being " sore " for three days, but the old headaches did not recur. She was discharged in two weeks, feeling very well and with no sign of tumour or pulsation.

Summary

1. The aetiology of cirsoid aneurysm of the scalp does not appear to have been definitely settled.

2. A theory attractive to the practical surgeon is that cirsoid aneurysm, racemose aneurysm, the aneurysm of anastomosis, and the pulsating angioma are due, as is arterio-venous aneurysm, to an abnormal communication between the arterial and venous systems, and that such communication may be either developmental or traumatic in origin.

3. Even large cirsoid aneurysms of the scalp may be extirpated by a method which depends for its success on the easy stripping of thrombosed and oedematous tissue, including the aneurysm, from the skin layer of the scalp flap.

4. Despite the fact that the vascular layer is stripped from the flap with the aneurysm, the skin flap receives sufficient nourishment, and very little sloughing takes place even with a large flap.

5. In the case described the whole aneurysm was excised. Searby, dealing with a larger aneurysm, excised only two-thirds of it, and the results were excellent, because the abnormal communication between the arterial and venous systems was abolished.

6. An interval of ninety-six hours between the two stages of the operation should not be exceeded, as necrosis of the flap edges might occur. An interval of seventy-two hours may not allow such easy stripping of the aponeurosis and thrombosed tissue from the skin flap. Probably an interval of eighty-four hours would be ideal.

7. The operation should not be a formidable one in practised hands, despite the vascularity of the scalp; haemorrhage will naturally be greater and the operation time will be longer if aneurysmal tissue has to be cut through.

8. It is unlikely that quinine and urethane injections, as have been lately used in the treatment of naevus, would be successful in the treatment of extensive pulsating tumours.

I am indebted to Dr. A. H. B. Pearce, chief medical officer of the Colony, for permission to publish this article.

The illustrations are by Alohihakau Eva, a student of the Central Native Medical School, Suva, Colony of Fiji.

REFERENCES

[1] Bryant: *Ann. of Surg.*, August, 1887, p. 116, quoted by Jacobson and Rowlands, 1907.
[2] Cheyne and Burghard: *A Manual of Surgical Treatment*, 1912, ii, 190.
[3] Choyce: *A System of Surgery*, 1923, i, 442.
[4] Jacobson and Rowlands: *Operations of Surgery*, fifth edition, 1907, ii, 769.
[5] Kerr, R. A.: *British Medical Journal*, 1933, ii, 566.
[6] Searby, H.: *Australian and New Zealand Journ. of Surg.*, 1931, i, No. 2.
[7] Sabin, Florence: *Reports of Johns Hopkins Hospital*, 1913, Monograph, New Series, No. 5.
[8] *Tillman's Textbook of Surgery*, 1900, ii, 24, translated by Tilton.
[9] Thompson, Miles, and Wilkie: *A Manual of Surgery*, eighth edition, i, 311; ii, 328.
[10] Berry, W.: *Lancet*, December 22nd, 1906, quoted by Searby.

G. Murano (*La Pediatria*, June 1st, 1936, p. 521) records the case of a male infant, aged 18 months, who nine days after the onset of varicella developed a tremor in the right lower limb which rapidly became generalized. An intense horizontal nystagmus was also noted together with general muscular hypertonus and a slight exaggeration of the tendon reflexes. The cerebro-spinal fluid was normal. The nervous symptoms disappeared in twenty-five days, but about a fortnight later the child developed broncho-pneumonia, and died after a week's illness. There was no necropsy. A history of repeated abortions in the maternal grandmother, the mental deficiency of the father, and the retarded psychical development and positive Wassermann reaction in the child are constitutional factors which may have reduced the child's resistance.

