Cor Pulmonale as a Result of Chronic Nasopharyngeal Obstruction Due to Hypertrophied Tonsils and Adenoids

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It is important to recognize that heart failure in young children can be due to causes other than congenital heart disease. A syndrome has recently been recognized in children which can lead to gross cardiac failure and yet can apparently be permanently cured simply by removing the tonsils and adenoids. It is the syndrome of cor pulmonale due to hypoventilation resulting from chronic nasopharyngeal obstruction. Since the first reports (Menashe, Farrehi, and Miller, 1965; Noonan, 1965) others have appeared, bringing the total of reported cases to 16 (Luke *et al.*, 1966; Levy *et al.*, 1967; Ainger, 1968). We present here the clinical, electrocardiographic, and radiological findings in 3 patients, together with the results of cardiac catheterization in 2 of them.

Case Reports

Case 1. This child, aged 2½ years, was first admitted to another hospital when 15 months old, with a history of marked stridor, particularly at night, a persistent purulent nasal discharge, and a chest infection. He improved after treatment with antibiotics and was discharged after a fortnight. 2 months later he was re-admitted with a history of cough and breathlessness for 4 days and purulent nasal discharge. Stridor had been noticed which was worse at night, and cyanosis had also been observed when he was asleep. In addition, his parents had noticed that he was drowsy in the morning and that he took a long time to become completely awake. His chest infection was cured by antibiotics, but the cyanosis at night, and morning drowsiness persisted. He had no heart murmurs or heart failure, but x-ray of his chest showed cardiac enlargement. An electrocardiogram (ECG) showed evidence of right atrial and right ventricular hypertrophy.

He was referred for a cardiological opinion because of the unexplained cardiomegaly. When first seen he had signs of a moderate degree of pulmonary hyper-

tension and his heart size was increased radiologically. His ECG showed marked right atrial and right ventricular hypertrophy. Primary pulmonary hypertension was considered the most likely diagnosis, but some form of cardiomyopathy was also considered. Since he had improved and his heart size had decreased considerably by the time we saw him, he was not regarded as an urgent problem and his name was listed for cardiac catheterization. Within 3 weeks, however, his condition again deteriorated and he was admitted to the Cardiac Unit as an emergency.

On examination he was in gross right heart failure, with pitting oedema of the lower limbs to the level of the groins, marked facial oedema, moderate hepatomegaly, and shifting abdominal dullness. He was mildly cyanosed but there was no finger clubbing. His facies were adenoidal, and he snuffled and breathed through his mouth all the time. At night, or whenever he was lying on his back, he developed marked inspiratory stridor, increasing cyanosis, and sometimes complete respiratory obstruction. It was difficult to rouse him from sleep and he did not reach full consciousness for 10 minutes. He frequently fell asleep during the day. His tonsils were greatly enlarged, meeting in the mid-line. He had moderate sinus tachycardia, a 5 cm. A wave in the venous pulse, and a slight right ventricular lift. There were no murmurs, but there was a loud pulmonary click and the second sound was single and much accentuated. No added sounds were audible in the chest.

ECG (Fig. 1) showed marked right ventricular hypertrophy and strain, and severe right atrial hypertrophy. The chest x-ray (Fig. 2) showed a greatly enlarged heart, with a particularly dilated main pulmonary artery, and diffuse fluffy opacities in both lung fields. Three blood cultures were sterile, and blood Hb was 13.7 g./100 ml., white cell count 13,200/cu.mm., and erythrocyte sedimentation rate 1 mm./hr.

He was treated with digoxin and large doses of frusemide with potassium supplements, but despite this he deteriorated, continuing to gain weight, and becoming more somnolent and cyanosed. He was put in an oxygen tent one evening; the following morning he was no less cyanosed and it took 20 minutes of vigorous

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shaking to rouse him from sleep. The remarkable resemblance between this behaviour and that of a chronic bronchitic whose respiratory centre has become insensitive to a rise in arterial PCO_2 led to the correct diagnosis. O_2 therapy was discontinued, and an arterial blood sample taken while the child was still asleep showed a PCO_2 of 69 mm. Hg, a PO_2 of 65 mm. Hg, and a pH of 7.35. That such a sample could be taken at all is a measure of the depression of his central nervous system during sleep.

