TABLE II.—Times of Completion of Clotting of Pure Blood shed into Paraffined Vessels after the Internal Administration of the Haemostatics Named.

Name of Haemostatic.	Amount Administered.	Mode of Administration.	Dose in Man Recommended by Makers.	Time of Completion of Clotting at 17° C.	Time of Completion of Clotting at 37.5° C.
Coagulen (Ciba)	1.25 c.cm. of a 3% solution	Intravenous	10 to 20 c.cm. of a 3% solution	min. sec. 24 0	min. sec. 10 55
Fibrogen (Merrell)	0.5 c.cm.	Intramuscular	3 c.cm. orally followed by 1 c.cm. subcutaneously for each 75 lb. of body weight	9 20	5 20
Thromboplastin hypodermic (Squibb)...	2.5 c.cm.	Intravenous	10 to 20 c.cm. every 24 to 48 hours	20 10	8 5
Thromboplastin hypodermic (Squibb)...	7.5 c.cm.	Intravenous		19 0	7 20
Hemoplastin (Parke, Davis)	0.25 c.cm.	Intravenous	2 c.cm. either intravenously or subcutaneously; in severe cases 5 c.cm.	11 0	8 0
Sodium citrate... ..	5 c.cm. of a 10% solution	Intramuscular	30 c.cm. of 10% solution intramuscularly	17 30	5 40

Note.—Hypercoagulability is evident in a few minutes after injection the results are naturally slower. In the experiment after injection, in that with sodium citrate one hour after

intravenous injection (two to three minutes). In intramuscular recorded with fibrogen (Merrell) the blood was shed three hours injection.

Conclusions.

Each of the newer haemostatics accelerated the clotting of pure blood *in vitro*. Table I shows that preparations which appear inefficient at room temperature (17° C.) are active coagulants at body temperature. The standardization of haemostatics by reactions at room temperature is therefore undesirable. It also follows that coagulant haemostatics should be applied locally at body temperature and the wound should not be allowed to cool.

The application of a haemostatic by continuous irrigation is inadvisable, as the clots formed are disturbed by the moving liquid and complete clotting of the escaping blood is thus hindered. The use of a hot bandage (37° to 40° C.) soaked in the haemostatic avoids disturbance of the clots whilst providing a temperature at or near the optimum for haemostatic action. This practice is also useful when the haemostatic is administered internally when haemorrhage continues from an accessible wound.

Of the preparations investigated only those mentioned in Table II are used internally. They do not produce either a severe fall in blood pressure or intravascular clotting in the cat when administered by the routes and in the concentrations named. The experimental results thus agree with the clinical observations. Periodicity is evident in the coagulant action of some of the preparations, the lower dilutions being more active than the higher. These results correspond with those shown in the experiments of Waksman⁷ on the coagulant action of cephalin. The greater activity at lower dilutions may account for the sudden cessation of bleeding which sometimes occurs when initial haemostatic action is small.

The increased coagulability of the blood after the intra-

venous injection of small amounts of sodium citrate is not described, as it is established both experimentally^{8, 9} and by numerous clinical observations.^{10, 11, 12}

Summary.

1. A method of standardizing the coagulant action of haemostatics is described.

2. Those haemostatics which precipitate blood plasmas are ineffective.

3. All the haemostatics named in Tables I and II are effective coagulants when the temperature of the blood is maintained at 37.5° C. Some are also effective at room temperature.

4. Stress is laid on the maintenance of the temperature of the wound at 37.5° C. in the use of haemostatics.

5. The experimental results recorded in this paper agree with the clinical observations on the efficacy of the haemostatics investigated, but are at variance with the findings of those investigators who have employed blood kept fluid by either oxalates or citrates.

REFERENCES.