BLOOD TRANSFUSION

A REPORT OF SIX FATALITIES

BY

N. S. PLUMMER, M.D., M.R.C.P.

ASSISTANT PHYSICIAN TO CHARING CROSS HOSPITAL

This is a report of six fatalities following blood transfusion. Five of them occurred from heart failure, although ordinary precautions had been taken in the preliminary tests and in the performance of the operation. Death occurred, often with pulmonary oedema, but without evidence of haemolysis, from thirty minutes to nineteen hours after transfusion. These five cases form a group of which only few isolated examples have been reported; this, however, does not appear to be a true index of their incidence, for they were collected within a period of two and a half years at one hospital. Only two of these five were personally observed; in each of the other cases the notes were amplified by personal communication with the operators concerned.

The sixth case was clearly the result of incorrect grouping; haemolysis was evident, and death occurred from uraemia four days later. None of the cases was *in extremis* before transfusion; they can all, therefore, be fairly described as transfusion fatalities. A careful post-mortem examination was made in each case, and I am indebted to the late Professor Donaldson for reporting on the histological appearances.

Case I

Addison's Anaemia: Transfusion: Pulmonary Oedema: Death in Ten Hours

A woman aged 60 had had three operations many years previously for a pelvic condition. She was admitted to a surgical ward with a story of having had epigastric pain and vomiting and loss of weight for three years, and with weakness and dizziness of six months' duration. A test meal showed achlorhydria, and two *x*-ray examinations suggested a carcinoma of the pylorus; haematoporphyrin was present in the faeces. The haemoglobin was 30 per cent., but no further blood count was made. Laparotomy was decided upon.

The patient belonged to Group IV, and 300 c.cm. of blood from a relative (also Group IV) were given by the citrate method at 6.30 p.m. The patient's serum was tested against whole blood of the donor and no agglutination appeared. Shortly after completion of the operation she experienced a sense of fullness in the throat and shivered slightly. At 8 p.m. the pulse rose to 140 and the temperature to 100° F.; pulmonary oedema developed. Later, adrenaline and atropine were given; the patient became comatose and died at 5 a.m., ten hours after the transfusion.

Post-mortem Examination.—The stomach was normal; no carcinoma. The bases of the lungs were congested and oedematous; there was excess of frothy secretion in the trachea and bronchi. The heart showed excess of fat over the surface; the muscle was soft, and the right side was dilated. The liver was soft and deeply congested. The bone marrow of the humerus was deep red. The spleen was slightly enlarged and very deep red. The kidneys were normal and the arteries healthy. The urine was clear. The histological examination was as follows: Perles' reaction showed much iron in the liver, spleen, and kidneys; in the last-named there was slight cloudy swelling only. The spleen sinusoids were engorged with blood; normoblasts and megaloblasts were present, and a fairly large number of eosinophils. Iron was present in the central parts of the liver lobules, but there was no marked fatty change. There were focal reticulo-endothelial cell collections in the cardiac muscle, but no fibrosis and no fatty degeneration. The bone marrow showed a well-marked normoblastic and megaloblastic reaction; many eosinophils were seen.

Case II

Carcinoma Ventriculi : Moderate Anaemia : Transfusion : Death in Nineteen Hours

A man aged 62 was treated in hospital from May until October, 1929, for a chronic gastric ulcer with relief. In December, 1930, he was treated again, and returned to work with some occasional pain. In April, 1931, owing to persistence of the symptoms and a radiological filling defect, carcinoma was diagnosed. He was admitted on April 16th, 1931, following a haematemesis. His recovery from this was unsatisfactory ; pain persisted, and occult blood was always present. A test meal showed complete achlorhydria and considerable delay in emptying. The haemoglobin figure was 40 per cent.

A transfusion was performed on May 11th with a view to operation. The donor, a relative, and the recipient were grouped and cross-grouped, and 600 c.cm. of blood were given by the citrate method at 10 a.m. There was no reaction until 1 p.m., when there was shivering and the pulse rose to 140. Adrenaline was given. At 5 p.m. the pulse was weaker, and cardiac stimulants, including strophanthin subcutaneously, were given. At 8 p.m. there was definite improvement, the pulse being 100. At 5 a.m. the next day the breathing suddenly became shallow and the pulse feeble ; the patient died in fifteen minutes, nineteen hours after the transfusion.

