TOTAL BRONCHOSPASM

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Partial bronchospasm is a fairly common complication of general anaesthesia and is usually not difficult to control. Total bronchospasm, which is fortunately rare, is a very alarming complication indeed. Not only bronchial asthma, but any chronic pulmonary disease predisposes towards it. Thiobarbiturates have been known to cause it, and neostigmine is used to produce it experimentally. The difference between bronchospasm as seen in asthma and total bronchospasm as seen under anaesthesia is that as the latter is total no sounds can be heard from the lungs at all.

CASE REPORT

This report concerns a well developed boy aged 14 years and weighing 130 lb. He was an asthmatic, and attacks of asthma always followed the eating of bananas. He was also considered to be sensitive to house dust.

He was to undergo tonsillectomy, and on examination the day before operation the pharynx was found to be slightly congested, but the temperature and pulse were normal and auscultation of the chest revealed no abnormality. That evening he was given two 'sonergan' tablets and slept well. Next morning he complained of slight nasal obstruction, and on examination by the ENT surgeon this was considered to be due to allergic rhinitis. The lungs were clear and there was no respiratory difficulty.

Premedication consisted of pethedine (100 mg.) and atropine (gr. 1/100) given 1 hour before the operation.

Anaesthesia was induced with 350 mg. of thiopentone sodium, and before the injection was completed the patient began coughing very violently. Suxamethonium chloride, 50 mg., was given to facilitate intubation, and the coughing ceased.

A mask was applied over an oropharyngeal airway and an attempt made to inflate the lungs with oxygen in the usual way. This was found to be impossible and the immediate assumption was that the relaxant had failed to act and that laryngeal spasm had occurred. On laryngoscopy, however, the cords were found to be wide open and intubation was performed with ease. It was now found to be impossible to inflate the lungs through the tube (which had been tested for patency) and a diagnosis of bronchospasm was made. The hardest compression of the reservoir bag failed to force any oxygen into the lungs, and the chest remained absolutely immobile and silent.

An attempt was now made to force air out of the lungs in the hope that this might break the spasm. To this end, manual compression of the chest was attempted, but in spite of the use of quite considerable pressure no air could be forced out of the lungs.

Bearing in mind the reports of lung massage by Smolnikoff[1] and Mostert,[2] I had obtained a scalpel as soon as the diagnosis had been made, but in spite of the most intense cyanosis the heart beat could be clearly seen and felt, and because there was still a circulation it was decided to attempt intravenous therapy before resorting to thoracotomy. I rapidly gave 250 mg. of aminophylline intravenously, and within 2 minutes it had become possible to force a little oxygen into the lungs. Halothane (0·7%) was immediately added in an attempt to increase the bronchodilatation, and inspiratory rhonchi were heard throughout both lung fields on compression of the bag. No expiratory sounds could be heard, and at first it was feared that the lungs were merely being inflated and that lung massage might still be necessary to remove the carbon dioxide. However, the cyanosis, which by this time was extreme, now began to improve, and 3 minutes later spontaneous respiration returned and the patient 'pinked-up' rapidly.

The halothane was now discontinued and normal respiration continued on oxygen only for about 3 minutes more. At

this stage respiration suddenly stopped; once again it was found impossible to inflate the lungs, and on auscultation no lung sounds could be heard. Cyanosis supervened rapidly, and 250 mg. of aminophylline, followed by 100 mg. of hydrocortisone, was given intravenously. As before, within 2 minutes it became possible to force a little oxygen into the lungs and spontaneous respiration again returned within 3-4 minutes. Inspiratory and expiratory rhonchi could be heard throughout both lung fields but there was no cyanosis.

Shortly afterwards marked sternal retraction developed on inspiration, and the trachea was aspirated through a catheter passed down the endotracheal tube. A considerable amount of mucus was removed and respiration improved.

It had been decided not to perform the proposed operation, but at this stage I was loth to remove the endotracheal tube. Besides a very small amount of halothane, the patient had only received 350 mg. of thiopentone sodium and, even accepting the fact that the hypercapnia must have caused some unconsciousness, it was obvious that within a short time the tube would cause irritation, with a possible further bout of bronchospasm, and laryngeal spasm as well if it were removed at that stage. It was therefore decided to deepen the anaesthesia and try to increase the bronchodilatation. Ether is probably the most potent bronchodilator but, in view of the copious mucus already removed, it was felt that halothane, with its drying action, would be better. Accordingly, for the next 45 minutes 0·7% halothane was given, with 5 litres of oxygen and 3 litres of nitrous oxide per minute. On 3 occasions during this time sternal retraction occurred, and each time mucus was aspirated from the trachea.

