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President—Professor A. G. WATKINS, M.D., F.R.C.P.

[*February* 22, 1952]

Dystonia Musculorum Deformans in Siblings. Treated with Artane (Trihexylphenidyl).—BERYL D. CORNER, M.D.

Family history.—The two children described are the only siblings. Parents healthy; no Russian or Jewish ancestry; family have lived in Bristol for several generations. A paternal uncle has had this disease since the age of 8 years. He is now 44 years and completely bedridden.

Case I.—A. S., female, aged 14 years.

History.—January 1946: Started dragging left leg. In the morning she walked normally but by midday walking deteriorated with twisting and kicking of her left leg. Her hands shook on movement. Diagnosed as hysteria: psychiatric investigation carried out at Bristol Child Guidance Clinic. I.Q. 109. Considered to be immature and given psychiatric treatment for six months. She was subsequently seen by Miss D. Kinloch Beck who diagnosed dystonia musculorum deformans.

May 1947: Steady deterioration: now unable to walk by midday; persistent complaints of shaking and unable to hold cup and saucer.

On examination: Much spontaneous movement of athetoid type; both arms tend to be held in extension with considerable muscular rigidity; some lumbar lordosis; hips flexed and adducted: typical torsion spasm of calf muscles on walking, feet plantar flexed and inverted. No sensory changes. Euphoria marked. Reflexes normal.

Investigations.—C.S.F. normal. Lange curve negative. W.R. negative. Liver function tests no abnormality. Electro-encephalogram: Normal rhythms from the pre-central areas. In the post-central regions a very high potential complex delta and theta discharge was seen which was almost completely inhibited by opening the eyes. Alpha rhythm from the occipital lobes was 8–9 c/s and was responsive to physical stimuli.

23.2.50: Very marked deterioration. Her face was expressionless but contortions occurred on attempted movement of other parts of the body. Speech and mastication were impaired. There was permanent flexion of all large joints of the lower limbs, initiation of movement was very difficult and she could only walk 10 yards. She was unable to dress or feed herself except in the early morning, and could only write with considerable difficulty.

Further investigations.—Serum copper normal. No response to therapeutic test with BAL. EEG little change, but occasional spike and wave patterns were elicited.

Treatment (30.3.50).—Artane mg. 2 b.d. was increased to mg. 4 b.d. during a period of three weeks. Dramatic improvement occurred. She was able to undress herself; walking distances much improved; hands much steadier; less muscular rigidity also noted.

Progress.—Treatment has continued with the same dose of Artane (mg. 4 b.d.) In December 1950 she was able to join in school games and walked a mile.

February 1952: All flexion deformities have disappeared: no detectable muscular rigidity: writing completely normal. Child is living a normal active life, can use a sewing machine and swim a few strokes. She has no symptoms and is extremely cheerful.

This case was shown to the Section of Neurology in 1947 by Diana Kinloch Beck (*Proc. R. Soc. Med.*, **40**, 551).

I showed both children to the Section of Pædiatrics in June 1949, at the meeting held in Bristol.

Case II.—R. S., male, aged 11½ years.

History.—October 1948: He was first seen with history of difficulty in walking, late in the afternoon, and for six months tendency to twist right leg. Examination showed definite gait characteristic of early torsion spasm affecting both legs, but right worse than left.

Investigations.—January 1949: C.S.F. normal. Serum potassium 17·2 mg.%. Urine copper excretion 0·0045 mg./ml. (Normal value.)

February 1950: Progressive deterioration. By midday unable to straighten his back and great difficulty in walking; difficulty in change of posture; typical gait and movements of torsion spasm; head and upper extremities not greatly affected.

May 1950: Treatment with Artane 2 mg., increasing to 7 mg. daily. Further increase in dosage produced blurred vision and vomiting. Improvement in gait was noted after six weeks, and the same dosage has been maintained since.

May 1951: Walking normally. Attending ordinary school. Playing games and swims 50 yards.

February 1952: No abnormal physical signs. Only abnormality is a tendency to stand with left hip slightly flexed when he is very fatigued.