Post-mortem Examination.—The stomach contained two pints of altered blood ; there was a large neoplasm at the pylorus, five inches long, with metastases in the glands. The liver was pale, but there were no secondary deposits. The kidneys were congested, the urine was clear, and the spleen was small. The lungs were emphysematous. The heart showed slight hypertrophy of the left ventricle ; the muscle was pale, but there was no fibrosis. The histological examination of the stomach revealed a spheroidal-celled carcinoma. The kidneys were healthy, apart from slight congestion. In the spleen the Malpighian bodies were small, and there were no hyaline changes. The erythrocytes were well defined ; a small amount of iron was present, and there were more eosinophils than usual.

Case III

Multiple Telangiectases : Severe Anaemia : Transfusion : Death Four Hours Later

A man aged 72 had been anaemic for three years. He had telangiectases on the nose and cheeks which used to bleed some years previously whenever he shaved, so that he now grew a beard. At no time did these bleed very much, and he had never had epistaxes. His brother had similar spots on his nose which bled, but there was no history of epistaxis in the family. In November, 1932, the patient was very pale, with a haemoglobin figure of 20 per cent. The tongue was normal, the spleen was not palpable, there were no retinal haemorrhages, and the blood film did not correspond with the picture of Addison's anaemia.

The patient was grouped and cross-grouped, and a blood transfusion of 300 c.cm. was performed by the citrate method. There were no untoward symptoms during the operation, but two hours later he experienced a dull pain behind the sternum and some shivering. He suddenly became worse, developed air hunger with marked collapse, and died four hours after the transfusion.

Post-mortem Examination.—Telangiectases were present round both eyes and in the mouth. The lungs were emphysematous, but otherwise normal ; the trachea contained a little blood-stained mucus. The right side of the heart was dilated. The liver showed mottled, congested lobules, and a small cyst containing caseous material. The spleen was a little enlarged, firm, and congested. The kidney had a narrow cortex, with arteriosclerotic vessels. Histological examination showed the kidney to be congested ; the erythrocytes were well formed and normal. In the spleen there were small Malpighian bodies, with much hyaline degeneration of the arterioles, and more iron than in the other cases. Very little blood was seen ; many doubtfully granular cells were noted in groups, some of which showed active mitosis. The bone marrow contained many polymorphs, myelocytes, eosinophils, megakaryocytes, and nucleated red cells—certainly not aplastic.

Case IV

Haemolytic Anaemia, Mitral Stenosis : Transfusion : Pulmonary Oedema : Death in Two Hours

A woman, 26 years old, had had rheumatic fever at the age of 17 ; she had since been quite well up to the birth of her only child eighteen months before admission. Although it was a normal confinement she did not seem to pick up after it. She was left with shortness of breath, a sharp pain in the left shoulder, and dizziness on exertion, with a cough and a feeling of tiredness. Twelve months before admission she had weaned her child and taken a long holiday ; after this she felt much better for three months, when the symptoms returned and gradually became worse up to the time of her admission.

On examination the patient was very pale and slightly jaundiced, and her legs were oedematous. The urine contained bile salts and pigments, but the stools were normal in colour. The heart was slightly enlarged, and there was evidence of mitral stenosis. The spleen was palpable three inches below the costal margin, but the liver was not felt. Respiration was rather embarrassed, even at rest, and the temperature was slightly raised. A blood count showed: red blood cells, 2,480,000 per c.mm. ; haemoglobin, 35 per cent. ; colour index, 0.7 ; white cells, 9,800 per c.mm.; anisocytosis and polychromasia were well marked. The reticulocytes numbered 19.5 per cent. The resistance of unwashed cells was 0.475/0.4. Three weeks later the resistance of washed red cells was 0.55/0.45—a slight increase in fragility. The direct van den Bergh reaction was a slight immediate positive and the indirect was positive, 1 unit. A test meal showed achlorhydria, with pepsin absent. The electrocardiogram showed "some right ventricular preponderance suggesting mitral stenosis. The slight indenting of ventricular complexes shows some myocardial disease as well."

