apparently extending to the apex of the space. Dilatation of the superficial veins was present in the brachial and pectoral regions and antero-lateral chest wall. At no period was there any pain or tenderness, and the brachial and radial pulses were normal. A full blood count, including an estimation of the platelets, revealed no abnormality. The Wassermann reaction was negative, and an x-ray of the chest and the root of the neck showed no unusual feature. He was treated by rest, elevation, and later by massage. During the weeks that followed, the dilatation of the superficial veins steadily increased as the swelling subsided. At the same time the thrombosed axillary vein became less defined and less indurated. At the time of writing—two months since the onset—the swelling has almost disappeared, but the skin still betrays a slightly cyanotic tint. There is marked dilatation of the superficial veins in the pectoral region, and the axillary vein can still just be defined. As far as possible, it would appear that trauma and strain can be excluded in this case. Although his occupation was that of a furniture remover, for five days prior to the onset of the swelling he had been enjoying his annual vacation. During these five days he had not indulged in sport or effort of any description.

DISCUSSION

It is the causation of this condition which has excited so much interest, particularly on the Continent. Among the recorded cases there are extremely few in which trauma and strain can be excluded. So much is this so that the condition is frequently known as "Phlébite par effort" (Cadenat). Pellot⁷ records a case without apparent injury, and two patients awoke in the morning to find their arms swollen (Ruge and Wilson). The present case appeared worth recording in view of the fact that in so few cases did the onset of swelling occur during sleep.

The various theories as to the causation may be summarized thus:

Cadenat.—A venous dilatation and stasis due to an expiratory effort, combined with single or repeated muscular strains, injuring the intima of the venous tributaries at their junction with the axillary vein.

*Lowenstein.*⁸—A venous dilatation and stasis due to an expiratory effort, combined with marked abduction of the arm. The latter movement produces indentation of the axillary vein by the costo-coracoid ligament. In Lowenstein's view this effect is increased by the action of the subclavius muscle.

*Pearce Gould and Patey.*⁹—A sudden contraction of the subclavius muscle tears the delicate subclavio-axillary valve in the long axis of the vein. This is more likely to occur during a marked expiratory effort causing venous dilatation and stasis, and with abduction of the arm.

Wilson.—Some specific lesion of the venous intima. One of his cases was reputed to be syphilitic, while the other died of advanced phthisis. The former case, however, had repeated negative Wassermann reactions, while in the latter the brachial artery was also occluded.

Earlier theories are those of Willan, who suggested that the axillary vein suffered damage against the outer border of the first rib during vigorous exercise with the arm in the abducted position, and of Ruge, who considered that the thrombosis of the axillary vein might be the first symptom of latent heart disease. There appears to be little or no evidence to support these contentions.

What the causation may be in these isolated cases, in which traumatism can, as far as possible, be excluded, remains unsolved.

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ACUTE HAEMOLYTIC ANAEMIA IN ADOLESCENCE

BY

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The following case exemplifies a type of acute haemolytic anaemia, occurring in adolescence, which has been recorded from time to time of late years. Dr. L. J. Witts, in the Goulstonian Lectures of last year, focused attention upon this class of case in an illuminating way, and, by differentiation, clarified the diagnosis of various anaemias in a manner most helpful to the general practitioner. It seems probable that the type referred to is not so rare as was supposed, and that improved methods now in use of staining, recognizing, and interpreting embryonic forms of red cells will help to bring this type of acute haemolytic anaemia to identification at an early stage, and so reduce its present high mortality.

CASE RECORD

D. B. S., a secondary school boy, aged 17, felt well and appeared to be in good health till July 24th, 1932. On that day, while in chapel, he felt slightly giddy and weak. Next day, in school, he felt shivery, with headache and lassitude. On the following day he went to bed with a temperature of 103° F. and a severe headache. He looked pale, vomited, and was thought to have influenza. Medical advice was sought on July 27th, when the following symptoms were pronounced: headache, pyrexia, restlessness, occasional vomiting, and faintness. There were no physical signs of illness in the throat, chest, or alimentary system. The urine and cerebro-spinal system were normal. There was an unusual pallor. Next day these symptoms were accentuated, and the patient was removed to a private hospital. The pallor was more noticeable, he felt faint on sitting up, and was unable to walk or stand unsupported. The maximum temperature was 104°, pulse 135, and respirations 26. During the same evening there was a copious normal movement of the bowels, and vomiting ceased from that time.