Comment.—These children are typical cases of dystonia musculorum deformans (torsion spasm) who at the start of treatment with Artane were severely handicapped and rapidly deteriorating. Artane (trihexyphenidyl) is a synthetic piperidyl compound with marked antispasmodic action on smooth muscle and an inhibitory action on the parasympathetic nervous system. The relief of spasticity in voluntary muscles is considered to be largely due to the latter action as well as to some direct effect on the motor centres. Other atropine-like actions such as mydriasis, control of sialorrhœa, and cardiovascular effects are considerably less than atropine and even in very large doses the cerebral excitation produced does not lead to convulsions. The drug has been most effectively used for the control of tremor and rigidity in parkinsonism; it was therefore hoped that since the characteristic movements and rigidity of dystonia musculorum deformans are also due to degenerative lesions of the basal ganglia this drug might be effective, and this has proved to be the case. Artane is given orally in capsules, three or four times daily, starting with 1 mg. daily and gradually increasing until the effective level is reached, usually 6 to 10 mg. daily.

Dysplasia Epiphysealis Multiplex.—L. G. Scott, M.D., M.R.C.P.

D. K., male, aged 8 years (Born 23.5.44).

He has had vague pains in his legs for the past two and a half years. His legs have been noticed to be thin and the knees relatively large. There is nothing else of significance in the history and no significant abnormality on examination apart from thin legs, flat feet, broad and coarse hands and fingers, and the fact that he is rather small for his age. In October 1951 his height was 43½ inches (average = 46 inches) and weight 40 lb. (average = 49 lb.). His mentality is good and he has not the appearance of a cretin.

Blood chemistry is normal: Serum alk. phosphatase 18 King-Armstrong units; serum phosphorus 4·3 mg.%; blood cholesterol 152 mg.%. W.R. and Kahn negative.

X-rays show changes of dysplasia epiphysealis multiplex in a mild form. There is irregularity in ossification of the epiphyses. The irregularity is both in density and shape. Subsidiary centres of ossification are present around the main centres, resulting in some peripheral stippling but this is not so complete as seen in the punctate type of epiphyseal dysplasia (*see* next case). There is delay in ossification of the epiphyses and only three carpal centres were present on 29.6.51. The external malleoli at the ankle-joints are abnormally low and this has been described in some of the previous cases.

This case has also been seen by Dr. Philip Evans, Professor Alan Moncrieff, and Sir Thomas Fairbank at The Hospital for Sick Children, Great Ormond Street, and I thank them for their permission to publish it.

Comment.—In 1946 and 1947 Sir Thomas Fairbank published papers on a series of 20 cases of what he believed to be a clinical entity and suggested the title Dysplasia Epiphysealis Multiplex. In 1951 he reviewed 26 such cases. The condition is a rare developmental error characterized by mottling or irregularity in density of several of the developing epiphyses, dwarfism, and stubby digits.

REFERENCE

Fairbank, H. A. T. (1951) An Atlas of General Affections of the Skeleton. Edinburgh and London.

Dysplasia Epiphysealis Punctata.—L. G. SCOTT, M.D., M.R.C.P.

C. S., female, aged 4 months (Born 26.10.51).

At birth the child was noticed to have bilateral flexion deformities of the wrists, and she soon became snuffly. Subsequent radiographs of the skeleton showed the appearances of dysplasia epiphysealis punctata. There is widespread stippling of the epiphyses as if they were ossifying from many separate centres. The shafts of the humeri are short and thick and the ends splayed to a marked degree (*see* Figs. 1A, 1B and 2).

This baby has the typical flexion deformities of other joints besides the wrists. On 13.1.52 measurements showed:

Lt. arm at elbow 30° from full extension	Lt. leg at knee 50° from full extension
Rt. arm at elbow 20° from full extension	Rt. leg at knee 60° from full extension

The child has bilateral cataract which is being treated by needling, by Mr. S. S. Freedman.

She also has relative lengthening of the forearms and legs compared with the arms and thighs. Measurements on 8.2.52 were:

Total Height 20½ in.

Both Arms	Both Legs
Tip of acromion to olecranon 3 in.	Greater trochanter to patella 4 in.
Olecranon to styloid process of ulna 2¾ in.	Patella to lateral malleolus 4½ in.

The other systems appear normal, and no thickening of the skin has been noticed.

I wish to thank Dr. E. Owen Fox for the radiographs of this case.

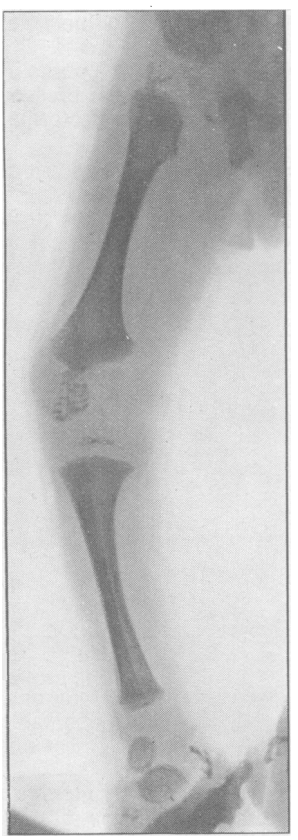

FIG. 1A.