A diagnosis of mitral stenosis with a haemolytic anaemia of obscure origin was made. After three weeks' treatment with liver and large doses of iron there was no improvement in the anaemia, although the oedema and dyspnoea had disappeared ; a blood transfusion was therefore advised with a view to splenectomy at a later date.

The patient was a Group II case and the donor a Group II. The serum of the patient was tested against the whole blood of the donor with no agglutination ; 600 c.cm. of blood were given slowly by the citrate method. During the transfusion the patient developed a mild dry cough, and immediately afterwards she felt sick. Forty-five minutes later she collapsed, and was grey in colour. Adrenaline, 5 minims, and an hour later morphine, 1/4 grain, with atropine, 1/50 grain, were given because of pulmonary oedema, but the patient died two hours after completion of the transfusion.

Post-mortem examination of the lungs revealed some recent pleurisy on the right side, with subpleural petechial haemorrhages in the main fissures. They were bulky and failed to collapse on removal ; deeply engorged and oedematous. The smaller bronchi were clearly dilated, and contained mucopus. The trachea was full of frothy fluid, and there was marked soft enlargement of the bronchial glands. The heart showed dilatation and hypertrophy of both auricles and the right ventricle ; the mitral valve was stenosed, and admitted the tip of one finger only. The liver was very firm and congested, and somewhat nodular near the free margin ; it was normal in size. The pancreas was normal. The spleen weighed 1,550 grams ; there were recent patches of perisplenitis. It was very red and firm, and the Malpighian bodies showed up distinctly. The kidneys were firm and the pyramids purple. The bone marrow of the humerus was dark red. The pathologist's summary was: "Mitral stenosis followed by chronic bronchitis and congestion of liver and fibrosis of this organ. Also evidence of haemolytic anaemia, precise nature unknown. Death due to acute oedema of lungs resulting from blood transfusion."

Histological examination of the kidneys showed no haemoglobin plugs ; these organs were not unduly congested, and were healthy. The spleen was very congested. The Malpighian bodies were well marked, the pulp was full of erythrocytes, and there were no hyaline changes. The marrow showed a well-marked normoblastic and myelocytic reaction, a greater normoblastic reaction than in Case VI. The liver was moderately congested ; areas of focal necrosis were

chiefly peripheral. There were groups of leucocytes round the bile ducts. No true cirrhosis was present, but possibly early periportal fibrosis.

Case V

Chronic Empyema: Second Transfusion from Same Donor: Death in Half an Hour

A boy aged 18 had had a rib resection for drainage of an empyema following pneumonia in 1922. In 1924 this was drained again. In January, 1930, he was readmitted with pain at the site of the discharging sinus, fever, and a persistently high pulse rate. At an operation on January 14th portions of the third to the eighth ribs were resected. The fever continued and, since the haemoglobin was 40 per cent. on February 14th, the first transfusion of 360 c.cm. was given after careful grouping and cross-matching. A mild febrile reaction without haemoglobinuria or jaundice followed. On February 28th a second thoracoplasty was performed. On March 2nd a second transfusion was given, using the same donor, the patient's brother, without any further tests being done; 270 c.cm. of citrated blood had been run in when the patient complained of backache, his breathing became stertorous, there was cyanosis, and the sphincters were relaxed. From this he rallied, but suddenly collapsed while talking, and died thirty minutes after the completion of the transfusion.

Post-mortem Examination.—Marked scoliosis was present, and, on the right side, a sinus led to an abscess cavity under the fourth rib. The blood was fluid, and there was no post-mortem clotting. The right pleura was very thick and fibrous, and the right lung bronchiectatic and collapsed. The left lung was emphysematous and oedematous. The heart muscle was pale and the liver pale and flabby. The spleen was twice the normal size, soft, and congested. Histological examination of this organ revealed small Malpighian bodies; one or two vessels showed hyaline changes. There were many polymorphs and eosinophils, more so than in the section of Case II; there was some congestion, but no iron. The kidneys were healthy but congested; rather more polymorphs were present than usual, but no iron was seen.