Sternal retraction had now ceased and respiration was normal. Rhonchi could still be heard but were less marked.

Pharyngeal toilet was followed by extubation, and oxygen was administered by a mask. This was discontinued after 5 minutes and the respiration and colour remained satisfactory.

Intramuscular hydrocortisone was given (100 mg.) and the patient was returned to the ward. Within half an hour he had recovered consciousness and was apparently none the worse for his ordeal. On examination 6 hours later, air entry was equal and normal throughout both lung fields and no abnormal sounds could be heard.

DISCUSSION

Diagnosis

The diagnosis of bronchospasm depends to a considerable extent upon the time at which it occurs.

A. *Immediately after induction* respiratory obstruction may result from the following conditions:

1. Laryngeal spasm, which is thought to be due to the fact that thiopentone depresses the parasympathetic nervous system less than the sympathetic, and thus leads to central vagal stimulation. It may also be due to stimulation of the vagal nerve endings in the larynx by mucus, blood, or regurgitated gastric contents, etc., i.e. peripheral vagal stimulation.[3]

2. Bronchospasm, which may be due to the same causes, or to histamine release. It is claimed that d-tubocurarine can cause bronchospasm in this way, but the claim is based on experimental work in guinea-pigs and has never been proved in man.[4] Histamine stimulates the bronchi directly and is not therefore blocked by atropine. In normal human beings this action is negligible, but patients with bronchial asthma, bronchitis, emphysema, and even cardiac asthma, are much more sensitive to it.[5]

3. An attack sustained by a true asthmatic immediately after induction may be the result of any of the above factors but it may also be caused by fear, excitement, or whatever provokes his normal attacks. Inspiration and expiration are both impeded, but the latter more so, and therefore there is an increase of residual air.

4. Inhalation of some foreign material that completely blocks the trachea.

5. Obstruction by the tongue, cheeks, etc., in a case where an oropharyngeal airway has not been used.

B. *During anaesthesia* respiratory obstruction may result from any of the following conditions:

1. Inadequate depth of anaesthesia. The mechanism here is sustained contraction of thoracic and abdominal muscles. This form of respiratory embarrassment is most likely to occur in the plethoric, bull-necked, emphysematous patient. The picture is either sudden cessation of respiration, or forceful prolonged expiratory efforts accompanied by wheezing and 'bucking'. In either case it is difficult or even impossible to inflate the lungs, and cyanosis follows rapidly. Because the condition is due to contraction of voluntary muscles, muscle relaxants are rapidly effective.[6] It is probable that it is this type of case that has given muscle relaxants the reputation of being efficacious in the treatment of bronchospasm.

2. Obstructed endotracheal tube. This may be caused by kinking, foreign material in the tube, impingement of the end of the tube on the carina, a faulty cuff covering the end of the tube, etc.

3. Bronchospasm, which may be caused by the factors mentioned under A but also by 2 other causes, viz.: (*a*) Mechanical stimulation, due to direct irritation of the respiratory tract, e.g. by the tube, by irritant vapours, etc. (*b*) Neurogenic stimulation. Here a great number of different reflex pathways exist, with afferents arising in different parts of the body, such as the skin, viscera, etc. The commonest cause during anaesthesia is naturally surgical stimulation. In every case the efferent pathway is the vagus nerve. The stimulation may be central and not peripheral; cyclopropane, in common with thiopentone, has this effect.[4]

Prophylactic Treatment

1. Promethazine has long been regarded as a useful drug in the pre-operative preparation of asthmatic and bronchitic subjects. There is some evidence that it has a mild stimulant action on respiration, both minute volume and rate being increased. Bronchial musculature is relaxed and there is an inhibition of secretions.[7] In the case here reported this drug was used in the form of 'sonergan' the night before operation. I have since used it in combination with pethidine ('pamergan') as routine premedication for all such patients. Intramuscular aminophylline (500 mg.) can be added to this, but it is a painful injection and many patients complain about it; alternatively it may be given in suppository form, but again this does not always meet with the patient's approval.