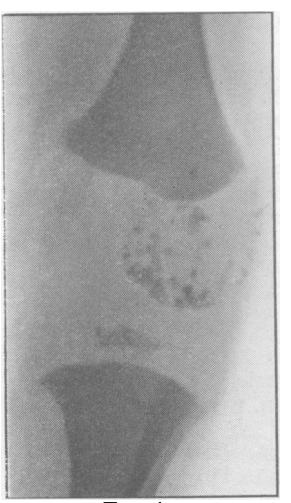

FIG. 1B.

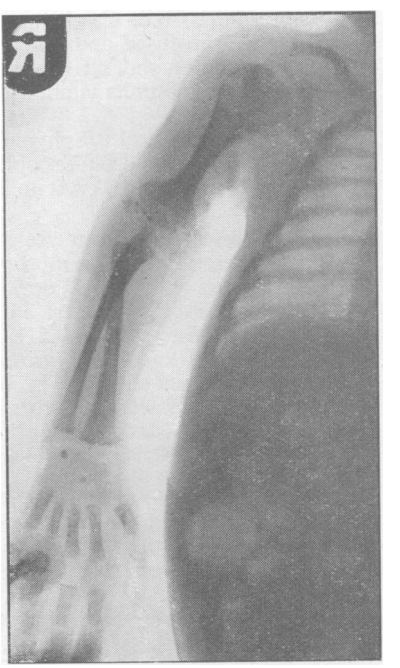

FIG. 2.

FIG. 1A.—Radiograph of the right lower limb at the age of 7 weeks, showing widespread stippling of the epiphyses.

FIG. 1B.—Radiograph of the left knee at the age of 7 weeks.

FIG. 2.—Radiograph of the right upper limb at the age of 7 weeks, showing the characteristic short and thick humerus with marked splaying of each end, as well as stippling of the epiphyses.

Fanconi Syndrome in Two Siblings.—S. B. DIMSON, M.D., M.R.C.P.

Case I.—J. J., boy, aged 4 years.

Admitted to Sydenham Children's Hospital 9.5.51 in tetany: ten days previously had brief attack of dysentery. Enuresis and thirst always. Stools always normal. Appetite good. Parents and 3 siblings (apart from Case II) normal. No consanguinity.

Past history.—Birth weight 7 lb. Teething 8 months. Bow legs noticed at 1 year. Walked at 1 year 4 months. Had cod-liver oil irregularly.

On examination.—Typical tetany. Pallor, fair hair, bossed skull, genu varum, moderate beading of ribs, enlarged epiphyses, distended abdomen. Stools, B.P. and fundi normal. Undersized; infantile behaviour. Weight 19 lb. Height 30 in.

Urine colourless. S.G. 1002–8. Albumin moderate cloud. Occasional granular casts, few R.B.C., few epithelial cells, pH 7·1–8·2. Ammonia coefficient 3·2.

Tetany promptly relieved by Ca gluconate 5 c.c. 10% intramuscularly.

Blood chemistry: Serum calcium 10·6 mg.% immediately after injection: 4·2 mg.% later. Plasma α amino-nitrogen 5·69 mg.% (normal 4–6 mg.%). Plasma phosphates (inorganic) 3·2–5 mg.%. Plasma protein 6·8 mg.% (alb. 5·6; glob. 1·2). Plasma alk. phosphatase 11·3–12·2 King-Armstrong units. Blood chlorides 450 mg.% (as NaCl). Blood sugar (fasting) 75 mg.%. Blood urea 195 mg.%. Serum alkali reserve 38·5 vols. CO_2%. R.B.C. 2,950,000. Hb 52%. W.B.C. normal. Blood group A, Rh positive.

Urine: Cystine present. Benedict's reagent: faint reduction due to glucose, confirmed by paper chromatography. Sulkowitch test negative. Protein: heavy precipitate. Cyanide nitro-prusside: raised cystine. Chromatogram: Gross amino-aciduria, including cystine: Typical Fanconi pattern. Polarograph: Cystine (mg.) / Creatinine (grammes) = 340 (normal 30–45).

Slit lamp: Cystine crystals seen in corneæ, throughout substantia propria and also in conjunctivæ. Visible as a haze to the naked eye.