Further medical treatment and training in sleeping in the prone position, which relieved respiratory obstruction almost completely, improved his condition sufficiently for cardiac catheterization to be performed. Right heart catheterization was performed from the right saphenous vein without sedation; the femoral artery was also catheterized by an open Seldinger technique.

The results (Tables I and II) showed pulmonary hypertension, the pulmonary artery mean pressure never dropping below 24 mm. Hg. The highest mean value (44 mm. Hg) was the first record when the child was asleep and breathing air, at a time when there was considerable desaturation of the aortic blood ($\text{O}_2\text{sat.} = 70.5\%$) and a high PCO_2 of 76 mm. Hg. Crying was accompanied by a reduction in the pulmonary artery pressure to 26 mm. Hg, and at the same time, as expected, by a rise in arterial oxygen saturation and a fall in PCO_2 . The arterial PCO_2 was abnormally high throughout the test and the arterial O_2 saturation abnormally low except when O_2 was breathed. It was not possible to test the effect of breathing O_2 while the child was asleep, but 100% O_2 given while he was crying had little effect. Intubation with an endotracheal tube was carried out but was technically difficult because of his short neck, large tonsils and tongue, and lax epiglottis. He required atropine 0.2 mg., and suxamethonium 10 mg. i.v., followed by fluothane and nitrous oxide before intubation was possible. The patient was then allowed to recover consciousness. The figures at 100 min. (Table II) were taken just before he woke up, with the endotracheal tube *in situ*. Shortly afterwards it was coughed out.

Since these results were considered to prove the diagnosis of cor pulmonale secondary to hypoventilation due to upper respiratory obstruction, his tonsils and adenoids were removed on May 30, 1968. At operation the epiglottis was short, and was curled and collapsed on to the posterior wall of the pharynx. When the epiglottis was lifted up he had an excellent airway. The tonsils were large, but no bigger than those often seen in children without stridor or obstruction of the upper airways. The tonsils and adenoids were removed without difficulty. Within 24 hours the child was much improved and was breathing well without any stridor. The surgeon thought that the respiratory obstruction had been caused by the combination of the large broad epiglottis and the large tonsils.

The child continued to make excellent progress. When reviewed at Out-patients 1 month after his operation he was still mouth breathing at times but was otherwise well. Examination of his heart was normal,

there being no evidence of pulmonary hypertension. His ECG 1 month after operation showed normal P waves in lead II and the R wave in V3R had fallen from 21 mm. to 7 mm. (Fig. 3). A further record on October 22, 5 months after operation, was normal, and by this time the child no longer breathed through his mouth. Radiography showed a heart of almost normal size (Fig. 4).

Cardiac catheterization was repeated without any sedation on October 24. The results (Table II) showed a pulmonary artery pressure of 30/5 mm. Hg (mean pressure 12 mm. Hg); O_2 saturations showed no evidence of any left-to-right shunt.

Case 2. This child, aged 20 months, had been admitted to hospital on numerous occasions during his life with the diagnosis of asthmatic bronchitis and nasal obstruction. On July 27, 1967, skull x-ray after insertion of Hypaque into both nostrils showed posterior nasal obstruction due to adenoidal pads and excluded choanal atresia. He was readmitted to hospital on April 26, 1968, with the same complaints of dyspnoea, nasal discharge, and noisy breathing, and in the course of his stay in hospital developed severe right heart failure which required vigorous treatment with digitalis, diuretics, and antibiotics. At no time did he appear somnolent.

At the time of admission on May 20, he had marked inspiratory stridor particularly in the supine position, slight cyanosis, and early finger clubbing. There was slight hepatomegaly but no oedema. There was a slight right parasternal heave. Auscultation revealed no abnormality except for an accentuated pulmonary second sound. His tonsils were moderately enlarged. There was no nasal discharge.

ECG showed right atrial hypertrophy and deep symmetrical T wave inversion over the right ventricular leads, as far as V4, though on QRS voltage criteria there was no right ventricular hypertrophy. The frontal QRS axis was -170° . The chest x-ray showed moderate cardiac enlargement and some congestion of the lung fields.

