TREATMENT OF EXPERIMENTALLY INDUCED BRONCHOSPASM BY AIR-AND-OXYGEN MASSAGE OF THE LUNGS

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Total bronchospasm sometimes occurs during thoracic operations and is a complication which anaesthetists dread. It is also seen in bronchial asthma and as a result of poisoning by certain chemical agents (Ariev, 1952; Buyanov, Riabov, and Smirenskaya, 1959). There is a dearth of literature on bronchospasm and effective methods of its treatment. As a rule it is treated by anticholinergic and sympathomimetic agents, but these drugs are often either of little use or totally ineffective.

At the Institute of Thoracic Surgery, Moscow, total bronchospasm that had developed in a patient suffering from mitral stenosis was effectively treated by direct massage of the lung. This confirmed the hypothesis advanced by Smolnikoff that total bronchospasm may be regarded as a form of pulmonary arrest which is similar to cardiac arrest and which therefore can be treated by direct massage. We decided, however, to look for less traumatic means of massaging the lungs.

In the medical literature at our disposal we were unable to find any reference to lung massage for bronchospasm, and so we designed a very simple apparatus (Figs. 1 and 2) with which several cases of experimentally induced bronchospasm in dogs have been successfully treated.

FIG. 1.—Diagram of the lung-massage machine. (1) Air pump. (2) Metal three-way cock. (3) Inspiratory tubes. (4) Expiratory tubes. (5) Endotracheal tube. (6) Curved cannulas. (7) Body of the three-way cock. (8) Three-way cock piston.

connects the bellows to the three-way cock, and two other tubes lead away from it. One of them is connected to the endotracheal tube, the other to two tubes each bearing a curved cannula. These serve to "intubate" the pleural cavities. This apparatus creates passive inspiration and expiration. When the three-way cock connects tube "a" with tube "b" the pumped air is

directed into the lungs. Air which is found in the pleural cavities will be displaced by the expanding lungs and will be forced outside, through the curved cannulas, into the tubes (6). When tube "a" is connected with tube "c" the pumped air will be directed into the pleural cavities. This results in collapse of the lungs, and the air is displaced from the lungs outwards via tube "b."

Thus by alternating the position of the valve it is possible to obtain passive inspiration and expiration.

FIG. 2.—Transverse section of the three-way cock. Position I: (7) body; (8) piston. Channel a-b: position of the piston during inspiration. Position II: piston's position during expiration, when the air is pumped into the pleural cavities.

Method

Experiments were made on nine dogs weighing between 15 and 35 kg. Premedication, which was given to the animal 30 to 40 minutes prior to the experiment, consisted of a subcutaneous injection of a 1% solution of "omnopon" (3 mg./kg. body weight). The dog was then placed on the operating table and its right or left femoral artery and vein were denuded. The artery was cannulated for continuous recording of arterial pressure. Initial data such as respiration rate, pulse, arterial pressure, electrocardiogram, and electroencephalogram were then recorded.

The induction with an 0.8% solution of thialbarbitone ("khemital") was followed by intubation. Spontaneous breathing was preserved. An intratracheal spiogram was recorded (see (a) in Fig. 3). The skin was then painted at the sides of the chest wall in the seventh interspace on the right and in the eighth interspace on the left (owing to the proximity of the heart), along the axillary line. An incision of 0.5 cm. was made and curved cannulas (diameter 3-4 mm., length 5 cm.) were inserted.

Intrapleural spiograms and variations of intrapleural pressure were continuously recorded (see (b) in Fig. 3). Then 0.05% neostigmine, 0.3-0.6 mg./kg. of body weight, was injected (average 9-18 mg.). Bronchograms were taken at the height of the bronchospasm, and in some cases pieces of lung tissue were taken for biopsy. In three experiments the usual doses of neostigmine were found inadequate to produce bronchospasm and an extra 3 to 13 mg. was added.

After two to five minutes of bronchospasm air-and-oxygen massage of the lungs was started. A bronchogram was taken and was repeated 5 to 10 minutes after the removal of the bronchospasm. As spontaneous respiration returned, the respiratory rate and pulse were recorded and pieces of lung tissue were again taken for histological examination.

In order to register all the above data we connected the endotracheal tube and the intrapleural cannulas with a Marey capsule and a mercury manometer.

Results of the Experiments

We carried out 11 experiments on nine dogs. Our data show that with an initial respiratory rate of 11–13 a minute, an intratracheal pressure of 3–4 mm. Hg, and an intrapleural pressure of 7–8 mm. Hg neostigmine-induced bronchospasm developed at variable intervals of time from 30 seconds to 4 minutes (Fig. 3, I).

FIG. 3.—Spirograms of spontaneous breathing and of the beginning of the bronchospasm. Kymograms were recorded in the following order from above below: (a) record of respiration; (b) record of intrapleural pressure. (I) Spontaneous respiration, the beginning of bronchospasm, and the respiratory arrest. (II) Air-oxygen massage of the lungs four minutes after respiratory arrest. (III) Air-oxygen massage of the lungs 42 minutes after respiratory arrest. (IV) Resumption of spontaneous respiration after 57 minutes.

