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Reflex bradycardia: a grave complication of oesophageal atresia repair

by

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Among 39 infants operated upon for oesophageal atresia in newborn age, 5 presented attacks of reflex bradycardia during meals. One patient died at the age of one and a half years. Four patients became symptom-free on atropine treatment. They are healthy and develop satisfactorily.

Since the first successful operation of oesophageal atresia in 1943 [4] a number of papers has been published on the complications of the procedure.

There are few reports on functional disturbances including reflex bradycardia and most of these discuss adult cases [2, 3, 5, 7, 8, 9]. The term reflex bradycardia or vago-vagal reflex means a slowing down of the heart rate in consequence of some kind of irritation of the vagus nerve. It mostly occurs if a space-reducing process exerts pressure on one or both vagal nerves. In most cases a sino-auricular block is seen in the ECG. The process may deteriorate into transitory asystole or even cardiac arrest. Bauer et al. in 1959 [1] were the first to report on the condition in two neonates. One had an oesophageal atresia, and the great distance had been bridged over by oesophago-gastrostomy. The intrathoracic part of the dilated stomach had compressed the vagus, and this elicited frequent bradycardiac apnoeic attacks. Small doses of atropine were given in the last three days, but this treatment was insufficient and the infant died with apnoea and bradycardia on the 34th day of his life.

The other neonate had been born with myxoedema; the attacks had stopped after the administration of atropine, and the baby made a smooth recovery under substitution therapy.

A further child reported by Kenigsberg et al. [6] had been operated upon for oesophageal atresia and then developed well until the age of 2 years. Subsequently, after swallowing coarse food he had become cyanosed and fainted. At 4 years of age the ECG had first shown the disappearance of P-waves, and then a gradual slowing down of ventricular activity, from 120/min to 60/min. A radiographic study carried out simultaneously revealed that the bradycardia set in when the barium meal had reached the site of anastomosis. The oesophagus displayed incoordinated contrac-

tions but no stenosis. Although the attacks could successfully be relieved with atropine, the child was operated upon and the right vagal nerve was found to have been compressed by the anastomosis. The trunk of the vagal nerve was resected from the recurrent nerve down to the anastomosis, and since then the child is symptom-free.

We have observed 5 cases of reflex bradycardia among 39 newborn infants operated upon for oesophageal atresia; 21 of these infants are alive and well.

Case Reports

Case 1. M. L., a male baby born from an uncomplicated pregnancy with 3.400 g body weight was admitted at the age of one day with oesophageal atresia and a tracheo-oesophageal fistula. Thoracotomy, transection of the fistula and primary anastomosis were carried out. After an uncomplicated postoperative period he was discharged. At the age of 3 months the baby after a meal developed bradycardia with deep cyanosis and had to be resuscitated. Thereafter his condition seemed to be normal but for a low serum Ca-level and a negative Ca equilibrium. To control this, chronic administration of large doses of Ca and vitamin D and dihydrotachysterol was necessary. Under such treatment he had repeatedly attacks after meals, although in milder form. Then, at the age of one year, the baby after having had thick food for dinner had a severe attack and died from massive aspiration filling the trachea and the main bronchi. No other changes were found at necropsy.

Case 2. F. N., a male baby born after uncomplicated pregnancy with 2,950 g weight, was operated upon at the age of two days for oesophageal atresia with an inferior oesophageal fistula. After thoracotomy the fistula was dissected and a primary anastomosis was created. The postoperative tracheo-bronchitis and pneumonia recovered after antibiotic treatment. Subsequently, systemic oesophageal dilatations were carried out. He was discharged with 500 g weight gain at one month of age. At the age of three months the patient had to be readmitted, since after meals apnoeic episodes had appeared and once he had to be resuscitated in a regional hospital. The performed examinations excluded a stricture of the oesophagus or recurrence of the fistula. Apnoeic episodes associated with bradycardia appeared also in our hospital. They were immediately relieved on atropine injections. Homatropine methylbromide with papaverine was prescribed for home treatment. Under this treatment the child has no attacks and thrives satisfactorily.

Case 3. N. G. born with 3,000 g was admitted on the first day of life and operated upon for oesophageal atresia with an inferior oesophago-tracheal fistula. After dissection of the fistula a primary anastomosis was carried out. After uncomplicated recovery, at the age of six weeks he produced an apnoeic episode with cyanosis and bradycardia and had to be resuscitated. During the next attack atropine was injected. This ensured complete relief. Under continuous oral homatropine therapy no more attacks were observed. At the age of four months the infant was discharged and the therapy was continued at home. When he reported for oesophageal dilatations the infant had seemed to develop well somatically and mentally and had no attacks.