X-rays: Gross rickets, flattened heads of femora, no obvious osteoporosis, no calcified deposits in abdomen. IVP: failure to excrete dye, none in bladder even after thirty minutes. Cystogram: Bladder filled normally, dye did not enter ureters. Retrograde pyelogram normal. Cystoscopy: Normal ureters.

Treatment.—Ca gluconate intramuscularly for attack of laryngeal spasm nine days after admission. "Adexolin" ℥ xx b.d. Ca gluconate 25 grains b.d. Ferri ammon. cit. 6 grains t.d.s.

13.6.51: Transfused and put on 1–1½ tablespoons t.d.s. (50–75 c.c.) of Albright's mixture (citric acid 140 grammes, sod. cit. 98 grammes, water to 1 litre), low sodium and high potassium diet.

21.6.51: 600,000 units vitamin D_2 ("Sterogyl") intramuscularly. Ascorbic acid 1,000 mg. daily for seven weeks.

13.7.51: Tocopherol ("Ephynal") 100 mg. daily.

11.8.51: Discharged on Tocopherol, "Adexolin", iron and Albright's mixture.

19.9.51: Calciferol 100,000 units daily in place of "Adexolin".

Progress.—

Alkali reserve after four weeks of Albright's mixture 50 c.c. daily : 38·2 vol. CO_2%

Alkali reserve after two more weeks on 75 c.c. daily : 66·5 vol. CO_2%

Alkali reserve on 12.9.51 on 75 c.c. daily : 57·6 vol. CO_2%

Alkali reserve on 14.2.52 : 36·1 vol. CO_2%

Blood urea 26 mg.% (20.9.51); 44 mg.% (14.2.52).

1.8.51: X-ray knees and wrists—some improvement in rickets.

11.8.51: Amount of cystine in corneæ is less.

26.9.51: X-ray knees and wrists—rickets has improved further.

January 1952: Much less cystine in corneæ than previously.

13.2.52: X-ray wrists: Slight improvement in rachitic changes which, however, are still gross.

Case II.—G. J., girl, aged 5 years.

Past history.—Admitted to hospital at 2½ years for investigation of glycosuria, thirst, enuresis, increasing constipation. Noted as thin, pale, listless, with bossing of forehead. Hard fæces palpable on abdominal examination. Height 33 in. weight 22 lb. X-ray: Pituitary fossa normal. Bones not X-rayed.

Blood chemistry: "Lag" diabetic curve: fasting blood sugar 100 mg.% rising after 35 grammes glucose to 250 mg.% at one hour, falling to 110 mg.% at two and a half hours. Glycosuria nil with blood sugar 100, faint trace at 110, and present at 150 mg.% and over. No ketosis. Serum calcium 9·5 mg.%. Serum inorganic phosphorus 5·06 mg.%. Blood urea 61 mg.%. Blood chlorides 482 mg.% (as NaCl). Hb 68%: R.B.C. 3,750,000.

Urine: S.G. 1005, pH 6·8–8: albumin nil—light cloud, no casts.

June 1951: Aged 4½ years. First seen by me at Sydenham Children's Hospital as a result of the discovery that her brother (Case I) was suffering from Fanconi syndrome. Polyuria, night enuresis, thirst, constipation and lassitude had continued.

On examination.—Small stature (height 35 in., weight 26½ lb.), infantile behaviour, fair hair, pale, beading of ribs, enlarged epiphyses, frontal bossing, genu valgum, prominent abdomen, small umbilical hernia.

Cystine crystals (few) in corneæ and conjunctivæ.

Urine: Colourless. S.G. 1005, pH 6·9–7·3. Albumin trace. No casts.

Blood chemistry: Serum calcium 8·1 mg.%. Serum inorganic phosphorus 1·2 mg.%. Serum alkaline phosphatase 30·7 King-Armstrong units. Blood urea 58 mg.%. Blood chlorides 482 mg.% (as NaCl). Alkali reserve 45·8 vol. CO_2%. Plasma α amino-nitrogen 4·14 mg.%.

Urine: Sulkowitch test no precipitate. Paper chromatography: gross cystinuria and amino-aciduria. Cyanide nitro-prusside test: raised cystine. Cystine (mg.)/Creatinine (grammes) = 300 by polarograph.

X-ray: Gross rickets. The bone architecture of fully formed bone is normal.

Treatment.—June 1951: "Adexolin" ♏ xx b.d. X-rays (28.9.51)—rickets not improved.