Two days later, cardiac catheterization (results in Table III) was carried out without sedation. The child's condition was better than that of Case 1 at the time of catheterization. The same abnormalities were present but were not nearly so striking. The arterial PCO_2 was abnormally raised except during crying, and the arterial pH correspondingly low. The administration of O_2 in the resting state was accompanied by a rise of arterial PCO_2 . The arterial O_2 saturation was abnormally low, and increased during crying. There was slight but definite pulmonary hypertension, which was increased during the administration of O_2 . The pulmonary artery pressure was increased during crying despite the fall in arterial PCO_2 and the rise in arterial O_2 saturation. This child was not intubated.

The upper respiratory tract was investigated on July 17, and enlarged tonsils and adenoids were found, but the epiglottis was normal. His tonsils and adenoids were removed and he improved. Digoxin was dis-

FIG. 1. ECG before tonsillectomy in Case 1, shows right atrial enlargement (tall peaked P waves in lead II); right ventricular enlargement (tall R waves in V3R and V1); T wave inversion from V3R to V5.

FIG. 2.—Chest x-ray before tonsillectomy in Case 1 shows marked cardiomegaly.

continued on July 28. When seen 1 month later, there was no evidence of heart failure and he was breathing well through his nose with his mouth closed. He continued to make satisfactory progress and there was no recurrence of heart failure. He died after an accident on September 10, in which his liver was ruptured. Necropsy was performed and there was no abnormality of the heart either macroscopically or microscopically.

Case 3. This boy aged 3 years showed the same syndrome but in a less severe form. He was admitted

FIG. 3.—ECG 1 month after tonsillectomy in Case 1, shows normal P waves in lead II and normal right ventricular activity.

FIG. 4.—Chest x-ray in Case 1, 5 months after tonsillectomy, shows marked reduction in heart size compared with Fig. 2.

to hospital for tonsillectomy and adenoidectomy because of frequent sore throats. He had large tonsils and cervical glands. The nursing staff reported that he had cyanotic attacks during the night. No heart murmurs were heard, but x-ray of his chest showed an enlarged heart (Fig. 5) and he was referred for a cardiological opinion. On April 7, 1967, he was mouth breathing and had minimal cyanosis at rest. There was profuse purulent nasal discharge. There were no significant heart murmurs, but his chest x-ray showed cardiac enlargement with a large right atrium and

TABLE I

Results of Cardiac Catheterization in Two Patients with Cor Pulmonale due to Upper Respiratory Obstruction

Case No.	IVC	SVC	Right Atrium		Right Vent.		Pulm. Art.		Aorta	
	O ₂ Sat. (%)	O ₂ Sat. (%)	Press. (mm. Hg)	O ₂ Sat. (%)	Press. (mm. Hg)	O ₂ Sat. (%)	Press. (mm. Hg)	O ₂ Sat. (%)	Press. (mm. Hg)	O ₂ Sat. (%)
1	67	63.5	-3	65, 64, 66.5	76/-3	62	76/28	62	134/64	70.5
2	63.5	65	-2	64, 65, 66.5	33/0	64.5	33/4	62	121/68	86

Note: O₂ saturations measured with Kipp Haemoreflexor. Pressures measured with Satham 23D transducer, relative to sternal angle.

TABLE II

Variations in Aortic and Pulmonary Arterial Pressures, Blood Gas Tensions, and pH under a Variety of Conditions in Case 1

Time (min.)	State of Patient	Aortic					Pulm. Artery
		O ₂ Sat. %	Po ₂ (mm. Hg)	Pco ₂ (mm. Hg)	pH	Mean Pressure (mm. Hg)	Mean Pressure (mm. Hg)
0	Asleep, breathing air	70·5	42	76	7·28	88	44
40	Crying, breathing air	94	76	52	7·39	88	26
75	Crying, breathing 100% O ₂	100	570	48	7·37	84	28
100	Intubated, asleep breathing air	86	64	66	7·24	66	24
105	Intubated, awake breathing air	89	66	48	7·32		
Repeat	cardiac catheterization after tonsillectomy						
0	Awake, breathing air	94	100	25	7·39	60	12

Note: Blood gas tensions and pH measured by Astrup.

