Neill) has been shown to be less than that calculated by the factor 1.34 c.cm. O₂/1 g. Hb, suggesting that the haemoglobin dissociation curve has been shifted "to the right." This is in direct contrast to the work of Valtis and Kennedy (1954), who demonstrated a shift to the left in stored blood, and of Van Slyke *et al.* (1946), who showed that "inactive" haemoglobin in freshly drawn blood disappeared after standing at room temperature for two hours.

Variables such as these cannot be predicted before the start of perfusion, and, once perfusion has begun, cannot be readily influenced. They are among the important limiting factors of the effectiveness of the perfusion.

While not firmly established, evidence is accumulating that high partial pressures of oxygen in arterial blood exert a deleterious effect on the function of cerebral tissues and to a less extent on other tissues. Dickens (1946) and Mann and Quastel (1946) have demonstrated a toxic effect on cerebral aerobic glycogen metabolism; Donald (1947) and Lambertsen *et al.* (1953a) showed a deterioration of cerebral function; and Lambertsen *et al.* (1953b) and Clark *et al.* (1953) showed an effect on cerebral blood-flow. Kirklin (1958) has indicated that this is the cause of otherwise unexplained post-perfusion deaths. All the evidence is derived from experiments in which a higher partial pressure of oxygen has been used than can be obtained in our machine, but Rennie and Pappenheimer (1958) have shown that, despite very high ambient pressures, the alveolar pO₂ always exceeds that of arterial blood.

In addition, there are the theoretical considerations of micro-bubble formation—discussed by Gibbon (1959)—and interference with carbon-dioxide transport (Peters and Van Slyke, 1931). We have found, too, that with disk speeds in excess of 130 there is a tendency for the blood to foam in the oxygenator and for small bubbles to appear in the blood leaving the oxygenator.

The undesirability of a low content of arterial blood oxygen, though not well documented, is generally accepted, leading, amongst other things, to a decrease in circulating blood pH and an increase in blood acidemia (Biddulph *et al.*, 1958; Huckabee, 1958; and Magovern *et al.*, 1959). Tobian, Martin, and Eilers (1959) have shown that one of the effects of decreasing pH is to decrease the pressor action of noradrenaline.

If it is agreed that control of the degree of blood oxygenation leaving an artificial heart-lung machine is necessary, then we are sure that the polarographic method is the method of choice—the advantages being low cost, accuracy, and simplicity.

It has been suggested that oximetry could be used. In the range of haemoglobin oxygen saturation levels with which we are concerned, the oximeter is inaccurate owing to the flattening of the haemoglobin dissociation curve; it gives no information about pO₂ level above those necessary to cause 100% haemoglobin oxygen saturation (Nahas *et al.* (1952) has shown this to occur at 400 mm. Hg pO₂); it is expensive and cannot be readily adapted for continuous monitoring.

Summary and Conclusions

The reasons for the introduction of a monitoring device of the level of blood oxygen content leaving the heart-lung machine are discussed, the literature is briefly reviewed, the method of use of the Clark oxygen electrode is described, and some results are presented.

We would stress that the polarograph electrode circuit is simple and very reliable, the electrode is easily and

quickly calibrated, and the pump operator can at all times determine if the oxygenator is acting as an adequate respirator.

The platinum oxygen electrode is as supplied by Yellow Spring Instruments Co. Inc., Yellow Springs, Ohio. The galvanometer is a Pye Scalamp Type 7904/3, and the associated circuit was made from standard radio components in our electronics workshop.

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AETIOLOGY OF CONGENITAL TALIPES

BY

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The frequent occurrence of malformations in association with congenital talipes is well known. Attention has often been drawn to this fact. One of the more comprehensive reviews of the subject is by Ehrenfried (1912), who reviewed 232 cases of congenital talipes, with an incidence of associated anomalies of 13.4%. The anomalies included such conditions as congenital dislocation of the hip, mental deficiency, club-hand, cleft palate, amniotic bands, congenital herniae, and scoliosis. He ascribed the cause to heredity in those cases with a strong family history (about 10%), faulty nutrition of the zygote due to chorionic abnormalities, and mechanical distortion of the foetus.