Case 4. N. Z., a female baby born in the 36th week of a first pregnancy with 1,950 g weight, had been admitted at 7 hours of age for oesophageal atresia with an inferior oesophago-tracheal fistula. After the correction of acidosis, tracheal suction, etc., the fistula was ligated and an end-to-side oesophago-oesophagostomy was carried out according to Beardmore. The postoperative period was uneventful and regular oesophageal dilatations were begun at the age of four weeks. At 4 months of age she de-

veloped apnoeic attacks with bradycardia. The attacks stopped on atropine administration, but she had aspirated several times and the repated aspiration pneumonias had raised the possibility of a recurrent oesophago-tracheal fistula in the hypotrophic infant. (The fistula had not been excised.) At the age of six and a half months, recanalisation of the fistula was demonstrated with methylene blue. It was then closed with Histoacryl-N-blue adhesive via the bronchoscope. Subsequently, the baby started to develop until a month later the injected adhesive material had fallen into the bronchus of the right inferior lobe. The adhesive was removed through the bronchoscope and at 8 months of age a new thoracotomy was done, the patent fistula was ligated and transected. The postoperative period was uneventful and oesophageal dilatations were continued regularly. Under homatropine treatment she had no attacks and her mental and somatic development was satisfactory. At the age of one year she contracted a cold and vomited the drug repeatedly so that she had to be readmitted. During the night she had apnoea and bradycardia and had to be resuscitated. After the successful introduction of homatropine she has no more attacks.

Case 5. Ny. C., a 14 months old female infant, who had been operated upon for oesophageal atresia and tracheo-oesophageal fistula after birth, had been admitted from another hospital with the suspicion of recurrent fistula. In the history repeated aspirations and pneumonia were mentioned. Since several months she had attacks with apnoea, cyanosis, bradycardia and had to be resuscitated several times. On admission a ventricular septal defect was detected. The wasted, hypotrophic infant had to be fed by tube. Swallowing and methylene-blue tests showed no recanalization of the fistula, there were only a blind pouch and mild stenosis at the site of its orifice. After several dilatations the gastric tube could be removed and the infant fed orally. The apnoeic attacks associated with bradycardia manifested themselves also in our ward but after the introduction of homatropine therapy they did not return any more.

Discussion

The attacks observed in five patients are a rare complication of oesophageal repair. The symptoms appeared at the age of some months, with sudden cyanosis, apnoea, laryngospasm and bradycardia, ending with syncope, always during or after a meal. Vomiting and even aspiration were frequent but we have observed bradycardia and syncope also without vomiting. The condition was often so serious that resuscitation was necessary. The attacks lasted for several minutes and were then followed by exhaustion and sleep. The presence of an oesophageal stricture as well as of a patent oesophago-tracheal fistula was excluded by serial examinations in every single case. The serum Ca, P, alkaline phosphatase and Mg levels were regularly controlled in each patient. Although a low serum Ca level was frequent in patients operated upon for oesophageal atresia, the slow heart beat failed to normalize on normalization of the Ca level.

The attack was successfully treated with intravenous atropine and as maintenance therapy homatropine in a dose of 1 mg t.i.d. was beneficial and caused no side effects such as pupillary dilatation, flushes or constipation. Atropine inhibits vagal stimulation in a direct way, but high doses have to be given [1]. In cardiac

reflectory activity both vagal nerves play a role. Stimulation of the left vagus nerve acts on the AV node, slowing down impulse conduction. Stimulation of the right branch affects primarily the sino-auricular node and by depressing the impulses causes bradycardia and even asystole.

In the course of the surgical therapy of oesophageal atresia with tracheo-oesophageal fistula, a primary anastomosis is generally done with transection of the fistula. When the fistula is excised and the upper pouch is mobilized to perform the anastomosis, the right vagus nerve is in the operational area. Moreover, the lifted parietal pleura falls back to the reconstructed oesophagus and fixes the nerve to the dissected part. Furthermore, there are considerable postoperative adhesions between the oesophagus and the right vagus nerve, and so the nerve is tugged during swallowing. This explains why attacks of bradycardia had developed during the ingestion of coarse food like vegetables or fruits when the food had reached the site of anastomosis.

The effect of atropine was always favourable. The infants demanding repeated resuscitation became symptomless. Regular administration of homatropine is of special importance since, as it had happened in our Case 4, if the therapy is discontinued, the attack might reoccur.

Our patients, in spite of the repeated episodes, developed well both mentally and somatically. EEG changes did not appear in any of the cases. There was no need so far to liberate the vagal nerve surgically as Bauer et al. [1] had done.

The phenomenon described probably occurs more often than one would suppose from the few pertaining reports. The common episodes that develop in patients operated upon for oesophageal atresia are usually diagnosed as aspiration, dysphagia or, in serious cases, unexpected sudden death. Some of these complications seem to be episodes of reflex bradycardia.

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