? Congenital Lesion of Heart or Pericardium.—H. V. MORLOCK, M.D.

C. V., male, aged 2 years 9 months, complained of cough during summer 1934, worst at night. Tonsils removed August 1934; some improvement with regard to the cough since then, though the child still has occasional nocturnal dyspnœa.

Past history.—Operation for inguinal hernia August 1933; pertussis April 1933; diphtheria followed by pneumonia November 1933; measles followed by pneumonia March 1934.

Condition on examination.—Well nourished; good colour. No clubbing or cyanosis; no dyspnœa when at rest. Apex beat in fourth space in nipple line. Impaired percussion note in first and second interspaces immediately to right of sternum. No other physical sign of disease.

Skiagrams.[1]—Large opaque mass with well-defined rounded margin extends from the mediastinum into the right lung field under the screen. The upper part pulsates but appears distinct from the lower part of the shadow which also pulsates.

The skiagrams only of this case were shown by Dr. R. W. B. Ellis at the December meeting of the Section, and the diagnosis of a mediastinal dermoid was made. At this time, however, the patient was in the Chest Hospital, Victoria Park, undergoing the production of a complete artificial pneumothorax in order that the mediastinum might be inspected with the thoracoscope.

On thoracoscopic examination both the anterior and the posterior mediastinum were seen to be clear of any form of tumour or glands.

The anterior mediastinum was filled with the pericardium and the great vessels occupying the position of the shadow seen on the skiagram. Within the pericardium, in the position of the lower part of the shadow could be seen waves of contraction which were passing from below upwards. The frequency of these contractions corresponded to the frequency of the heart beat. The position of the upper part of the shadow was occupied by a very large pulsating vessel, the pulsations of which followed immediately upon the contraction occurring in the lower part of the shadow.

The large vessel was considerably bigger than the aorta of a child of three years.

Comment.—The thoracoscopic examination left no doubt that the cause of the shadow was within the pericardium, and in view of the child's age and the absence of a history of previous illness it seems fair to assume that the lesion is of congenital origin.

Maud Abbott in her monograph on "Congenital Heart Disease" shows a skiagram with a similar shadow, which was due to a coarctation of the aorta, and this may be the interpretation in this case, although the child shows none of the collateral arterial hypertrophies which are usually associated with a coarctation, neither does he exhibit a higher branchial blood-pressure than that in the femoral artery. (Both equal at 100/60.)

The absence of the associated signs of a coarctation of the aorta may be due to the age of the child, and it may be that as he grows and becomes more active and more stress is put on his arterial system, these arterial changes will become manifest.

Although no absolutely certain diagnosis can be made, as the result of the thoracoscopic examination, one has been able to give a much more favourable prognosis, than before the examination, when the possibility of a mediastinal new growth had not been excluded.

Case showing the Effect of Prostigmin on Myasthenia Gravis.—M. B. WALKER, M.R.C.P.

D. C., female, aged 40.

History.—In spring 1930 she noticed undue fatigability of the arms and drooping of the right upper eyelid. In July 1930 she was admitted to the Middlesex Hospital with diplopia, and was discharged, appreciably better, within a month. January 1931, readmitted with impairment of all movements of the left eye and weakness of the arms and legs, which became worse towards the end of the day. In

[1] See *Proceedings*, 1935, xxviii, 448 (Sect. Dis. in Child. 22).

April she had difficulty in swallowing and regurgitation of fluids through the nose. She was treated with ephedrine with improvement, and whenever the ephedrine was stopped the symptoms became worse. She was at work from September 1931 to September 1933, taking ephedrine all the time. In May 1933 she again complained of diplopia, and in September she was readmitted to the Middlesex Hospital where she was treated with glycine and ephedrine, but did not respond so well. No ephedrine was given after she left the Middlesex Hospital.

In September 1934 the symptoms became worse and she was transferred to St. Alfege's Hospital on October 24.

Condition on admission.—Ptosis of left upper eyelid; partial external ophthalmoplegia; weakness of arms, especially of flexors of fingers; weakness of muscles of back, and of lower limbs. The thyroid was enlarged, but the thymus was not.

She had great difficulty in raising herself up in bed, could only walk a few yards unsteadily, could not feed herself after the first few mouthfuls, and complained of diplopia. Speech was slow and became indistinct after a few sentences. Swallowing was difficult and fluids regurgitated through the nose.

Her condition has remained unchanged except that she has not complained of diplopia for the last few days, and her grip is stronger than it was on admission.

A hypodermic injection of prostigmin relieves these symptoms temporarily. Atropine given at the same time prevents colic and nausea without affecting the action of prostigmin on the motor nerve-endings.

Since December 18, 1934, she has had 2·5 mgm. of prostigmin and 0·66 mgm. of atropine daily at 10 a.m. Five minutes after the injection the ptosis and external ophthalmoplegia disappear, a few minutes later she sits up in bed easily, in ten to fifteen minutes she can walk several hundred yards without feeling tired. After the effect of the injection on the fatigability has worn off, the muscles feel a little stiff.