On the 29th the temperature remained normal and the patient felt better; but on the following day his condition relapsed, the temperature again rose, and remained between 100° and 102° for several subsequent days. The boy was now extremely pale and restless, and a blood count disclosed a high degree of anaemia, with 1,200,000 erythrocytes, 40 per cent. haemoglobin, and a colour index of 1.6. Next day the red cells were down to 1,040,000 and haemoglobin to 35 per cent., colour index 1.7. For the next two or three days he was incontinent of urine, delirious, and extremely restless. Vomiting had entirely ceased, however, and he was retaining milk-and-soda, orange juice and glucose, and liver both in the form of extract (four phials per day, each estimated as equivalent to three and a half ounces of raw liver) and also three ounces daily of fresh raw liver. On August 1st, while he was at his worst, transfusion of 350 c.cm. of blood was performed. Next day the restlessness was decidedly better and his mental condition had improved. On August 3rd continence returned, and there was an improvement in general condition. This improvement was more pronounced on the following day, when the temperature was down to normal, the pulse to 80, colour was returning, and appetite had reappeared. Red cells were now 1,280,000, haemoglobin 45 per cent., and colour index 1.7; megaloblasts had appeared in the film, and nucleated red cells were very numerous.

From this time on a flowing tide of recovery set in without remission. By August 9th his appetite was voracious, and he was eating freely of red meats, vegetables, puddings, and fruit juices, in addition to two phials of liver extract and three ounces of raw liver taken in hot soup. During the subsequent three weeks the improvement continued steadily in every respect, while the patient manifested the euphoria and *joie de vivre* so characteristic, especially in youth, of convalescence from severe illness.

The successive red count during the recovery was as follows:

Date	Red Cells	Haemoglobin	Colour Index
		Per cent.	
30/7/32	1,040,000	35	1.7
4/8/32	1,280,000	45	1.7
9/8/32	2,150,000	60	1.4
17/8/32	3,736,000	75	1.02
22/8/32	4,240,000	80	0.95
30/8/32	4,424,000	80	0.90
23/9/32	4,990,000	85	0.85

The regenerative nature of the anaemia became very clear as time went on. In the first blood examinations on July 29th and 30th there was slight variation in the size of the red cells, and there were a few poikilocytes. Unusual activity of the haemopoietic system was manifest on August 4th—three days after the transfusion and six days after the administration of liver had begun. The laboratory report then was:

"Anisocytosis very marked, with a big percentage of macrocytes. Polychromatophilic cells dominate the picture, being of a deep colour. Much punctate basophilia, both fine and coarse granules. Nucleated red cells very numerous. Normoblasts, young, old and degenerated (Howell-Jolly bodies), are to be seen to the extent of two or three in every field. Two megalocytes."

On August 13th the report stated that the film showed a very large number of nucleated red cells and macro-normoblasts—"megalocytes abundant and anisocytosis very marked. The whole picture is typical of acute pernicious anaemia." It was not till August 18th that vital staining was employed, and reticulocytosis was demonstrated.

From first to last in this case no septic focus could be discovered to explain the early symptoms, and no growth was obtained from the blood. The differential count from July 29th to August 30th showed an average of 9,600 whites, with a maximum of 16,550 on the seventh day. With the exception of the first count, when the proportion of polymorphs was 32 to 45 lymphocytes, the average proportion of polymorphs to lymphocytes was 64 to 25. A gastric analysis made during convalescence, after the administration of hydrochloric acid had been stopped for two days, showed a normal acidity. The medicinal treatment of the case, which was begun as soon as vomiting had ceased, and before the nature of the anaemia was diagnosed, consisted of a mixture containing ferri et ammon. citrat. xxx grains, ac. hydrochlor. dil., m xx, glycerin of pepsin one drachm, three times daily; in addition, liver treatment was persisted in as detailed above. When the physical and mental restlessness was at the worst sodium amytal, also morphine and atropine, proved helpful.