2. Another medication that I have found useful is 'expansyl spansule'*, which contains ephedrine, a tranquillizer and an antihistaminic. It acts for 12 hours and can therefore be given orally long before the operation, thus avoiding discomfort to the patient.

3. If time permits, the bronchitic patient can be im-

*I wish to thank SKF Laboratories (Pty.) Ltd. for the supply of expansyl spansules.

proved by postural drainage, physiotherapy and perhaps a course of antibiotics, and by reduction of smoking.

4. Thiopentone should be used with caution.[8]

Treatment of the Spasm

A. *Lung massage,* Smolnikoff[1] first drew attention to lung massage, describing 3 cases, 2 of which occurred during thiopentone induction. Thoracotomy was performed, and in all three the lungs were found to be grossly distended. Cardiac massage proved fruitless and the first two patients succumbed. In the third case intravenous ephedrine and atropine had no effect but, when a combination of heart and lung massage was resorted to, normal respiration and heart beat returned within a few minutes and the patient survived. He points out that although in some cases oxygen can be forced into the lungs under considerable pressure, the elastic recoil is not sufficiently strong to force the CO_2-laden air through the spastic bronchi, and therefore hypercapnia results. This causes myocardial failure followed by hypoxia and cardiac arrest. He feels therefore that lung massage must precede cardiac massage. He suggests that total bronchospasm may be regarded as a form of pulmonary arrest, similar to cardiac arrest, and therefore to be treated in the same way. He points out that no one spasmolytic agent is effective in every case and that time is much too precious to waste in trying one drug after another. Mostert,[2] too, points out that no surely efficacious bronchodilator drug exists and, moreover, that the venous return to the heart must be seriously curtailed as a result of the excessive inflation of the lungs.

B. *Drugs* may be used to produce the following effects:

1. Bronchodilatation, through a direct relaxing effect on the bronchial muscle. Theophylline is the most effective (aminophylline BP is a mixture of theophylline and ethylenediamine).

2. Bronchodilatation, by potentiation of the effects of the adrenaline that is liberated normally at the sympathetic nerve endings. Adrenaline, ephedrine and ether act in this way.

3. Vagal inhibition, resulting from unopposed sympathetic action. Atropine acts in this way.

4. Antihistaminic action, which relieves bronchoconstriction caused by histamine.

5. Steroid therapy. Acute attacks of asthma have occurred during bronchoscopy, in which the mucosa was seen to become congested and to swell up over the end of the instrument and almost occlude the lumen.[9] It has been suggested that this mucosal swelling is the main obstructive element in asthma and that its reduction on steroid therapy is the reason for the relief of symptoms. Apparently the cause of the reaction makes no difference, for, given a large enough dose, response will usually occur in all types.[10]

CONCLUSION

Total bronchospasm is one of the most alarming complications of general anaesthesia. Being so rare and coming, as it does, so completely 'out of the blue', it tends to catch the anaesthetist on the wrong foot. Cyanosis supervenes extremely rapidly and one's natural inclination is to pump oxygen into the lungs at any cost. This is wasted effort and—much more important—it is wasted time. Every second is of paramount importance, and even if some oxygen is forced into the lungs it will at best only relieve the cyanosis temporarily. It will still be impossible to get the carbon dioxide out of the lungs and cardiac arrest will follow very rapidly.

It is impossible to make hard and fast rules for a condition as rare as bronchospasm, but from my experience in this case I feel that if hydrocortisone and aminophylline are given intravenously immediately the diagnosis is made there will still be time to perform lung massage if necessary.

The survival of our patient was probably due to 2 factors, viz. (*a*) that he was young and fit, and able temporarily to withstand the insult of practically complete deprivation of oxygen and accumulation of carbon dioxide, and (*b*) that the diagnosis and subsequent injections were made fairly quickly.

SUMMARY

A case is described of total bronchospasm occurring under general anaesthesia.

The differential diagnosis is discussed and the vital importance of speed, not only in making the diagnosis, but also in acting upon it, is emphasized.

Some suggestions are put forward for minimizing the chances of the condition occurring.

The rival merits of lung massage or intravenous drugs in the treatment of the bronchospasm are discussed.

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