October 1951: Calciferol 100,000 units daily, 1½ tablespoons t.d.s. (75 c.c.) of Albright's mixture. X-rays (13.2.52)—definite improvement in rachitic changes, which are still well marked.

Comment.—Fanconi syndrome is considered by Dent (1947, 1952) to be due to an inherited defect of reabsorption affecting primarily the proximal tubules of the kidney and involving amino acids, sugar and phosphates. He believes the defect to be inherited as a Mendelian recessive. The syndrome is characterized by dwarfism, gross rickets, hypophosphatæmia, amino-aciduria (including cystinuria), renal glycosuria, cystine deposits in body tissues, and acidosis. In spite of this acidosis, the urine is almost invariably alkaline to neutral. As renal failure ensues, the blood inorganic phosphates increase and, as a concomitant, the blood calcium falls. Hence, in Case I, tetany was the presenting symptom as in one of Fanconi's original cases (1936). Clinical examination showed rickets to be present and this was confirmed by X-ray. The next step was to exclude steatorrhœa and anomalies of the renal tract. Now that the Fanconi syndrome seemed likely Dr. Dent was approached. He clinched the diagnosis by paper chromatography and advised that the corneæ be examined for cystine crystals. It is of interest to note the normal plasma levels of α amino-nitrogen while gross amino-aciduria was present. I am greatly indebted to him for these and subsequent investigations and suggestions regarding treatment.

The sheet anchors of treatment in these cases have been vitamin D and Albright's citric acid—sod. citrate mixture which are well known to influence the bone changes and acidotic conditions respectively in the Fanconi syndrome, but is not yet known to have any material effect on the fatal outcome.

Vitamin D "Adexolin" ♏ xx b.d. for four months, together with the injection of 600,000 units of "Sterogyl", produced some improvement in the rickets in Case I, but "Adexolin" alone had hardly any effect in Case II. Calciferol 100,000 units daily for the past five months was given to both children without any ill-effects. In Case I the rickets improved a little more but in Case II the benefit was marked. These results seem to show that vitamin D must be given in very high dosage to be of any use.

Albright's mixture.—Treatment for six weeks corrected the acidemia in the boy but there is reason to believe that it was taken irregularly latterly with the result that his alkali reserve has fallen to its previous figure.

In view of the invariably fatal outcome expected in these cases, a new approach to treatment was felt necessary. Normally, cysteine is oxidized to cystine and the latter reduced to cysteine. On Dr. Dent's suggestion, ascorbic acid was tried in order to attempt to reduce the cystine deposits to cysteine, but later tocopherol (a biological anti-oxidant) was used to see if it could prevent cysteine from being oxidized to cystine.

Ascorbic acid.—1,000 mg. daily for seven weeks produced no appreciable improvement. For the last month of this treatment, "Ephynal" (tocopherol) was given as well: cystine deposits in corneæ were noted to be rather less than before.

Tocopherol ("Ephynal"—Racemic alpha-tocopherol acetate).—100 mg. daily. After six months much less cystine present in the eyes. In addition, the blood urea fell to 26 mg.% after ten weeks' treatment and now, even though acidemia has recurred, it is still only 44 mg.%. Part of this improvement may have been due to Albright's mixture but, in Dr. Dent's experience, the most this alone could do would be to halve the blood urea. The results so far achieved are biochemically gratifying but only further trials can decide whether tocopherol has a part to play in the treatment of Fanconi syndrome.

REFERENCES

DENT, C. E. (1947) *Biochem. J.*, **41**, 240.

—— (1952) *J. Bone Jt. Surg.*, **34B**, 266.

FANCONI, G. (1936) *Jb. Kinderheilk.*, **147**, 299.

Cat-Scratch Fever.—P. J. N. Cox, B.M., M.R.C.P. (for Professor Alan Moncrieff, C.B.E., M.D.).

L. H., girl, aged 8 years. Admitted 27.11.51.

History.—Two weeks ago onset of slight fever, a painless lump in the left groin, and two red spots on the left thigh. (Primary lesion.)

Three days ago developed itching red plaques on both shins. No chemotherapy had been given.

Examination on admission.—Not ill. One large soft left inguinal gland with some redness of the overlying skin. Two small painless red papules on the left thigh 4 in. above the knee and, in a similar position on the right thigh, a cat-scratch. Well-marked erythema nodosum on both shins.

