Should we pre-oxygenate?

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Summary

The importance of routine pre-oxygenation before induction of anaesthesia was demonstrated in animals. The onset of dangerous levels of hypoxia after the administration of suxamethonium can be delayed considerably by the administration of pure oxygen for 5 minutes before induction. This allows time for an atraumatic and a panic-free intubation before controlled ventilation.

After pre-oxygenation the volume of oxygen in the functional residual capacity of the lungs shows a sixfold increase. This is the principal oxygen store during apnoea.

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Pre-oxygenation before anaesthetic induction and endotracheal intubation in the poor-risk patient is a routine procedure. The question arises whether all patients should be pre-oxygenated before general anaesthesia. The time required for the arterial oxygen partial pressure (Pa_{O_2}) to achieve dangerous hypoxic levels after the onset of apnoea is an important consideration. This problem was evaluated in the animal laboratory during trials on the characteristics of different anaesthetic circuits.

Method

Three baboons, weighing 9,6 - 10,2 kg each, were immobilized with ketamine and anaesthetized with a pentobarbitone infusion. Four rabbits, weighing 1,6 - 2,1 kg each, were anaesthetized with a pentobarbitone infusion. The animals were intubated after the administration of muscle relaxants.

With the use of a capnograph ventilation was adjusted to maintain a constant end-expired carbon dioxide level. The ECG, intra-arterial pressure and temperature were monitored.

The animals were stabilized on oxygen in nitrous oxide mixtures delivered from calibrated flow meters. Flow rates were adjusted to deliver 21%, 50% and 100% oxygen to the animals. The sequence of the oxygen concentrations was varied, and in some animals the same percentage of oxygen was repeatedly delivered to provide a control. After a stabilizing period of 20 minutes on a specific oxygen percentage, baseline arterial blood samples were collected in heparinized 2 ml syringes for blood gas analysis. The anaesthetic circuit was disconnected and the endotracheal tube left open to air.

In the baboons arterial blood gas sampling was done at 30-second intervals and in the rabbits at 15-second intervals. Between sampling, the cannulas were flushed with a heparin-saline solution.

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Results

Baboons

The initial mean Pa_{O_2} achieved with 100% oxygen was 56,50 kPa (425 mmHg). After 3,5 minutes of apnoea the mean Pa_{O_2} had fallen to 11,30 kPa (85 mmHg) and after 4 minutes to 7,32 kPa (55 mmHg) (Fig. 1).
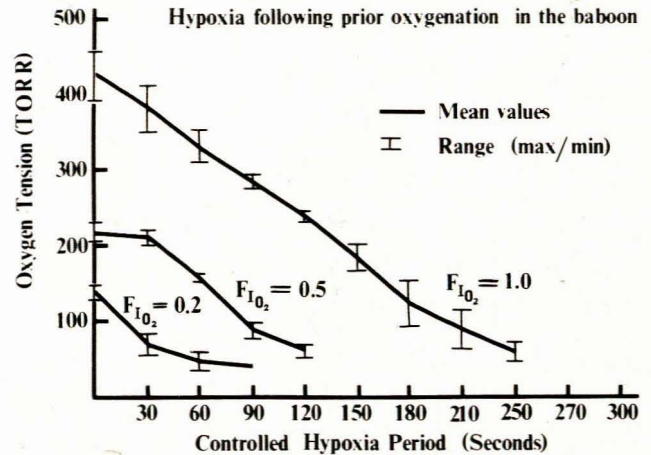

Fig. 1. Relationship of inspired oxygen (Fi_{O_2}), arterial oxygen tension (torr) and controlled hypoxia period (seconds) in the baboon.

On breathing 50% oxygen the initial mean Pa_{O_2} was 28,60 kPa (215 mmHg). After 1,5 minutes of apnoea the mean Pa_{O_2} was 11,30 kPa (85 mmHg) and after 2 minutes 7,98 kPa (60 mmHg). With 21% oxygen the mean Pa_{O_2} fell to 5,32 kPa (40 mmHg) after 1,5 minutes of apnoea.