Case VI

Oesophageal Ulcer, Secondary Anaemia: Five Previous Transfusions: Haemolytic Reaction: Death Four Days Later from Uraemia

A woman, 33 years old, swallowed a piece of pineapple in 1914 which seemed to stick in her throat. After this she had frequent attacks of pain on swallowing until, in 1926, oesophagoscopy revealed an ulcer one inch above the diaphragm. Despite two months' rest and light diet this did not heal, so a gastrostomy was performed in 1927, and deep *x*-ray therapy was given. In February, 1928, two transfusions were performed for anaemia, and she was then able to work for nine months. In 1929 a third transfusion was given. In June, 1930, the gastrostomy was closed, the haemoglobin at this time being 36 per cent. Owing to recurrence of the dysphagia and symptoms of anaemia, the patient was readmitted in January, 1931. The blood count showed: red blood cells, 2,327,000 per c.mm.; haemoglobin, 29 per cent.; colour index, 0.64; and white cells, 5,000 per c.mm. On January 22nd, 1931, she received a fourth transfusion of 360 c.cm. of Group IV blood. Five days later she had a fifth transfusion of 510 c.cm. of blood, which was followed by a fairly severe reaction, the temperature being 102° F. and the pulse 100. On February 2nd the haemoglobin was 36 per cent. By the usual tests the patient was, on February 9th, considered to belong to Group I; 540 c.cm. of citrated blood of Group I was therefore given. During the transfusion there were no untoward symptoms. Fifteen minutes later there was a sharp reaction with a rigor and high temperature and pulse rate. The next day deep jaundice appeared, the temperature being 104° F. and the pulse rate 107. The patient complained of pain and tenderness all over the body and of feeling very ill. On the second day after the transfusion the jaundice was deeper; there was complete anorexia with constipation and anuria. The temperature was 98° F. and the pulse rate 100. She vomited three ounces. On the third day she was much worse and weaker; no food was taken, and the pains increased. Chvostek's sign was positive, but there was no twitching. Intramuscular calcium was given without effect. On the fourth day the patient became comatose and died. Two ounces of urine were excreted just before death, but there is no record of this having been tested. Intensive alkali therapy, as advised by Osman, was not started until two days after the onset of anuria.

Post-mortem Examination.—A healed ulcer at the lower end of the oesophagus was found. Both pulmonary bases were congested and oedematous. The heart was normal. The liver was not enlarged, but it was soft and fatty. The spleen was twice the normal size. The two kidneys weighed 480 grams; they were soft, pale, and flabby. The Prussian blue reaction was negative. The histological examination showed many renal tubules blocked with "haemoglobin" and debris of "degenerated red cells"; the microscopical Perles reaction was negative. The spleen contained small Malpighian bodies; the sinuses were not distended; there were many erythrocytes well formed in the pulp; a few polymorphs; no hyaline changes; and no iron. The liver sinusoids were well filled; there were slight back-pressure changes, but no iron was seen. The marrow contained numerous megakaryocytes, myelocytes, and normoblasts—a normoblastic reaction. The heart muscle cells were rather thin; there was no fibrosis, but a slight increase of brown pigment.

Discussion

The various types of transfusion reaction have been discussed elsewhere.[13] There are two main types: the common febrile reaction, which is also known as the delayed or "anaphylactic" or proteolytic reaction, and the haemolytic reaction due to faulty grouping. The latter takes two forms: the immediate and the late, or uraemic reaction.

The common febrile reaction[13] is probably caused by the introduction of foreign protein in the form of old blood clot, of bacteria contaminating solutions, or of incipient coagulative changes in the donor's blood, governed somewhat by the time taken to run the blood into the patient's vein, and its temperature. This conclusion is based on the similarity of the clinical picture to that of reactions caused by intravenous serum therapy, and the fact that reactions are more common in recipients sensitized by previous transfusions or possessing an allergic taint.[14] That citrate plays an important part in causing reactions is disproved by Meleney,[10] and Lewisohn and Rosenthal.[9]

Cases I to V in this paper fall into the category of reactions of this type. Approximately 50 per cent. of transfusions are followed by such reactions of varying intensity, and yet only a very small percentage of them are fatal.