- 1 Mills, C. A.: *Journ. Biol. Chem.*, vol. 46, pp. 135-165, 1921.
- 2 Hanzlik, P. J.: *Journ. Pharm. and Exper. Therapeutics*, vol. 12, pp. 71-117; *Ibid.*, pp. 119-128, 1918-19.
- 3 Hanzlik, P. J., and Weidenthal, C. M.: *Journ. Pharm. and Exper. Therapeutics*, vol. 14, pp. 189-210, 1919-20.
- 4 Hamilton, H. C.: *Journ. Lab. and Clin. Med.*, vol. 5, p. 574, 1920.
- 5 Pickering, J. W., and Hewitt, J. A.: *Journ. Physiol.*, vol. 59, pp. 455-9, 1925.
- 6 Pickering, J. W., and Hewitt, J. A.: *Quart. Journ. Exper. Physiol.*, vol. 13, pp. 200-7, 1923.
- 7 Waksman, S. A.: *Amer. Journ. Physiol.*, vol. 46, pp. 375-395, 1918-19.
- 8 Howell, W. H.: *Amer. Journ. Physiol.*, vol. 31, p. 1, 1912.
- 9 Weil, R.: *Journ. Immunol.*, vol. 2, p. 535, 1916-17.
- 10 Neuhoff, H., and Hirschfeld, S.: *Annals of Surgery*, vol. 76, pp. 1-8, 1922.
- 11 Cheinisse, L.: *Presse méd.*, October 14th, 1922, pp. 887-8.
- 12 Renaud, M., and Juge: *Compt. rend. Soc. Biol.*, 1924, pp. 425-6.

ACUTE DUODENAL OBSTRUCTION DUE TO NEO-NATAL VOLVULUS.

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We think this case may be of interest as illustrating one of the congenital causes of vomiting in infants dealt with by Dr. H. C. Cameron in his Lumleian Lectures, which were published in the *BRITISH MEDICAL JOURNAL*.

J. W. B., a male infant, born in the obstetrical unit of the Royal Free Hospital on July 23rd, 1925, after a normal pregnancy and delivery, was healthy, weighing 8 lb. 1 oz., at birth. Breast-fed, his progress was in every way normal for the first six days of life, and on the seventh day his weight was 8 lb. 6½ oz. On this day he began to vomit at irregular intervals after feeds, sometimes immediately, sometimes after a lapse of ten to thirty minutes. For three days he was given mercury and chalk 1/8 grain twice daily, and his food was changed successively to diluted breast milk, to Nestlé's milk (one part in twelve), to albumin water and brandy, and, finally, to normal saline.

On the second day of illness the vomit contained much bile, and on the third day vomiting became projectile. On the fifth day visible peristalsis of the stomach was observed. The abdomen was scaphoid, and at no time could any tumour be felt.

At first the stools were normal milk stools, but on the twelfth day of life (fifth day of illness) two stools were passed consisting apparently of pure meconium. All later stools consisted of mucus only, tinged green. On two occasions the stomach was washed out with sodium bicarbonate; the fluid returned contained large quantities of bile.

The diagnosis of low duodenal obstruction was made and the question of operation considered. The general condition of the infant, however, contraindicated operative measures. The child gradually grew weaker, and on the evening of the eighth day the temperature rose suddenly to 104° F., death occurring on the tenth day of illness.

Throughout the illness the baby at no time showed any sign of pain or shock suggestive of acute abdominal lesion. From the seventh day of life, when his weight was 5½ oz. above birth weight, he lost weight progressively, until at death, on the eighteenth day, he was 2 lb. 9 oz. below birth weight.

Post-mortem Examination.

On opening the abdomen the much dilated stomach and first and second parts of the duodenum presented; they formed one continuous distended cavity, the stomach passing from the pylorus to the left and upwards, to press on the left lobe of the liver. The coils of small intestine were collapsed and purplish-black. Gangrene of the small intestine had begun, and there was a very

fetid odour. The caecum lay on the left side of the abdomen, with a retrocaecal appendix. The tense ascending colon passed from left to right across the fourth part of the duodenum, under cover of coils of ileum. The transverse colon lay coiled under the dilated stomach on the left side. The descending and pelvic colons and the rectum were normally situated and attached.

Between the pylorus and the splenic flexure of the colon there was no direct attachment of the intestines to the posterior abdominal wall. The duodenum had a mesentery. There was acute torsion of the third part of the duodenum, and, round this point as axis, there had been a volvulus of the entire gut distal to the third part of the duodenum and proximal to the middle of the transverse colon. The superior mesenteric artery was twisted spirally round the third and fourth parts of the duodenum. The presence of the artery, together with the acute torsion, effectually obstructed the lumen of the gut at this part. The vascular lumen was apparently also partially, or completely, obstructed.

The torsion of the duodenum was readily undone by twisting the mass of intestine from left to right, through an angle of 180 degrees. The duodenum was then seen as a straight tube

Diagram of position of viscera at birth. A volvulus of 180 degrees occurred, secondary to this condition, the caecum passing forward and to the left in front of the duodenum. The superior mesenteric artery was carried across the third part of the duodenum. A=Superior mesenteric artery. B=Duodeno-jejunal flexure. C=Transverse colon.

of the mesentery of the ileum. The ascending colon was also adherent to the root of the mesentery as it passed behind the duodenum. The rest of the gut had a free mesenteric attachment.

