrine in mg	Dose of Adrenaline in microgram
30 mg	330 mc
22 mg	250 mc
45 mg	500 mc
50 mg	750 mc
15 mg	200 mc
30 mg	300 mc
30 mg	300 mc
15 mg	150 mc

Table-III

Dose of Adrenaline in microgram	Time of cardiac arrest after spinal block
330 mc	7 minutes
250 mc	10 minutes
500 mc	6minutes
750 mc	5 minutes
200 mc	9 minutes
300 mc	8 minutes
300 mc	8 minutes
150 mc	11 minutes

Results

1. More the weight more the vasopressor adrenaline were required.
2. Again cardiac arrest was earlier in patient with greater body weight.

Discussion

If local analgesic is pushed in C.S.F in patients with higher intra abdominal pressure-there is tendency of L.A. to migrate up ward through C.S.F. Higher is the migration more is the block especially autonomic one which-needs smaller conc. to be effective.

For up ward significant migration of L.A through C.S.F the explanation is as follows.

The increased intra abdominal pressure leads to increased pressure on Inferior vena Cava which can not drain epidural plexus of veins easily by the segmental veins.

This leads to engorgement of epidural plexus of veins. As a result there is increased pressure by the engorged epidural plexus of veins on the under lying duramater which in turn sifts the spinal C.S.F. into cranium so the spinal C.S.F is smaller in volume.

Now if L.A is pushed/ Kg body weight irrespective height-there will be incased conc. of L.A. in C.S.F. After proper absorption of L.A. in the surrounding nerve roots and spinal cord parenchyma the excess local analgesic will rapidly migrate upward through C.S.F the migration more is the block.

Now question comes why the patients suffers from apnoea?

The concentration of L.A in C.S.F is so minimum that is sufficient for autonomic block but insufficient for analgesia and motor block. This autonomic block is associated with hypotension bradycardia, with impending or established cardiac arrest so blood supply to brain stem is reduced therefore the motor impulse from brain stem to diaphragm by phrenic nerve is interrupted and the diaphragm can not work so the final result is apnoea which is indirect or secondary to spinal induced hypotension.

Again the root value of phrenic nerve is 3 to 5 cervical the location of corresponding spinal segments is on the cervical forward convexity there by gravitation all migration of hyperbaric local analgesic to reach there is very unlikely-so phrenic nerve paralysis is also equally unlikely.

So when apnoea results few minutes ventilation by intermittent positive pressure together with mximum emphasis for cardiovascular support by vesopressor, inotrops should be the ideal way for management.

This few minutes ventilation by intermittent positive pressure will replace the lost oxygen lungs and the vasopressor, inotropes improves blood supply to the brain stem so the respiratory centre adequately perfused with oxygenated blood.

The motor power to phrenic nerve is re established the victim parents began to rebreathe -no requirement to continue the IPPV for longer time against the patients own normal, respiratory effort which creates an embarrassing situation for the patients like E.T.T. induced laryngo tracheal irritation and related C.V.S. Changes Terrific psychological insult, mask ventilation induced gastric dilatation and related consequences.

Conclusion

As the no of cases included in this experience based study is few in order to enrich the experience further more and more cases should be included here. As the situation regarding the management of patient of cardiac arrest is very time limited and alarming, the concerned anesthetist should have proper skill in this respect and that skill should be applied in due way otherwise grave consequences will result.

Reference

1. Datta S. The obstetric Anesthesia Handbook, 3rd ed. Mosby 2000.
2. Holdcroft TT. Principles and Practice of Obstetric Anesthesia. Blackwell, 2000.
3. Koren G, Pasruszak A, Ito S. Drugs in pregnancy, N Eng J Med. 1998; 338: 1128-1137.
4. Morgan P. Spinal anaesthesia in obstertrics. Can J Anaesth. 1995; 42: 1145-1163.
5. Axelrod I, Weinshilboum R. Catecholamines. N Engl Med. 1972; 287; 237-242,
6. Smith LDR, Oldershaw PL. Inotropic and vaso-pressur agents. Br. J Anoesth. 1984; 56: 760-767.
7. Allwood MI, Cobbold AF. Peripheral vascular effects of noradrenalin, Isoprophylnor-adrenaline, and dopamine. Br Med Bull. 1963; 19: 132-136.
8. Aviado DM. Cardiovascular effects of some commonly used pressor amines. Anestheiology. 1959; 20: 71-92.
9. Responses to anesthesia and surgery. Br J Anesth. 1984; 56; 725-739.

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