TABLE III

Variations in Aortic and Pulmonary Arterial Pressure, Blood Gas Tensions, and pH under Variety of Conditions in Case 2

Time (min.)	State of Patient		Aortic				Pulm. Artery				Mean Pressures (mm. Hg)	
			O ₂ Sat. (%)	Po ₂ (mm. Hg)	Pco ₂ (mm. Hg)	pH	O ₂ Sat. (%)	Po ₂ (mm. Hg)	Pco ₂ (mm. Hg)	pH	Aorta	Pulm. Art.
0	Crying, breathing air	..	94	75	34	7.37	64.5	38	38.5	7.31	108	24
73	Asleep, breathing air	..	86	62	44.5	7.30	62	40	45	7.29	92	16
99	Asleep, breathing O ₂ -enriched air	..	98.5	141	49	7.25	71.5	47	50.4	7.24	104	20
121	Asleep, breathing air	..	93.5	79	40	7.32	64.5	40	47	7.28	104	18

congestion of the lung fields. ECG (Fig. 6) showed right axis deviation, peaked P waves in lead II measuring 4 mm., rsR complex in V₁, with R = 19 mm. The cause of this right atrial and right ventricular hypertrophy was obscure but there was no evidence of heart failure. We recommended that tonsillectomy and adenoidectomy should be performed because the upper respiratory obstruction was so severe; this was done on May 8. The tonsils and adenoids were very large indeed. No comment was made about the epiglottis.

The child subsequently made excellent progress,

and when seen on July 4 was breathing well without obstruction. There was no cyanosis, and the heart had decreased in size, now being within normal limits on x-ray (Fig. 7). The P waves in lead II now measured 2 mm. and were no longer peaked; the R wave in V₁ measured 9 mm. He was seen again on August 27 when his condition was excellent. Examination of his heart showed no abnormality and his ECG had returned to normal, the R wave in lead V₁ now measuring 4 mm., and the P waves in lead II being normal (Fig. 8).

In retrospect we feel that had not tonsillectomy been

FIG. 5.—Chest x-ray before tonsillectomy in Case 3 shows increase in transverse diameter, prominent pulmonary artery, and fluffy hilar opacities.

FIG. 7.—Chest x-ray in Case 3, 2 months after tonsillectomy, shows reduction in heart size and pulmonary vascularity.

FIG. 6.—ECG in Case 3 before tonsillectomy shows right atrial hypertrophy (peaked P waves in lead II), and right ventricular enlargement (tall R wave in V3R and V1).

FIG. 8.—ECG in Case 3, 15 months after tonsillectomy. Record is now within normal limits.

recommended for this boy, despite the fact that he was thought to have a cardiac lesion, he would probably have progressed in the same way as Case 1, where tonsillectomy was deferred until the cardiac lesion had been diagnosed.

Discussion

Our initial experience in recognizing this condition was very much the same as that of others (Luke *et al.*, 1966; Levy *et al.*, 1967; Ainger,

1968). Preliminary diagnoses of cardiomyopathy or primary pulmonary hypertension were made, and only rejected when we observed the response to administration of O_2 , by which time the patient's condition had become critical. Yet, in retrospect, the clinical features of the disease are so striking that a firm diagnosis may be made with no more technical assistance than an ECG and an arterial blood sample.

All 19 cases described in the literature, including our own, had marked stridor while in the supine position; in every case this was relieved by removal of tonsils and adenoids. Of these 19, 12 (the most severely affected) also showed somnolence, sometimes to the point of semiconsciousness. Cyanosis was also a feature in severe cases. In every case there was clinical evidence of pulmonary hypertension. In 14 this had resulted in right heart failure which was often considerable, and was resistant to treatment with digitalis and diuretics.

The ECG in all except one (Case 2, Menashe *et al.*, 1965) showed right atrial hypertrophy, and in all cases except 2 (Case 2, Luke *et al.*, 1966; and Case 2 this series) right ventricular hypertrophy. 5 of the 8 published electrocardiograms, including those shown here, exhibited striking deep T wave inversion over the right chest leads.

All of the 13 chest x-rays described (including our 3 cases) showed cardiomegaly, usually with prominence of the pulmonary artery, and 9 of 13 were thought to show pulmonary oedema in the lung fields.

Cardiac catheterization has been performed in 8 cases in addition to our own, and pulmonary hypertension has been the invariable finding except in 2 cases (Cases 3 and 4, Luke *et al.*, 1966). Most authors have mentioned the marked dips in pulmonary artery pressure associated with inspiration due to the inspiratory obstruction, and our cases showed this. In every case there has been arterial hypoxaemia and CO₂ retention, and as one study in particular has shown, sleep produces progressively greater hypoxaemia and CO₂ retention together with progressive increase in pulmonary artery pressure (Menashe *et al.*, 1965).