Rabbits

The initial mean Pa_{O_2} achieved with 100% oxygen was 46,55 kPa (350 mmHg). After 1,25 minutes of apnoea the mean Pa_{O_2} had fallen to 11,97 kPa (90 mmHg) and after 2 minutes to 8,65 kPa (65 mmHg) (Fig. 2).
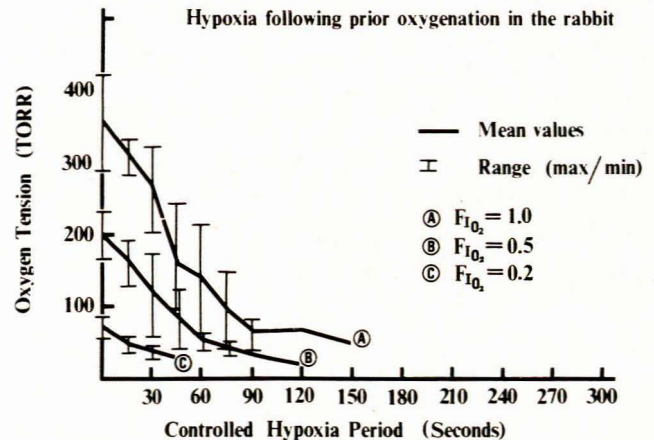

Fig. 2. Relationship of inspired oxygen (Fi_{O_2}), arterial oxygen tension (torr) and controlled hypoxia period (seconds) in the rabbit.

On breathing 50% oxygen the initial mean Pa_{O_2} was 25,93 kPa (195 mmHg). After 0,75 minutes of apnoea the mean Pa_{O_2} had fallen to 10,65 kPa (80 mmHg) and after 2 minutes to 8,64 kPa (65 mmHg).

With 21% oxygen the initial mean Pa_{O_2} fell from 9,31 kPa (70 mmHg) to 3,32 kPa (25 mmHg) after 45 seconds of apnoea.

Discussion

After pre-oxygenation with 100% oxygen a dangerous level of hypoxia developed in the baboons within 4 minutes and in the rabbits within 2 minutes. When they breathed 21% oxygen this length of time was reduced by 2,5 minutes (more than 50%) in the baboons and by 1,75 minutes in the rabbits.

The relevant importance of these figures to the anaesthetist can be found in the fact that the oxygen stores of the body are small.

The following figures are given by Nunn[1] for the adult man while he breathes room air (Table I).

The minimum basal oxygen requirements are 250 ml per minute. Oxygen stores will therefore be rapidly depleted during apnoea. The picture is further complicated by the fact that myoglobin will release its oxygen only when the Pa_{O_2} has fallen to 2,70 kPa (20 mmHg); because of the configuration of the haemoglobin-oxygen dissociation curve, sufficient oxygen cannot be released from the haemoglobin before the Pa_{O_2} decreases to dangerously low hypoxic levels. The functional residual capacity of the lung thus remains as the main oxygen store. When the subject breathes room air this volume is small (450 ml). The oxygen level in the blood is therefore rapidly depleted within a short period of apnoea, as can be seen from the Pa_{O_2} levels in the animal models.

TABLE I. BODY OXYGEN RESERVES (ml) IN MAN ON BREATHING ROOM AIR AND OXYGEN

	Air (ml)	100% oxygen (ml)
Lungs (FRC)	450	3 000
Blood	850	950
Myoglobin	200	200
Tissue fluid	50	100
Total	1 550	4 250

FRC = functional residual capacity.

The administration of pure oxygen will give a sixfold increase in the oxygen stores of the lung. Pre-oxygenation for at least 5 minutes before induction of anaesthesia should allow a safe period of at least 4 minutes before hypoxia sets in. Unless a crash induction is indicated, this period allows for a calm and atraumatic endotracheal intubation before institution of controlled ventilation.

It is obvious that pre-oxygenation is mandatory in children, who have even smaller oxygen stores and a higher oxygen consumption. The problem of patient acceptance of a mask before intravenous induction can be overcome by explanation during the pre-operative visit. The cost of oxygen should not be a deterrent to pre-oxygenation. It is a small price to pay for an ultimate goal, viz. better patient care and safety.

REFERENCE

1. Nunn, J. F. (1978): *Applied Respiratory Physiology*, 2nd ed., p. 412. London: Butterworths.

Faulty inflation and deflation of urethral balloon catheters

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Summary

Attention is drawn to the faults encountered with urethral balloon catheters, with particular reference to difficulties with inflation and deflation of the balloon. Needle puncture has been found to be a simple method of removing catheters that cannot be deflated by the usual methods.

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During a recent 9-month period, 9 faulty urethral balloon catheters were encountered. Some could not be inflated and others could not be deflated. In some cases it was almost impossible to introduce the front of a syringe into the inflation channel, although these catheters have been constructed to accept a syringe without a hypodermic needle. The catheters were responsible for patient discomfort and unnecessary expense.

Cases

Case 1. A No. 16 5 ml balloon catheter (Fig. 1) was inflated with 5 ml water. The catheter fell out of the bladder as the patient was moved off the operating table. A transverse split was noted in the balloon.

Case 2. A No. 22 30 ml catheter (Fig. 1) was used after a prostatectomy and positioned with the balloon in the prostatic