Progress.—The erythema nodosum faded rapidly. The inguinal gland became fluctuant and thick greenish-yellow pus was aspirated twice in the first week. She ran a slight fever up to 99·6° F., without constitutional disturbance, for about two weeks and at the end of this period was discharged well, the inguinal gland enlarged but not fluctuant. No chemotherapy was given. During the subsequent two months she has remained well; pus has been aspirated from the gland on one further occasion, but it is now getting smaller.

Investigations.—W.B.C. 8,100, Neutros. 59%, lymphos. 41%. B.S.R. 21 mm. per hour (Westergren). Mantoux 1/100 negative. Chest X-ray normal.

Pus from gland: No organisms seen or cultured. No acid-fast bacilli or virus elementary bodies seen. Inoculation of guinea-pig and other animals, negative (Dr. J. A. Dudgeon).

Biopsy of primary lesion on thigh: A largely monocytic granuloma with some giant cells, but the picture is not that of tuberculosis.

Frei test negative.

Complement-fixation reaction for lymphogranuloma negative.

Skin test for cat-scratch fever (Mollaret, 1950) positive. An antigen prepared from this child has given positive skin reactions in other similar cases.

Comment.—Cases of cat-scratch fever have been reported with increasing frequency in the last two years, chiefly in France and America: this is the first report of a case occurring in England. It is a benign disease characterized by mild constitutional disturbance and gross localized enlargement of lymph nodes, which frequently suppurate; there is usually a small primary skin lesion, commonly originating from a cat scratch. The cat is unaffected by the disease and is presumed to be a passive carrier of the infective agent; this has not yet been isolated, but it has been suggested that it may be a virus of the lymphogranuloma-psittacosis group.

The case described here shows the typical features of the disease. The occurrence of erythema nodosum is uncommon, but has been described before. The condition is probably quite common in this country and is most likely to be mistaken for tuberculosis, or a simple pyogenic adenitis, in which the pus has been sterilized by chemotherapy.

REFERENCE

Mollaret, P. (1950) *Pr. méd.*, **58**, 1353.

Congenital Partial Heart Block.—H. G. Dunn, M.R.C.P.

S. T., a girl, aged 13 months, is the second child of healthy parents. The mother was well during both pregnancies, and the first child is normal.

Nine days before the second delivery, irregularity of the fœtal heart rhythm was noted and dropped beats were demonstrated by phonocardiogram (Fig. 1).

The fœtal heart continued to beat irregularly throughout labour. The infant was delivered normally and appeared healthy. A soft systolic murmur, audible from the cardiac apex to the left lower sternal border, was noted and was best heard after dropped beats.

At the age of 4 and 8 days, electrocardiograms (ECG) showed a variable degree of partial atrio-ventricular block with Wenckebach periods; the P–R interval ranged from 0·20 to 0·28 second (Figs. 2 and 3). At 12 days, the ECG demonstrated 3 : 2 and 4 : 3 block at an auricular rate of 155 to 160 per minute. After a crying attack this rate rose to 215 and all beats were conducted, the P–R interval being 0·22 second. Again, twenty-five minutes after a subcutaneous injection of atropine 1/200 grain the auricular rate was 200 per minute and all beats were conducted, with a P–R interval of 0·23 second. Chest X-rays and screening with barium swallow showed nothing abnormal.

At the age of 1 month, the ECG showed 2 : 1 and 3 : 2 block at an auricular rate of 155 to 170 per minute. Compression of the left carotid sinus caused auricular slowing to 115 per minute with dropped beats after every two to five ventricular responses and finally sino-auricular block with auriculo-ventricular nodal escape.

During the subsequent two months, the heart showed variable degrees of block. At the age of 3 months it was beating regularly at a rate of 70 per minute, and the ECG demonstrated 2 : 1 block with a P–R interval of 0·26 second.

At nearly 5 months, 2 : 1 block was again shown electrocardiographically and now proved persistent thirty minutes after a subcutaneous injection of 1/100 grain of atropine, although the auricular rate rose from 140 to 194 per minute and the P–R interval was reduced from 0·28 to 0·26 second.