The first three patients died from heart failure. They were all the subjects of long-standing anaemias, so that presumably the heart muscle in each case was functionally weak, although at necropsy there was little evidence of organic change. In Case I, suffering from pernicious anaemia, pulmonary oedema indicated a left-sided heart failure. The next two patients died from syncope. Case II was complicated by the presence of a neoplasm of the stomach, although there was no marked cachexia. In Case III, with anaemia secondary to bleeding from telangiectases, there were the additional factors of arteriosclerosis and old age. In each case the reaction started at least half an hour after the completion of the operation. It is this interval which helps to differentiate this type from the immediate reaction due to faulty grouping, when symptoms usually come on after only 100 c.cm. of blood have been given.[12]

There is no reason to think that blood of the wrong group had been used in view of the double testing beforehand, the absence of symptoms during the operation, and in particular the absence of backache and the fact that the urine in the bladder in Case I was clear at

necropsy. Carrington and Lee[5] report an identical case in which the venous blood was carefully tested for agglutination and haemolysis just before death, also the urine for red cells and haemoglobin, with negative results. Brines[4] also records a similar case with a post-operative infection, in which the reaction started half an hour after the transfusion, and death followed ten hours later with pulmonary oedema.

Case IV was a particularly bad risk for blood transfusion, but the explanation of this fatality is essentially the same as that in Cases I to III. There were obvious signs of circulatory failure three weeks before transfusion, due to mitral stenosis and severe haemolytic anaemia; these signs, however, had subsided with rest. During the course of the transfusion of 600 c.cm. of blood the dry cough should probably have been interpreted as an early sign of pulmonary oedema due to heart failure, and the operation stopped accordingly.

Two similar examples of fatalities from blood transfusion in cases of valvular disease of the heart are recorded, one by Beisenberger[2] and the other in the discussion following a paper by Kordenat and Smithies.[7] The presence of haemolytic anaemia in Case IV may have been an additional factor in causing death. Dawson[6] stresses the danger of transfusion in cases of acholuric jaundice, and Payne[11] reports a fatality in a case of acute haemolytic anaemia of Lederer. In Payne's case, however, death took place four days after the operation with suppression of urine.

In Case V the patient was severely ill as a result of septic absorption from a chronic empyema and of two thoracoplastic operations, yet a mild febrile reaction following the first transfusion did not upset him. Fourteen days later, without further preliminary tests, the same donor was used for a second transfusion. When 270 c.cm. of blood had been run into his vein symptoms developed which led to his death half an hour later. One must assume that as a result of the first transfusion the recipient had become sensitized to this donor's blood. It is to be noted that in this case there was no delay in the onset of symptoms as in the first four cases.

Thalhimer,[16] Levine and Segal,[8] Astrowe,[1] and Smith and Haman[15] record very severe reactions after second transfusions from the same donor. The majority of them were haemolytic in character, but the patients all recovered. In none of these was the cross-agglutination test performed on the second occasion. Traum[17] described three such cases of severe anaphylactic shock which recovered without showing haemolysis. In one case there was an interval of eighteen months between the two transfusions. He is so impressed with this danger that he advises that a donor should not on any account be used a second time for the same patient, even if the cross-matching shows no incompatibility.

The sixth case is an example of a typical haemolytic reaction following transfusion of blood of the wrong group. Jaundice and suppression of urine occurred, and the kidneys gave the usual finding of brown pigmented debris in the tubules. This patient had had five previous transfusions from Group IV donors, but on the sixth occasion, owing to an error, he was given blood from a Group I donor. It is now known that the blood group of an adult never changes (Polayes and Lederer),[14] although, as a result of sensitization or other cause, the titre of agglutinins in the serum may be raised sufficiently to interfere with the preliminary tests. Several such haemolytic reactions[3] are recorded resulting in death from uraemia four to twelve days after the transfusion, and the clinical picture contrasts strongly with that in Cases I to V reported above.