There is an interesting contrast between 2 of the cases described here with respect to the effects of crying. In the 2 cases that had cardiac catheterization, crying produced a rise in arterial Po₂ and a fall in arterial PCO₂ but in Case 1 this was associated with a fall in pulmonary artery pressure, whereas in Case 2 the pulmonary artery pressure was raised during crying. Crying in a normal child will raise the pulmonary artery pressure by increasing the cardiac output. It seems that in Case 1 this effect was more than counteracted by the pulmonary vasodilatation resultant upon the improvement in ventilation. In Case 2, which approaches more nearly the normal situation, presumably the reduction of pulmonary vascular resistance occasioned by the improvement in ventilation was small compared with the increase in cardiac output, so that the over-all effect upon the pulmonary artery pressure was to raise it.

Little information is available as to the acute effects of administration of O₂ and improving the airway on these patients. In 1 child studied by Noonan (1965) a rise in pulmonary artery pressure was produced by administration of 100% O₂, the PCO₂ meanwhile rising from 62 to more than 100 mm. Hg. On the other hand Menashe and associates (1965) found that in their second case O₂ administration caused a fall in pulmonary artery pressure, despite an increase in CO₂ retention. In the case described by Levy *et al.* (1967), the insertion of a nasopharyngeal tube to relieve the airway obstruction caused a fall in pulmonary artery pressure at the same time as a rise in arterial O₂ saturation and a fall in arterial PCO₂. Subsequent O₂ administration caused a small rise in PCO₂ and a fall in the pulmonary artery pressure. In the first study described here, it was not possible to test the effect of O₂ administration during sleep, but endotracheal intubation resulted in a marked fall in mean pulmonary artery pressure from 44 to 24 mm. Hg, while the arterial O₂ saturation rose from 70.5 to 89%, the PCO₂ fell from 76 to 48 mm. Hg, and the pH rose from 7.28 to 7.32. However, the drugs given in order to assist intubation may themselves have affected these results. In the second case, intubation was not performed, but the administration of O₂-enriched air to a point sufficient to raise arterial Po₂ from 62 to 141 mm. Hg resulted in a rise in mean pulmonary artery pressure from 16 to 20 mm. Hg, while arterial PCO₂ rose from 44.5 to 49 mm. Hg, and pH fell from 7.30 to 7.25.

The studies described here confirm that in this syndrome pulmonary hypertension is associated with a low arterial O₂ saturation and a high arterial PCO₂, the classical changes of alveolar hypoventilation. They also show that relief of the upper respiratory obstruction results in return of all these abnormal values towards normal, and that administration of O₂ results in further CO₂ retention which may depress consciousness to dangerous levels, and may be associated with either a rise or a fall in pulmonary artery pressure. That artificially-imposed upper respiratory obstruction causes reduced ventilation and CO₂ retention has been demonstrated experimentally (Cherniack and Snidal, 1956).

What factors in the human are responsible for the control of pulmonary vascular resistance? There is little doubt that a major factor is arterial Po₂, a fall in which causes pulmonary vasoconstriction (Euler and Liljestrand, 1946; Fishman, 1961; Enson *et al.*, 1964). The role of arterial PCO₂ and pH are more controversial, but there seems to be

a growing amount of evidence for the important role of pH in the control of pulmonary vessel tone, since Liljestrand first pointed out that hypoxia, by promoting lactic acid release, increases hydrogen ion concentration which itself may cause pulmonary vasoconstriction (Liljestrand, 1958). The lower the arterial O_2 saturation the greater is the effect of a change in pH , and the higher the arterial pH , the less sensitive is the pulmonary artery pressure to hypoxia (Enson *et al.*, 1964; Harvey *et al.*, 1967). These findings have been confirmed in the newborn calf (Rudolph and Yuan, 1966). Of particular interest in the context of this paper is a report of a study on a patient with the Pickwickian syndrome, the mechanism of which is presumably similar to that of cases of cor pulmonale due to upper respiratory obstruction. With voluntary hyperventilation a fall in pulmonary artery pressure was produced as well as a rise in arterial O_2 saturation and arterial pH . The administration of 100% O_2 caused no change in mean pulmonary artery pressure despite a significant increase in arterial O_2 tension; and with administration of THAM, there was a decrease in both hydrogen ion concentration and pulmonary artery pressure, though the O_2 tension remained essentially unchanged (Vogel and Blount, 1965).