Waller (1923) reviewed 735 cases of talipes and found a 4% incidence of associated anomalies. He reported that anomalies occurred twice as frequently in associa-

tion with talipes calcaneo-valgus as with talipes equinovarus. Keith (1940), in discussing "foetal dysplasia," described, amongst other foetal anomalies, cases with amniotic bands, intrauterine amputations, and talipes. He concluded that talipes was due to an area of mesenchymal dysplasia on the medial aspect of the foot, producing a cicatricial contracture which in turn produced the deformity.

Record and Edwards (1958), writing on the aetiology of congenital dislocation of the hip, reported an incidence of associated anomalies of 16%. The anomalies consisted of congenital herniae, hare-lip, spasticity, mental deficiency, and talipes (about 4%).

A similar review of sacral agenesis by Blumel, Burke Evans, and Eggers (1959) revealed anomalies ranging from arthrogryposis multiplex congenita to recto-vaginal fistulae in more than 90% of 50 patients exhibiting sacral agenesis. Congenital talipes occurred in 56% of these patients.

The present review was undertaken to ascertain the incidence of other congenital anomalies occurring in association with talipes in this clinic and to assess the significance of the findings in relation to the aetiology of congenital talipes.

Material

The report deals with 172 patients with congenital talipes attending the talipes clinic of this department and a review of the diseases occurring in the mothers of these children during the relevant pregnancy. All the patients are attending regularly and will continue to attend until maximum correction has been obtained and maintained. The methods of treatment follow the usually accepted principles of manipulation, splintage with Denis Browne splints, callipers, boots, and corrective surgery where indicated.

Of the 172 patients 40 (23.2%) have other congenital lesions (Table I). The anomalies (Table II) are of many varieties. Nine of the patients had siblings also included in the series, so the mothers numbered 167, all of whom were either interviewed personally or circularized. Of the 167 mothers 41 (25.1%) could not be interviewed or failed to return the circulars, but an accurate medical and family history was obtained from the remainder

TABLE I

Number of cases	172
C.T.E.V.	164
C.T.C.V.	8
C.T.E.V. with associated anomalies	40
C.T.C.V.	0
Incidence of other anomalies	23.2%
Family history of talipes	12.8%

TABLE II.—40 Associated Anomalies in this Series

Congenital herniae	3
Spina bifida occulta	6
" " with neurological damage	2
Dysplasia of hip	1
Microcephaly	1
Arthrogryposis multiplex congenita	1
Spastic (one had pyloric stenosis)	3
Congenital dislocation of hip	2
Sacral agenesis	1
Multiple anomalies (see Table VI)	3
Arachnodactyly	1
Congenital absence of radius	1
Cleft palate and hare-lip	1
Mentally retarded	2
Achondroplasia	2
Growth defect lower tibial epiphysis (with short legs and varus deformity)	2
Torticollis	1
Pyloric stenosis (including one spastic)	3
Monostotic fibrous dysplasia	1
Amniotic bands on legs, with congenital amputation of toes	3
Webbed toes	1

(126). Disease which may be significant occurred in 27 (21.4%); the diseases are tabulated in Table III.

Spence *et al.* (1954) quote an incidence of congenital anomalies of 1.5% in 1,142 infants. This figure represents the incidence of all major anomalies in a fairly representative selection of children in Newcastle upon Tyne, and corresponds fairly closely to the figure of 0.9% quoted by Fox *et al.* (1948) as being the incidence of all anomalies in the population as a whole. The anomalies occurring in Spence's survey are set out in Table IV.

TABLE III.—Diseases Occurring in 27 Mothers During Pregnancy

1. Toxaemia of pregnancy arising in the 3rd trimester of pregnancy; children had uncomplicated talipes	5
2. Hyperemesis gravidarum arising in the 1st trimester of pregnancy; 4 children had uncomplicated talipes, 1 child had amniotic bands and talipes	1
3. Mumps during the 1st trimester; child had achondroplasia	1
4. Anaemia, probably existed prior to pregnancy but treated during 2nd and 3rd trimesters; 4 children had uncomplicated talipes, 1 child had congenital absence of radius	5
5. Pulmonary tuberculosis diagnosed during 3rd trimester; children had uncomplicated talipes	2
6. Rubella occurring at 7 weeks; child had arachnodactyly and talipes	1
7. Congestive heart failure, probably arose during 2nd trimester; child had uncomplicated talipes	1
8. Diabetes mellitus (treated throughout pregnancy with insulin), also developed jaundice of unknown aetiology in 2nd trimester; child had uncomplicated talipes	1
9. Menstruation continued up to 6 months; child had uncomplicated talipes	1
10. Recurrent abortions; 1 child had talipes and torticollis, 1 child had uncomplicated talipes	2
11. Unknown eye lesion which necessitated many investigations, including lumbar puncture; child had uncomplicated talipes	1
12. Mother died in labour (thought to be due to a cardiac lesion); child had uncomplicated talipes	1
13. Generally unwell throughout pregnancy; child had uncomplicated talipes	1