Attention must be drawn to our ignorance in regard to the renal changes in such cases. It is ordinarily assumed that the brown pigment in the tubules is a precipitated breakdown product of haemoglobin, which has been liberated from the haemolysed donor's cells. So far as I know the nature of this pigmented debris has never been ascertained. That it does not consist of free iron is shown by the negative Prussian blue reaction in Case VI above and in Bordley's[3] third case. This, of course, does not disprove its origin from haemoglobin.

Witts[18] and Bordley[3] record cases showing uraemia and the characteristic renal changes associated with transfusion deaths in which there had been no jaundice or other evidence of haemolysis during life. It therefore appears improbable that such deaths can invariably be explained as the result of haemolytic changes.

Conclusion

It is the object of this paper to illustrate and stress some of the transfusion risks which are dependent upon the general condition of the recipients, quite apart from the question of faulty grouping. Except possibly for Case IV, none of Cases I to V would ordinarily have been considered to be unsuitable for blood transfusion; nevertheless they all died as a direct result of this procedure. With all ordinary precautions of grouping and selection of cases the operation of blood transfusion carries with it a risk which indicates that it has to be used with considerable discretion in a number of conditions.

Summary

1. Four cases of heart failure secondary to ordinary transfusion reactions are reported, illustrating some of the dangers of transfusion, quite apart from the question of faulty grouping.

2. These fatalities occurred in patients with long-standing anaemia or with heart disease.

3. The danger of using the same donor for a second transfusion is illustrated by one fatality (Case V) and reference to serious reactions recorded by other authors.

4. A haemolytic reaction (Case VI) is reported ending in death from uraemia. The clinical picture is in striking contrast with that in the other five cases.

5. Doubt is cast on the accepted theory of haemoglobin plugging of the tubules in all cases of uraemia following transfusion.

REFERENCES

[1] Astrowe, P. S.: *Journ. Amer. Med. Assoc.*, 1922, lxxix, 1511.
[2] Biesenberger, H.: *Wien. klin. Woch.*, 1928, xli, 923.
[3] Bordley, J.: *Arch. Int. Med.*, 1931, xlvii, 288.
[4] Brines, O. A.: *Journ. Amer. Med. Assoc.*, 1930, xciv, 1114.
[5] Carrington, G. L., and Lee, W. E.: *Ann. of Surg.*, 1923, lxxviii, 1.
[6] Dawson, Lord: *British Medical Journal*, 1931, i, 921, 963.
[7] Kordenat, R. A., and Smithies, F.: *Journ. Amer. Med. Assoc.*, 1925, lxxxv, 1193.
[8] Levine, E. C., and Segal, H. N.: *Surg., Gynecol. and Obstet.*, 1922, xxxv, 313.
[9] Lewisohn, R., and Rosenthal, N.: *Journ. Amer. Med. Assoc.*, 1933, c, 466.
[10] Meleney, H. E., *et al.*: *Amer. Journ. Med. Sci.*, 1917, cliv, 733.
[11] Payne, R. V.: *Guy's Hospital Reports*, 1934, lxxxiv, 65.
[12] Pemberton, J. de J.: *Surg., Gynecol. and Obstet.*, 1919, xxviii, 262.
[13] Plummer, N. S.: *Charing Cross Hospital Gazette*, 1935, xxxv, 220.
[14] Polayes, S. H., and Lederer, M.: *Journ. Lab. and Clin. Med.*, 1932, xvii, 1029.
[15] Smith, C. E., and Haman, J. O.: *California and Western Med.*, 1934, xli, 157.
[16] Thalhimer, W.: *Journ. Amer. Med. Assoc.*, 1921, lxxvi, 1345.
[17] Traum, E.: *Deut. Zeit. f. Chir.*, 1932, cclvii, 97.
[18] Witts, L. J.: *Lancet*, 1929, i, 1297.

Dr. Frederick H. Albee, the well-known surgeon of New York, during a recent trip through South America received the following honours from different countries: honorary presidency of the Brazilian Society of Orthopaedic Surgery and Traumatology; honorary professorship of surgery, San Marcos University, Lima, Peru; and membership of the Faculty of Medicine, University of Chile.