Respiration stopped entirely and the intratracheal spirograms became a straight line. There were single vibrations and changes of pressure which were probably due to fibrillation of the intercostal muscles, the diaphragm, etc. They sometimes lasted three to four minutes. When they appeared the intrapleural pressure rose sharply (up to 40–50 mm. Hg) and vibration frequency reached 17–19 a minute. Similar changes, which apparently had the same aetiology, were recorded in the trachea. Later on the intratracheal pressure did not change from the straight line (Fig. 3, II). The number of vibrations inside the pleural cavities perceptibly decreased—up to 6–11 a minute—and the pressure fell to 30–35 mm. Hg.

We started lung massage two to five minutes after the development of the bronchospasm. With the aid of the Oxford bellows we produced 7–10 passive inspirations and expirations. Intratracheal pressure varied between 30 and 32 mm. Hg and the intrapleural pressure between 40 and 45 mm. Hg (Fig. 3, II). Some 8 to 10 minutes after the massage had been started we

noticed that the pressures inside the trachea and the pleural cavities were equal. This equalization of pressures may have been due to decrease of resistance to expiration—that is, to partial decrease of the bronchospasm.

In some instances we experimented with double or triple inspirations followed immediately by double or triple expirations (Fig. 3, III). This manoeuvre was accompanied as a rule by a rise of the intratracheal pressure. Towards the end of the massage both the intrapleural and the intratracheal pressures decreased to 10 mm. Hg.

Spirograms of spontaneous respiration after lung massage showed that pressure in the pleural cavities exceeded that in the trachea. The maximum for intrapleural pressure was 20–35 mm. Hg and that for intratracheal pressure 15–18 mm. Hg. These results confirmed the data obtained with the aid of bronchography. Bronchograms were taken at the height of the bronchospasm, and also 5 and 10 minutes after the massage had begun. To take a bronchogram the head of the table was raised to an angle of 50 degrees. The opaque dye was injected into a No. 12 endotracheal tube with the aid of Janet's syringe (30–40 ml. of a 40% solution of lipiodol).

Bronchograms taken at the height of the bronchospasm (Fig. 4, A) indicated that the opaque dye filled the trachea, the right and the left bronchus, and their major ramifications—the segmental bronchi. The smaller bronchi and the alveoli usually remained free from the dye. Five minutes after the air massage of the lungs (Fig. 4, B) the penetration of the dye into the bronchioles and the alveoli was clearly visible. Some lipiodol remained in the larger bronchi. Ten minutes after massage (Fig. 4, C) the dye was in the alveoli. After about 40–64 minutes of massage, when the bronchospasm had been removed and spontaneous respiration established, the structure of the bronchial tree assumed the same appearance as in the initial bronchogram. If no massage was done the lumen of the bronchi was progressively reduced until death occurred.

Data on Additional Investigations

We have seen that with bronchospasm at its height the arterial blood-pressure rose from 130–140 to 170–180 mm. Hg. On starting massage of the lung the arterial blood-pressure fell rapidly and reached 60–70 mm. Hg. Then as the massage proceeded the blood-pressure rose gradually and had returned to its initial level by the time spontaneous breathing restarted.

After the injection of neostigmine the pulse slowed down from 120–130 to 70–75 a minute. With bronchospasm at its height a bradycardia of 40–45 a minute developed. Air massage of the lung increased the pulse rate to 90–100 a minute. This rate was maintained with the return of spontaneous respiration.

Some 40–60 seconds after the injection of neostigmine we observed nodal rhythm on the E.C.G. in the majority of instances. At the height of the bronchospasm this rhythm was transformed into a complete atrioventricular block. The air massage of the lung led to the re-establishment of sinus rhythm after 10–15 minutes in two cases, and after 20–25 minutes in five cases (E.C.G.s were not taken in three cases). When spontaneous respiration returned the E.C.G. resumed its initial configuration.

Investigation of the electrical activity of the brain showed that 10–19 seconds after the injection of neostigmine slow waves appeared with a certain increase of amplitude in the sensory and motor areas. With return of spontaneous respiration the E.E.G. reverted to normal.

The presence of the bronchospasm in our experiments was further confirmed by histological study of lung tissue. Lung biopsy taken at the height of the bronchospasm showed marked narrowing of the lumen of the large and the smaller bronchi, which had a festooned appearance with slit-like lumen, and contained mucus with leucocytes and epithelial cells. Atelectatic areas alternated with areas of gross dilatation (Fig. 5).

In one case we left the animals without the benefit of lung massage in order to study the clinical picture of bronchospasm in its natural state and the death of an animal which had received a lethal dose of neostigmine.

