SUDDEN DEATHS DURING LAPAROSCOPIC STERILISATION IN CAMPS

By

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SUMMARY

This Study observed four sudden deaths during laparoscopic sterilisation in camps among 24000 cases, one following sedation and 3 following pneumoperitoneum. Mortality rate was 1 in 6000, which is comparatively high. Procedural pitfalls, physiopathogenesis of catastrophies and preventive measures to avoid such deaths are discussed. Since artificial pneumoperitoneum is the main lethal cause, use of CO_2/N_2O as the insufflating agent with pneumo apparatus instead of O_2 through polythene tubing and restriction of number of cases for sterilisation in camps have been suggested.

Introduction

Various major and minor complications during laparoscopic sterilisation are well-known. If adequate time is obtained to table such complications the patient can be saved; but a sudden death jeopardises the whole atmosphere in a camp putting the surgeon and the organisers in a stressed situation.

Material and Methods

North Bengal Medical ollege teams have performed 24000 laparoscopic sterilisation cases during June 1985 to December 1987 in the camps of Darjeeling, Jalpaiguri and Coochbehar Districts. These teams have faced 4 cases of sudden deaths during the procedure. All other minor and major complications were tackled successfully and the patients were saved.

Number of cases attending each camp varied from 25 to 500 cases per day per two surgeons. Therefore thorough clinical screening of all the patients before operations could not be done. Inj. pethidine 100 mg., Inj. phenergan 25-50 mg. and Inj. atropine were given I.M. ½ hour before sending them to operation Table.

One case expired following sedation only. Half-an-hour after sedation she was put to O.T. where uterus was found to be 24 weeks' size due to pregnancy, which she concealed; she was sent back to pre-operative room where she fell asleep. During sleep she expired which was detected after four hours, the exact time of death was unnoticed.

Other three cases expired within 2-3 minutes following the completion of

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pneumo-peritoneum. Series of events were like this: sub-umbilical infiltration of 1% lignocaine was given in one case and no local anaesthesia was given in other two cases. Pneumo-peritoneum was done without any difficulty by about two litres of oxygen at a rate of one liter per minute. Immediately after withdrawal of the veress needle whole body of the patient went into convulsive movements with rigidity for a short while; they became unconcious veins of the lower extremities upto the hips were engorged. Respiration, heart sounds and peripheral pulse were gradually slowing down in both rate and volume and ultimately stopped within 2-3 minutes. There were no cyanosis and no 'mill-wheel' murmur in the heart.

In one case pneumo-peritoneum was released mmedately, but there was no improvement. As soon as convulsive movements started diazepam 10 mg. I/V and Decadron 16 mg. I/V were given. They were intubated, positive pressure oxygen and external cardiac massage were given but without any result, even after administration of adrenaline (1:1000) intravenously. They were watched further for two hours. Post-mortem examination could not be done though this was 100% essential for the confirmation of the causative mechanism of death.

Discussion

Without post-mortem examination it is not possible to pin-point the exact pathogenesis leading to death. In the first case who was moderately anaemic pethidine and phenergan might have produced hypotension and respiratory depression (Parikh, 1985) which was unnoticed until her death. Moreover she was starving for almost 24 hours causing hypoglycaemia resulting in less uptake of oxygen by the vital centers of the medulla (respiratory, cardiac and vaso-motor) which became subnormal in their function. Brain-stem reticular formations also became less active resulting in stuporous condition which probably became stil worse by the depressive action of the sedative applied on the reticular activated system (R.A.S.).

In other three cases irrespective of application of lignocain as the local anaesthetic agent the catastrophic episodes were the same. So lignocaine anaphylactic reaction could not be held responsible (in one case).

Vascular or gut injury by the veress needle cannot produce death so quickly.

Direct insufflation of a bigger blood vessel by the veress needle would have produced mill-wheel murmur in the heart which was absent in all the three cases. Gas embolism is generally put forward as the explanation for such deaths, but embolic manifestations were absent in these three cases.

Gas embolism in coronary arteries can be excluded by the fact that it would have produced ventricular tachycardia, fibrillation and cardiac arrest but not convulsions which indicate involvement of the cerebral cortex first.