There seems good reason to regard the transfusion in this case as the most important factor in the remarkable recovery from what appeared to be a desperate condition. At the same time the persistent stimulus to activity of the bone marrow produced by the daily ingestion of liver in quantity must be regarded as a highly important contributing factor. In handling the case I was much indebted to Dr. Carmalt-Jones, professor of systematic medicine, Otago University, who gave me much helpful assistance in diagnosis and treatment, and drew my attention to Dr. Witts's Goulstonian Lectures of last year. It remains to emphasize, in the recognition of similar examples of this unusual type of acute anaemia, the youth of the patient, the sudden onset, and the presence of a normal gastric acidity; and, in the treatment, the great value of transfusion.

Memoranda

MEDICAL, SURGICAL, OBSTETRICAL

SYPHILITIC NEPHRITIS

Syphilis can affect most—if not all—organs of the body, no part being immune; yet one rarely considers syphilis when dealing with kidney infection. The case described below is one of acute secondary syphilitic nephritis. This is a rare manifestation in the acute eruptive stage. Not many cases have been recorded—Fournier, in his *Traité de Syphilis*, collected 26—and only a few have been in the literature of late. The last account of this condition, by G. B. Dowling, appeared in the *Guy's Hospital Reports* of 1927 (July and October).

CASE RECORD

The patient, a man aged 44, gave a history of herpes of the penis during the first week in January, 1933. The primary chancre was no doubt associated with this condition. A secondary rash appeared on February 12th, and was confirmed by a positive Wassermann reaction in the blood. His medical attendant tested the urine before starting anti-syphilitic treatment. No albumin or any other abnormality was detected. On February 16th 0.45 gram N.A.B. and 1 c.cm. bisglucol were given, followed by another 0.45 gram N.A.B. and 1 c.cm. bisglucol on February 23rd. The patient then complained that he was "not feeling quite the thing." There was a little oedema round the eyes, but no swelling of the ankles. On examination the urine was found to be "solid" with albumin. It was then decided to transfer the patient to hospital.

On February 25th he was admitted into the Royal South Hants Hospital. He then showed a typical secondary rash,

with enlarged, hard glands—especially the inguinal group—and the remains of a hard chancre on the prepuce. Examination of the urine showed 30 parts of albumin per 1,000, with just a trace of blood. The blood pressure was 130/75, blood urea 65 mg. per cent.; urea concentration gave readings of 1.3, 1.4, and 1.5 per cent. The deposit in the urine showed red blood cells, leucocytes, and a number of finely granular casts.

For the first few days the patient was treated as a case of acute nephritis. No antisyphilitic treatment was given except hyd. c. cret. gr. ii t.d.s. For some days the albumin remained at 20 to 30 parts per 1,000. It dropped a little, but there were still 8 parts on March 6th. On March 9th 0.15 gram N.A.B. was given. By the 11th no albumin could be detected in the urine. N.A.B. 0.3 gram was given on March 14th. The urine was still absolutely normal.

The patient is now on a full course of N.A.B. and bismuth; no abnormality can be detected in the urine, and he himself feels perfectly fit.

DISCUSSION

This case in many respects is quite typical of a syphilitic nephritis in the secondary stage. All the cases described are characterized by the abundant albumin (up to 100 parts per 1,000), the presence of all kinds of casts, but blood in only a small quantity. Oedema is usually present, often in quite a marked degree. Renal efficiency tests, in spite of the massive albuminuria, are almost normal. The renal function in this case was somewhat impaired, the urea concentration test never reaching 2 per cent., whilst the blood urea was 65 mg. per cent. Pathologically, the condition is one of tubular degeneration—that is to say, a so-called nephrosis. The most constant feature of all the cases described is the dramatic effect of a single dose of an arsenic preparation. In this case two doses of N.A.B. had been given before the nephritis occurred. This fact at first made the diagnosis of a