In none of the cases so far described of cor pulmonale due to upper respiratory obstruction have the changes in cardiac output been measured; some of the rise in pulmonary artery pressure induced by a fall in arterial pH may be due to the resultant increase in cardiac output rather than to an increase in pulmonary vascular resistance.

In the cases reported here, as well as in others in the literature, there is a striking disparity between severity of right heart failure and severity of pulmonary hypertension. Right ventricular pressures of 200 mm. Hg systolic may be tolerated in pulmonary stenosis for some years without heart failure supervening, yet in this syndrome it appears that an increase in right ventricular systolic pressure to 50 mm. Hg for a few weeks may result in right ventricular failure. This may be the result of the acuteness of the right ventricular pressure load, or of depression of myocardial performance by a combination of hypoxaemia and acidaemia as Ainger (1968) has suggested, but it should be remembered that the incidental right ventricular pressure measured during right heart catheterization may be low by comparison with the pressure levels reached in the child after a night's sleep, with ever-increasing anoxia and CO_2 retention.

The clinical implications of the existence of this syndrome are far reaching. Thousands of tonsil-

lectomies are performed in this country each year and yet this complication of tonsillar and adenoidal hypertrophy has not been recognized. Many children with tonsils far larger than those of the children described here have no respiratory obstruction, and in fact are refused operation because there is insufficient indication for it. The first child described here had three sibs, all of whom had required tonsillectomy for various reasons, but he was the only one who developed this syndrome. This suggests that, in order to develop cor pulmonale in the presence of hypertrophied and infected tonsils and adenoids, some other factor must also be involved. The likeliest factor for this is an abnormal reactivity of the pulmonary vasculature to hypoxia such as that shown in approximately 20% of normal persons at altitude, and in some children with small ventricular septal defects (Grover *et al.*, 1963). This mechanism has also been used to explain why not all grossly fat people develop the Pickwickian syndrome.

A possible explanation for the recognition of this syndrome in the past few years is that, with the trend towards conservatism in the management of infected tonsils and adenoids, this syndrome has been allowed to develop in children who 15 years ago would have had a tonsillectomy.

Nevertheless, it remains quite likely that some children suffering from this syndrome have been operated on and cured without the situation being appreciated. This happened in Case 3, the first patient we saw with early symptoms of this syndrome. Clearly, the administration of opiate premedication to such a child is fraught with the danger of exacerbating hypoventilation and thus producing excessive CO_2 retention and even higher pulmonary artery pressures. Furthermore, the induction of anaesthesia is extremely likely to be complicated by complete respiratory obstruction unless special care is taken.

Ainger (1968) has reported 2 fatalities from this syndrome, so prompt recognition and treatment are important. Cardiac failure should if possible be abolished by medical treatment before operation, though if the failure proves intractable, operation may have to be carried out as an emergency.

Training the child to sleep in the prone position may be as valuable as any drug therapy, and the administration of O_2 , if it is given at all, should be subject to the same precautions as in an adult patient with cor pulmonale due to obstructive airway disease (i.e. 100% O_2 should never be given and O_2 -enriched air should only be given when constant watch is kept for the signs and symptoms of increasing CO_2 retention).

Summary

Three cases are reported of the syndrome of cor pulmonale in children due to chronic nasopharyngeal obstruction by hypertrophied tonsils and adenoids. The syndrome is characterized by: (1) Stridor in the supine position due to nasopharyngeal obstruction by hypertrophied tonsils and adenoids. (2) Somnolence. (3) Pulmonary hypertension. (4) Right heart failure. (5) Arterial hypoxia and hypercarbia. (6) Electrocardiographic changes suggestive of right atrial and right ventricular hypertrophy and right ventricular strain. (7) Radiographic appearances of cardiomegaly, dilatation of the pulmonary artery, and often pulmonary oedema.

The probable pathogenesis of the syndrome is that pulmonary hypoventilation leads to arterial hypoxaemia and acidaemia, which in turn lead to pulmonary vasoconstriction and hypertension. Cardiac failure should first be treated medically, and the tonsils and adenoids should subsequently be removed. Sleeping in the prone position often relieves the upper respiratory obstruction. O₂ should be given with care since it may exacerbate the symptoms.

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