In the present series oxygen was used as the insufflating agent in all the cases, which was used directly from the oxygen cylinder to the veress needle through polythene tubing. Oxygen was used because of its easier availability than CO_2 or N_2 O. But as the oxygen cylinder cannot be used in the carbon dioxide pneumo apparatus because of absence of fitting arangement, increased

intraperitoneal pressure could not be read. Argument for cardiac arrest due to vagal reflex from abdominal distension and cardiac compression due to Trendenlenerg position can also be discarded because convulsion preceded cardiac stoppage.

In our three cases events leading to death may be explained in the folowing way (vide chart): Artificial pneumoperitoneum led to the compression of inferior vena cava and elevation of the diaphragm. Compression of the inferior vena cava caused less venous return, engorgement of the veins of the lower limbs, less cardiac output and less blood flow to the brain resulting in cerebral ischaemia. Ischaemia of the sensory cortex produced loss of consciousness and that of motor cortex produced convulsions and rigidity via reticular formation and hypothalamus. As a result of medullary ischaemia which was severe and prolonged, vasomotor, respiratory and cardiac centers were depressed producing stoppage of respiration, cardiac slowing down and ultimately cardiac arrest within 2-3-minutes (Guyton, 1986). Fall of B.P. produced a vicious cycle adding further medullary depression. As the medullary depression was not mild and transient, C.N.S. ischaemic response was not noted (Ganang, 1981). As the diaphragm was lifted upwards there were rise of Intrathoracic pressure and less inhalation of atmospheric air resulting in asphyxia like condition with hypoxia and hypercapnoea which added to the ischasmia of cerebrum and medulla.

In the present series oxygen was insufflated at the rate of one liter per minute and not more than two liters in each case. Still catastrophy occurred in three cases only. Clinically there was no cardiac or pulmonary abnormality. It might be that in these particular cases low venous pressure in the inferior vena cava (normally 15-20 mm Hg) aggravated by the Trendenlenberg position (Semm, 1975) or low vena caval tone could not withstand artificially raised intra-abdominal pressure of 10-15 mm Hg resulting in diminished venous return and/or lesser tonus of the diaphragmatic muscles resulting in excessive elevation than usual with the consequence of lesser inhalation of atmospheric air.

Parikh (1985) commented that deaths from laparoscopic ligation should not be more than 1 in 10,000 cases and ideally it should be 1 in 1,00,000. Bhatt *et al* (1981) reported a mortality rate of 1.5 per 10,000 cases in camp sterilisation. American Association of Gynaecologic Laparoscopists observed a mortality rate of 0.8 per 10,000 in 1973-1974 and zero in 1974-75 in their cases of laparoscopic sterilisation (Hulka, 1977). In contrast to all these studies we had the mortality rate of 1 in 6,000 cases which is very high and in 3 of 4 cases death occurred after creation of pneumo-peritoneum.

Therefore, gases like CO_2 or N_2O pneumo apparatus should be used with utmost care for creation of pneumoperitoneum by the experienced surgeon in presence of expert anaesthetist to avoid such sudden deaths during laproscopic sterilisation in camps. It should not be done hurriedly and number of cases should be restricted to 50 per surgeon per day and never be 300-400 cases per day to satisfy the target set by the authorities. Meticulous preoperative check up, experience and postoperative surveillance are all necessary to minimise sudden deaths from laparoscopic sterilisation.