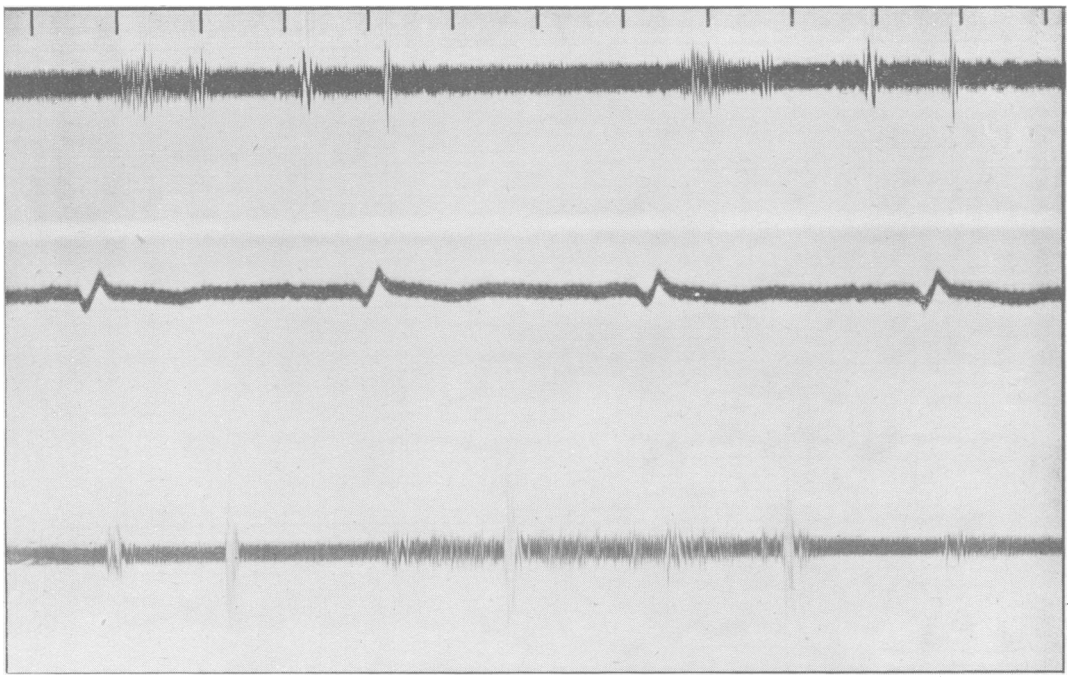

FIG. 1.—Cardiograms taken nine days before delivery. Upper tracing: fœtal phonocardiogram (medium frequency) recorded from the mother's abdomen. Note dropped beat (probably due to 3 : 2 heart block) and systolic murmur during subsequent contraction. Middle tracing: electrocardiogram from a lead connecting fundus uteri and mother's left leg. Lower tracing: maternal phonocardiogram (high frequency) recorded from pulmonary area. The main excursions in the electrocardiogram are maternal and coincide with the mother's first heart sounds. Time marker = 0·2 second.

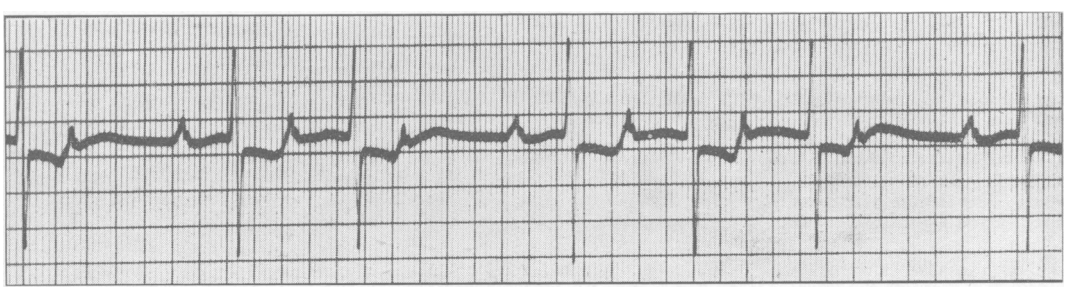

FIG. 2.—Electrocardiogram (lead CR_1) taken four days after birth and showing 3 : 2 and 4 : 3 heart block with Wenckebach periods. P–R interval = 0·20 to 0·26 second.

The child has developed normally and looks healthy. Her pulse-rate is usually about 70 per minute. B.P. 115/65. The systolic murmur remains soft, is now best heard at the apex and is not diagnostic of any congenital lesion. Slight general cardiac enlargement can be demonstrated radiologically but could be explained purely by the bradycardia. The ECG shows persistent 2 : 1 block and the P–R interval has now lengthened to 0·30 second.

Comment.—Congenital partial heart block is rarer than congenital complete block, but a complete block may sometimes develop from a partial one. The progressive increase in the degree of block in the present case is of interest in this connexion.