The presence of bronchospasm was confirmed by the histological examination of biopsy material taken at the height of the attack. In lung tissue examined one hour after clinical death there was no histological evidence of bronchospasm (Fig. 6). The bronchial lumen was wide open and the absence of the spasm in this case was probably explained by the development of post-mortem relaxation.

Summary

Total bronchospasm in anaesthetized dogs was induced with adequate doses of neostigmine.

With the aid of our apparatus for air-and-oxygen massage of the lungs it was possible to remove the bronchospasm and re-establish spontaneous breathing without resorting to thoracotomy.

All changes in the cardiovascular system and in the electroencephalographic pattern caused by the bronchospasm were gradually modified by massage of the lungs.

FIG. 4.—Removal of neostigmine-induced bronchospasm by air massage. A, neostigmine bronchospasm. Opaque dye injected at height of bronchospasm fills only the trachea and main bronchi. B, bronchogram five minutes after air massage of the lungs. The dye has penetrated into the smaller bronchi and the alveoli. C, bronchogram 10 minutes after air massage of the lungs. The bronchospasm has been removed; the dye is in the alveoli.

FIG. 5.—Photomicrograph of lung tissue. The smaller bronchi have a festooned appearance with slit-like lumen, filled with mucus.

FIG. 6.—Photomicrograph of lung tissue. Bronchi after death. There is no evidence of spasm. The bronchial lumen is wide open.

These changes reverted to normal as spontaneous respiration was re-established.

The method of lung massage suggested is less traumatic and more effective than the direct method suggested by Smolnikoff. When the aetiology of the bronchospasm is unknown or when the spasmolytic drugs are ineffective lung massage by our method may prove to be a life-saving measure.

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REVERSIBLE ACUTE RENAL TUBULAR NECROSIS DUE TO ANTI-Le^a

SUCCESSFUL TREATMENT BY HAEMODIALYSIS

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Since the discovery of the Lewis blood-group system by Mourant in 1946 there have been at least 10 reported instances of transfusion reactions due to anti-Le^a. According to Mollison and Cutbush (1955) these may be mild. We report one case which resulted in acute tubular necrosis of the kidneys, the uraemic and comatose patient recovering after haemodialysis on a Kolff twin-coil artificial kidney. We discuss the serology and emphasize again the dangers of using unmatched blood in transfusions.

Case Report

A married woman aged 20 reported transient intermenstrual vaginal bleeding one week before she was admitted to hospital complaining of pain in the lower

abdomen with vomiting and further minimal vaginal bleeding. Physical examination showed no signs of peritoneal irritation, her blood-pressure and pulse were quite normal, and a diagnosis of threatened abortion was made.

Next day she collapsed quite suddenly with severe abdominal pain; a laparotomy was performed and a ruptured right tubal ectopic pregnancy excised. In this episode her systolic blood-pressure remained at a level of 70 mm. Hg for a period of three hours. She was given 5 pints (2.8 litres) of group O Rh-negative blood which was not cross-matched. After transfusion her systolic blood-pressure rose rapidly to 120 and was maintained. Her general condition was good, and some 400 ml. of urine was removed by catheter. However, on the following day she produced only 180 ml. of urine and on the next day only 60 ml., which was noticeably dark in colour. This degree of oliguria persisted for a further four days. Her only intake was 10% glucose in water by mouth, with 250 mg. of tetracycline q.i.d., on a fluid-balance regime. During the final four days her blood N.P.N. rose from 156 to 238 mg./100 ml. Latterly, persistent vomiting occurred, and she was transferred to our hospital.

On admission seven days post-operatively she was semi-comatose, and was vomiting and screaming irrationally and intermittently. On examination she had facial oedema, generalized twitching, and a blood-pressure of 120/80, and was afebrile; the abdomen showed no special features. The serum ionogram showed serum sodium of 134.5 mEq/l., potassium 6.3 mEq/l., bicarbonate 17.9 mEq/l., and chloride 79.6 mEq/l.; this inferred an abnormally high degree of retention of sulphates, phosphates, or similar anions. Haemoglobin was 12 g./100 ml., and the blood counts showed nothing of note. The blood N.P.N. was 229 mg./100 ml. and creatinine 19 mg./100 ml.; the serum bilirubin was not elevated.

Catheterization provided too small an amount of cloudy yellow acid urine for specific-gravity determination; occasional polymorphs and red cells were present microscopically, and the urine urea concentration was only 800 mg./100 ml.

The clinical impression was of acute renal tubular necrosis, secondary to hypotension at operation or to a haemolytic transfusion reaction. It was felt that the degree of hypotension was possibly not adequate to explain the renal shut-down, and a serological review of blood samples and previous donors was requested.

The patient's coma deepened and she became practically unresponsive to pain stimuli. She was therefore dialysed on a Kolff twin-coil artificial kidney for five hours at an

FIG. 1.—Anti-Le^a transfusion reaction with acute renal failure.