TABLE IV.—17 Anomalies Occurring in Spence's Survey

Mongolian idiocy	4
Congenital heart disease (including 2 of the mongols)	4
Digital anomalies	4
Talipes	3
Spina bifida and hydrocephalus	1
Microcephaly	1
Cleft palate	1
Branchial cyst	1

Of the 40 patients showing other anomalies in the present series it was felt that many should be excluded from a statistical analysis. For example, the cases of pyloric stenosis, torticollis, and monostotic fibrous dysplasia are excluded as being of little or no significance in the present context. The six cases with spina bifida occulta are excluded because the incidence in the 40 patients (17.5%) is not in excess of the incidence (20%) one would expect in any 40 radiographs of the spine (Schinz *et al.*, 1952). A further eight cases, including the spastics, the patients with neurological damage secondary to spina bifida, the achondroplastics, and the patient with arthrogryposis, are excluded as the talipes in these patients is secondary to the major anomaly. The two patients with an abnormality of the lower tibial epiphysis are excluded because this condition may be the result of overenthusiastic manipulation, but it is strange that they are brother and sister.

The final selection of cases with associated anomalies is given in Table V; there are 20 cases, an incidence of 11.6%. Details of three cases with multiple anomalies are given in Table VI.

TABLE V.—20 Severe Anomalies in Present Series

Microcephaly	1	Congenital absence of radius	1
Congenital herniae	3	Cleft palate and hare-lip	1
" " dislocation of hip	2	Amniotic bands	3
Sacral agenesis	1	Dysplasia of hip	1
Multiple anomalies (see Table VI)	3	Mentally retarded	2
Arachnodactyly	1	Webbed toes	1

TABLE VI.—Three Cases Showing Multiple Anomalies in Present Series

1. Klippel-Feil syndrome. Spina bifida and meningocele, C.D.H., and congenital scoliosis
2. Hemivertebra L5 and sacral anomaly; ventricular septal defect
3. Multiple upper-limb and chest anomalies; subsequently died at 8 weeks

Discussion

Browne (1936) evokes mechanical compression of the foetus as a factor in the production of such anomalies as talipes, spina bifida, congenital dislocation of the hip, and pseudarthrosis of the tibia. The evidence produced is certainly impressive and could account for the association of these conditions with talipes, but it could not account for the significantly high association of such anomalies as arachnodactyly, cleft palate, webbed toes, and sacral agenesis, which are probably faults in mesenchymal differentiation.

Heredity as a factor in the aetiology of talipes is accepted, but is operative in no more than 10% of all cases. It is felt that an incidence of other congenital anomalies of 11.6%, combined with the fact that the anticipated incidence of most of the anomalies found in this series is less than one per thousand, points to some common factor, or factors, in the production of talipes and other congenital anomalies. If the incidence of anomalies occurring in this series is compared with the incidence of congenital anomalies in Spence's review there is an 8 to 1 increase in the present series (Table VII).

TABLE VII

	Spence	Present Series	Total
Normal	1,125	152	1,277
Abnormal	17	20	37
Total	1,142	172	1,314

Fourfold table formula: $\frac{(19,916)^2 \times 1,314}{1,142 \times 172 \times 1,277 \times 37}$
 $P < 0.01$; $\chi^2 = 56.14$.

One of the arguments advanced against developmental arrest in the production of talipes is that the deformity is unlike any position normally adopted by the foetal foot. This is not in accordance with Keith (1948), who states that the foetal foot is inverted up to the seventh month of foetal life. This can readily be confirmed by observing the position of the feet in any aborted foetus. Further evidence of the existence of such a position in the developmental history of the foot is furnished by the alteration in the angle of the neck and distal articular facet of the talus between foetal and adult life (Wood-Jones, 1949). In addition, Böhm (1929) described several stages in early development (two to three months), during which the foot adopts positions of equinus, supination, and adduction.