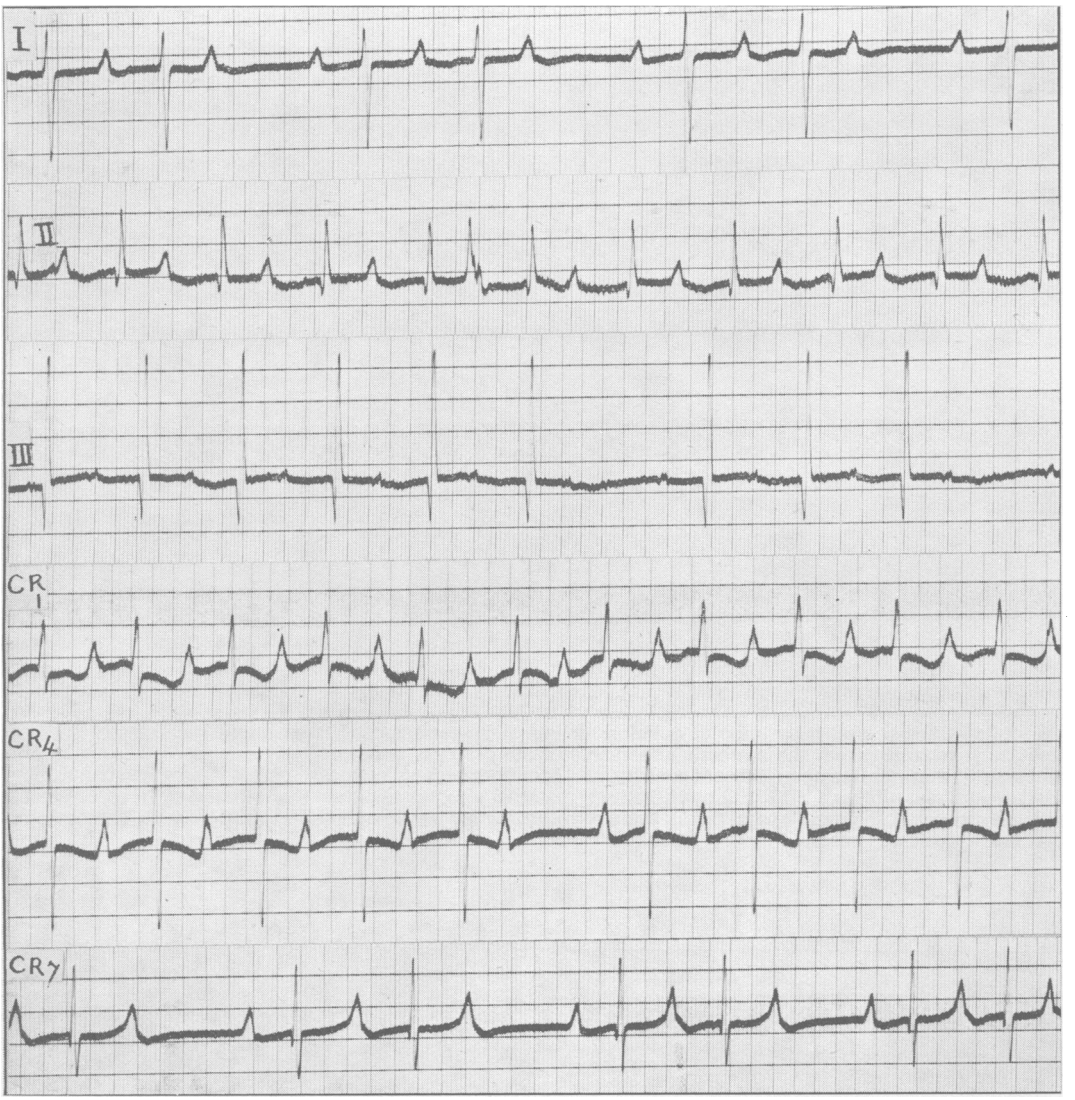

FIG. 3.—Electrocardiograms (limb and chest leads) taken eight days after birth and showing partial heart block. The degree of conduction defect varies from only prolonged P–R interval (leads II and CR_1) to 3 : 2 block (leads I and CR_7). Note Wenckebach periods. P–R interval = 0·22 to 0·28 second.

In a few recorded cases congenital complete heart block has been demonstrated phonocardiographically before birth. The present appears to be the first instance of a partial block thus documented.

I am grateful to Dr. K. H. Tallerman for permission to publish this case and to Dr. William Evans for allowing me to reproduce the cardiograms, which were recorded by the Staff of the Cardiac Department of the London Hospital.