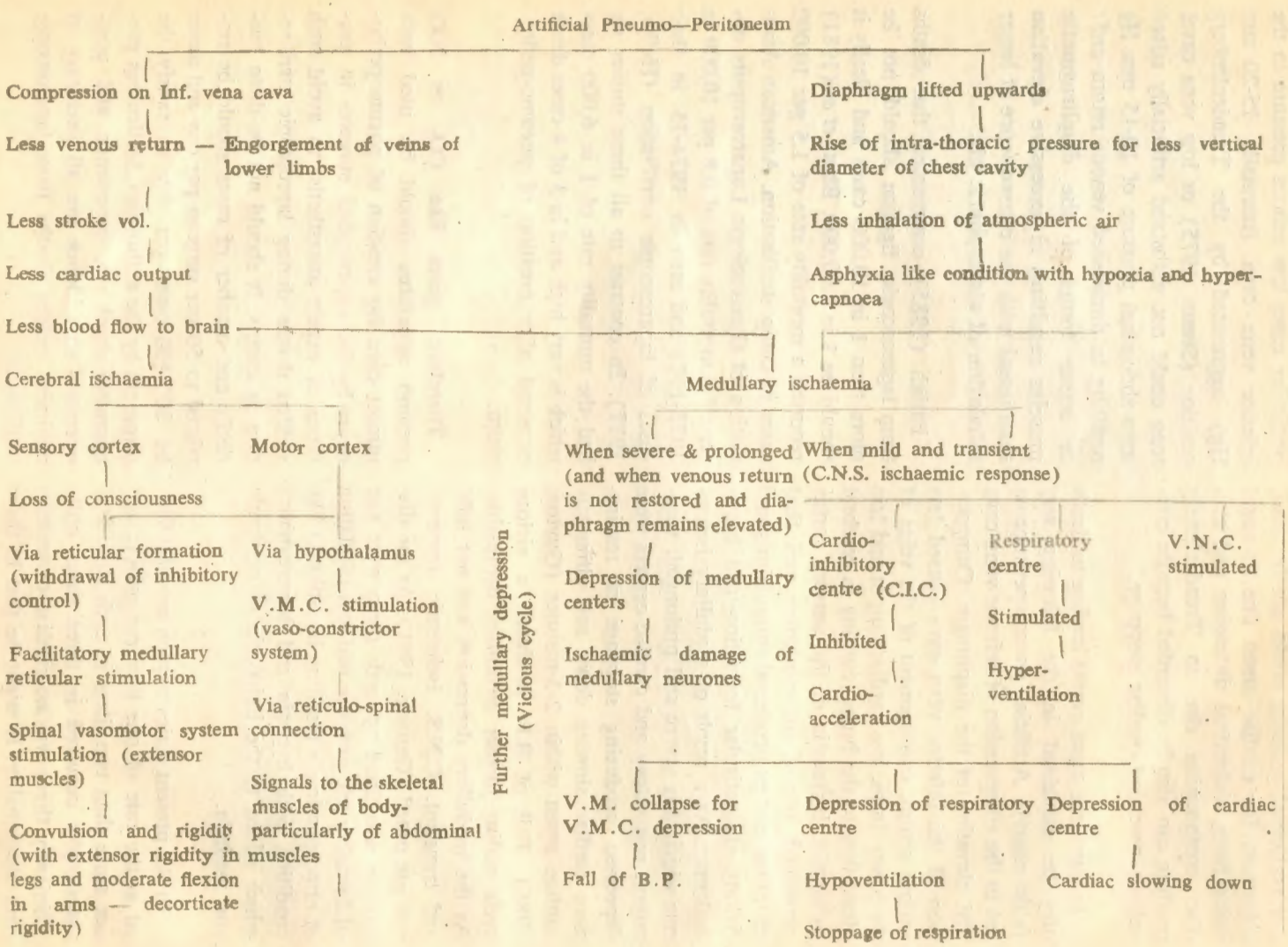
Artificial Pneumo—Peritoneum
Compression on Inf. vena cava
Less venous return — Engorgement of veins of lower limbs
Less stroke vol.
Less cardiac output
Less blood flow to brain
Cerebral ischaemia
Diaphragm lifted upwards
Rise of intra-thoracic pressure for less vertical diameter of chest cavity
Less inhalation of atmospheric air
Asphyxia like condition with hypoxia and hypercapnoea
Medullary ischaemia
Sensory cortex
Loss of consciousness
Via reticular formation (withdrawal of inhibitory control)
Facilitatory medullary reticular stimulation
Spinal vasomotor system stimulation (extensor muscles)
Convulsion and rigidity (with extensor rigidity in legs and moderate flexion in arms — decorticate rigidity)
Motor cortex
Via hypothalamus
V.M.C. stimulation (vaso-constrictor system)
Via reticulo-spinal connection
Signals to the skeletal muscles of body-particularly of abdominal muscles
Further medullary depression (Vicious cycle)
When severe & prolonged (and when venous return is not restored and diaphragm remains elevated)
Depression of medullary centers
Ischaemic damage of medullary neurones
When mild and transient (C.N.S. ischaemic response)
Cardio-inhibitory centre (C.I.C.)
Inhibited
Cardio-acceleration
Respiratory centre
Stimulated
Hyper-ventilation
V.N.C. stimulated
V.M. collapse for V.M.C. depression
Fall of B.P.
Depression of respiratory centre
Hypoventilation
Stoppage of respiration
Depression of cardiac centre
Cardiac slowing down

Chart Showing Physio-pathogenesis of Sudden Deaths Following Pneumo-peritoneum

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