If it is accepted that arrested development of the foot could produce the characteristic deformity of club-foot a search for the factors which might produce developmental arrest is necessary.

Warkany (1943, 1947) and Warkany *et al.* (1943) produced foot deformities in young rats by the production of deficiencies of vitamins A and D and riboflavin in pregnant rats. Duraiswami (1950, 1952, 1955) produced defects, including foot defects, in more than 70% of chick embryos by the injection of a variety of substances such as insulin, lead, and thallium nitrate into the yolk sac of fertilized hen's eggs. The interesting feature of the latter experiments was that it was found that there was an element of "time specificity" in that it was possible to reproduce consistently the same

deformity according to the stage of development reached when the injection was made. For example, foot defects could be produced by insulin when the injection was made on the third day of incubation, whereas other defects, such as hip dislocation, could be produced by insulin injections made about the sixth day of incubation.

Similar results have been obtained with chick embryos inoculated with virus, although in these experiments early death of the embryo resulted and the malformations were very gross. The malformations in these experiments were largely restricted to the optic and otic capsules and the spinal column. Blattner and Williamson (1951) used Newcastle disease virus and Heath *et al.* (1956) used influenza, herpes simplex, and vaccinia virus in the production of the lesions.

Although it is dangerous to draw analogies between the production of experimental lesions in animals and the effects of disease in man, it seems reasonable to compare these experiments with congenital lesions in man known to be associated with interference in early development as a result of maternal infection and apparently similar lesions occurring in association with maternal disease such as diabetes mellitus. Gregg (1941) and Swan *et al.* (1943) drew attention to the association between congenital cataract and maternal rubella, and the occurrence of congenital defects of many types in association with maternal virus infections such as mumps has been reported by other observers (Fox *et al.*, 1948).

Maternal diabetes is known to be associated with a high incidence of abortion, and about 6% of children born of diabetic mothers exhibit various anomalies. In a review of 50 patients with sacral agenesis mentioned earlier, Blumel *et al.* record diabetes in 14% of the mothers. Only one mother with diabetes was found in the present series, but other conditions were also found to have existed during the relevant pregnancies. Some of these diseases, such as rubella and mumps, have been incriminated as aetiological factors in the production of congenital lesions, while others, such as hyperemesis gravidarum, pulmonary tuberculosis, congestive heart failure, and anaemia, have an unknown relationship to the production of congenital lesions.

It is impossible to draw any definite conclusions with regard to the part played by most of the maternal diseases mentioned in Table III in the aetiology of talipes, but it is felt that surveys conducted throughout pregnancy, rather than in retrospect, would prove helpful in indicating the underlying causative lesion in a high percentage of cases with congenital talipes.

Conclusions

The high incidence of other congenital anomalies occurring in association with talipes (11.6%) is thought to indicate a developmental aetiology in the production of talipes.

The incidence of maternal disease found in this series (21.4%) suggests that this is a factor of aetiological significance.

The final elucidation of the problem will require large-scale investigation of the diseases occurring during pregnancy and the correlation of the findings with the occurrence of congenital talipes.

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- 1950; Stroebel *et al.*, 1951; Lawrence *et al.*, 1953; Gros, 1955; Moe, 1956; Herbeuval *et al.*, 1957; Sjöberg, 1957; Damon *et al.*, 1958; Frey, 1958; Lawrence and Donald, 1959; Omland, 1959), to which may be added a few cases stated to have been reported as "personal communications." An excellent review, adding also 10 new cases, was published by Damon *et al.* (1958).

Recently we had occasion to examine a case of coexisting hypernephroma and polycythaemia, which is reported in some detail below.

Case Report

A married farmer aged 60 was admitted to hospital on March 31, 1959. At the age of 16 he had scarlet fever, at 18 jaundice, at 22 "Spanish 'flu," and at 35 "gastric ulcer." For many years he had had a left-sided scrotal hernia. During the past 18 months he had been bothered by persistent pressure-pain below the left costal border; and during the past six months he had periodical haematuria, a tendency to sweat, and occasional oppression below the right costal margin. During the same period he had lost 10 kg. No headache, dizziness, or visual disturbances had occurred, no thrombotic episodes or haemorrhages apart from the haematuria, and no symptoms from other organs. Investigation about three weeks before admission had shown a haemoglobin level of 138–120%, R.B.C. 6,500,000/c.mm.; and W.B.C. 12,000/c.mm.