The effect is at its height an hour after the injection and begins to wear off gradually in about six hours. Occasionally a second injection is given at 4 p.m. and the effect lasts until late in the evening.

Smaller injections were given at first, with the following results:—

Amount of prostigmin Mgm.		Onset of effect (Minutes)		Duration of effect (Hours)
0·5	...	30-45	...	2-3
1·0	...	20	...	4-5
1·5	...	10	...	4-5
2·0	...	8	...	5·6
2·5	...	5	...	6·8

The larger the dose, the greater the increase of muscular power.

Given by the mouth 1·5 mgm. of prostigmin caused nausea, the effect was much less, and came on much later than when the drug was given hypodermically.

Control injections of ephedrine, lobeline, femergin, and water produced no effect on the muscular weakness.

Physostigmine salicylate, 1 mgm. approximately, removed the ptosis but made the patient feel sick and faint and disinclined to move. Atropine given with the physostigmine counteracts these ill-effects, without altering the action on the motor nerve-endings.

Physostigmine salicylate was given in a previous case of the disease which was admitted to St. Alfege's Hospital in April 1934, because it was thought that as the muscles in myasthenia gravis behave like muscles poisoned by curare, physostigmine, an antagonist to curare, might also counteract the unknown substance which might be exerting a curare-like effect on the motor nerve-endings in myasthenia gravis. The patient tolerated it well, and its effect on the weakness and fatigability was the same as that of prostigmin, 1·0 mgm.

The advantages of prostigmin (Roche)—a synthetic drug analogous to physostigmine and with similar actions—over physostigmine are that it has a less

depressing effect on the heart, less often causes nausea and vomiting, and is probably safer in large doses; 4 mgm. have been given without ill-effect, though in other cases the same dose has caused severe diarrhœa and cardiac and respiratory distress. The disadvantage is its expense, the price of an ampoule containing 0·5 mgm. of the drug, being ninepence.

Dr. P. HAMILL: Whatever may be the mechanism of the weakness and fatigability of the muscles in myasthenia gravis, physostigmine, and its ally, prostigmin, overcome it. The resulting voluntary movements are normal in type. If it is the case that nervous impulses set free acetylcholine—or some analogous substance at the nerve-endings, and that in myasthenia the supply is deficient, physostigmine, by delaying its destruction, would compensate for the lack.

On this hypothesis, defective innervation, whether resulting from some disability of the anterior-horn cells or impaired conduction of nerve-fibres, as in neuritis, should also be corrected by injection of physostigmine and prostigmin. This appears to be the case, the drugs having been used in cases of peripheral neuritis and in spinal-cord lesions with beneficial results. Muscular power is increased. As additions to massage in such cases, the drugs may help in maintaining nutrition of the paralysed muscles and thus accelerate their recovery. The economic aspects are important. A wide field of usefulness in neuro-muscular disorders is thus opened and is now being studied; the results will be communicated at a later date.

Portal Cirrhosis in a Child.—G. H. NEWNS, M.D. (by courtesy of Dr. WILFRID SHELDON).

W. F., male, aged 4½, admitted to King's College Hospital in January 1935, under Dr. Sheldon.

History.—The parents have never noticed anything wrong with the child, apart from recent epistaxis in the mornings. He is quite normal mentally. He has never had any serious illnesses and has never been jaundiced. He was sent to hospital by the school doctor, who had found an enlarged liver and spleen.

Family history.—There are seven other children, five older than the patient. All are well, and there is no ascertainable history of jaundice among them.

Condition on examination.—Slightly smaller than normal in stature. Rather sallow complexion, but not jaundiced. A few telangiectases on the face. Abdomen somewhat tumid. Liver enlarged almost a handbreadth below the costal margin. Consistency very hard, and edge extremely sharp. Surface not obviously nodular. Spleen also enlarged to about 2 in. below the costal margin, consistency very firm.

Investigations.—Wassermann reaction negative.

Blood-count: R.B.C. 4,920,000; Hb. 92%; W.B.C. 8,600; polys. 81·2%; lymphos. 10·8%; eosinos. 7·2%; basos. 0·4%.

Lævulose tolerance test:—

	10 a.m.	10.30 a.m.	11 a.m.	11.30 a.m.	Noon.
Blood-sugar %	0·095				
,, after 25 gms. lævulose		0·107	0·095	0·103	0·095

Unexplained Exophthalmos.—A. L. WINGFIELD, M.D.

F. C., engineer's fitter, aged 64.

1928: Struck on right side of head in a motor accident.

Summer 1934: Watering of the eyes and misty vision.

Autumn 1934: Eyes became bloodshot; were treated with lotion. Head began to feel heavy. Apparent loss of perspective.

November-December 1934: Hands became shaky. Hot flushes over body. Did not become nervous. Has lost 2 st. in weight since operation for perforated gastric ulcer in 1934. Occasional headaches and dyspnœa. Early this year patient had some diplopia but this has now disappeared.

Condition on examination.—Temperature normal. Pulse 78-96 since admission.