Findings on Examination

Polycythaemic facies; no dyspnoea at rest; no goitre or enlargement of lymph nodes; auscultation of lungs and heart showed no abnormality; spleen enlarged to the umbilicus; liver and kidneys impalpable; left-sided varicocele; prostate normal. Weight, 72.5 kg.; height, 172 cm.; blood-pressure, 140/85 to 125/75 mm. Hg.

Urine.—Protein, 1 plus to 0; no sugar or blood. Daily output, 470–1,020 ml. (specific gravity, 1016). Microscopical examination: 1 plus protein and 5–6 red cells when first studied; no abnormality the second time; no tumour cells on centrifugation, and no growth on culture.

Erythrocyte sedimentation rate, 3 mm./hour; Hb, 18.1 g./100 ml.; R.B.C., 6,580,000/c.mm.; colour index, 0.90; volume index, 1.00; W.B.C., 8,300/c.mm. (80% neutrophils, 3% eosinophils, 1% basophils, 9% lymphocytes, and 7% monocytes). Red blood picture normal. Platelets 260,000/c.mm.; prothrombin-proconvertin, 100%. The serological test for syphilis was negative. Blood group A Rhesus D negative (cde). Plasma volume, 3,750 ml. (determined with J-131-albumin (I.H.S.A., Amersham) by Dr. T. Munkner); haematocrit, 59 vols.%; total quantity of blood, 9,150 ml. (normal value for his height and weight, 4,900 ml.). Serum bilirubin, 0.9 mg./100 ml.; thymol, 0.03 unit, alkaline phosphatase, 16.4 units (normally 3–10 units); serum creatinine, 1.7–1.3 mg./100 ml.; serum calcium, 11 mg./100 ml.; serum phosphorus, 4.2 mg./100 ml.; serum uric acid, 6.4 mg./100 ml. Basal metabolic rate, +24%; pulse, 68. E.C.G.: left axis deviation, but no other abnormalities.

An intravenous pyelogram showed the right kidney to be normal, the left one somewhat elevated. The ureter was displaced slightly to the medial aspect. There was a suspicion of tumour of the left kidney. Retrograde left-sided pyelography revealed the ureter extending in an arch around a soft-tissue shadow in the left flank, and the pelvis situated at the upper pole of a mass extending to two centimetres distal to the iliac crest.

X-ray of chest: Heart normal. At the sternal end of the right first rib was a shadow, 3 by 1.5 cm., closely connected with the first rib. ?Ecchondroma.

Cystoscopy showed no abnormality.

Diagnosis: hypernephroma of left kidney, secondary polycythaemia.

Operation.—Left-sided nephrectomy was carried out on April 11 (Professor Dahl-Iversen). The tumour was 10 cm.

HYPERNEPHROMA AND
POLYCYTHAEMIA

BY

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The coexistence of hypernephroma and polycythaemia was reported for the first time in 1929 by Bliss, who described the presence of hypernephroma in a patient with polycythaemia as "an incidental complication."

The possibility of a direct causal relationship between hypernephroma and polycythaemia was suggested for the first time by Forssell (1946) and shortly after by Videbaek (1950). Both have described a case of hypernephroma with secondary polycythaemia which regressed after nephrectomy. Similar cases have subsequently been reported by Gros (1955), Conley *et al.* (1957), Damon *et al.* (1958), Campbell *et al.* (1958), Frey (1958), and Omland (1959).

Incidentally, the occurrence of hypernephroma and polycythaemia in the same patient is not particularly uncommon. The frequency of hypernephroma in patients with polycythaemia has been reported to be as follows: Videbaek (1950) found 2.4% in 125 patients, Lawrence (1955) 1.3% in 157 patients, and Damon and Holub (1958) 4.4% in 225 patients. The frequency of polycythaemia in patients with hypernephroma is, according to Damon *et al.* (1958), 2.6% (350 patients) and according to Berger and Sinkoff (1959) 1.8% (273 patients). A total of 41 cases of hypernephroma-polycythaemia have been reported in the accessible literature (Medvei, 1934; Fairley, 1945; Forssell, 1946, 1947, 1954, 1956, 1958; Lawrence, 1949; Videbaek,