The mesentery was drawn tightly round the third part of the duodenum, drawing with it the superior mesenteric artery, which thus passed successively downwards behind the duodenum, forwards round the right side of the duodenum between the duodenum and ascending colon, then transversely across the duodenum, passing into the mesentery of the ileum. A large branch of the artery passed obliquely downwards, crossing anterior to the duodenum, and then behind the duodeno-jejunal flexure, to supply the jejunum. This artery appeared to cause partial obstruction to the duodenal lumen. The lumen of the colon was obstructed by the acute flexure at the junction of ascending and transverse colons. The caecum and ascending colon contained faeces of a normal yellow colour. The ascending colon was tense and smooth.

The specimen suggests a primary malrotation in a direction the reverse of the normal, bringing the colon behind the duodenum, this rotation being arrested, at the stage where the ileum had passed forward across the duodenum, by the firm adhesion of the transverse colon to the mesentery of the ileum. Any further rotation could only occur by torsion of the duodenum and volvulus. After birth the irregular peristalsis induced by the obstruction at the duodeno-jejunal flexure, and more especially at the junction of the ascending and transverse colons, determined the occurrence of the volvulus, which carried the caecum through an angle of 180 degrees across the duodenum to the left, twisted the superior mesenteric artery tightly across the third part of the duodenum, and thus produced acute duodenal obstruction.

Our thanks are due to Professor McIlroy and to Mr. Norbury for permission to publish the case.

A CASE OF EMBOLECTOMY.

BY

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MR. GEOFFREY JEFFERSON is to be heartily congratulated on the successful case of embolectomy recorded in the *BRITISH MEDICAL JOURNAL* of November 28th, 1925. In a leading article in the same issue some comments are made (to which I shall refer later) on a case upon which I operated in 1911; the details of this were published in a clinical lecture on post-operative embolism of the femoral artery in the *Clinical Journal* of December 10th, 1913.

The publication of Mr. Jefferson's case has stimulated me to record a case on which I operated in 1914 at St. Bartholomew's Hospital, and which I should probably have published before but for the war.

A married woman, aged 27, was admitted to the maternity ward at St. Bartholomew's, under Dr. Barris, on March 2nd, 1914. She was suffering from mitral stenosis, and had severe albuminuria with oedema of both legs. On March 4th she was delivered with forceps. On the following day marked dilatation of the heart was noted, followed by dyspnoea. During the morning of March 13th sudden acute pain was felt in the right foot below the knee, and no pulsation could be felt in the leg below the groin.

I saw the patient about five hours after the onset of symptoms. She was desperately ill, with feeble pulse and severe dyspnoea. The right limb was somewhat mottled below the knee, cold and insensitive. No femoral pulse could be felt. I exposed the common femoral artery under local anaesthesia, the patient being propped up in the sitting position as she was unable to lie down. I removed an embolus from the bifurcation of the artery with some recent thrombus from above, passing a probe up the artery until blood flowed. The artery was closed with fine vaselined silk, and pulsation was restored to the limb immediately after the operation. Shortly after the patient was returned to bed it was noted that pulsation in the femoral could not be obtained. The patient died the same evening, about three hours later. Before death pulsation was absent from both femorals.

Post-mortem examination showed that thrombosis had occurred in the right common femoral artery up to the bifurcation of the aorta and down to the junction of the external and internal iliacs on the left side. Mitral stenosis, bilateral pleural effusion, and chronic interstitial nephritis were also found.

It is doubtful if this patient would have recovered in any circumstances in view of the severe kidney and heart disease, but the rapid and extensive thrombosis above the embolism showed that the operation was performed too late. It seems probable that after the embolus had been removed the probe passed through and did not dislodge to any extent the thrombus, which possibly was even then extending up to the bifurcation of the aorta. So feeble was the action of the heart that the channel was soon blocked up again—a block which before death occurred extended to the left common iliac.

This case emphasizes the importance of early operation if any benefit is to accrue.

In the case which I published in 1913 (operation in 1911), referred to in the *BRITISH MEDICAL JOURNAL* of November 28th, 1925, an embolus occurred in the common femoral artery a fortnight after laryngectomy for carcinoma. At the operation the embolus became dislodged when the artery was lifted up in the wound in order to apply a Crile clamp before arteriotomy, and was checked at the bifurcation of the popliteal. I did not expose the popliteal but awaited events. Gangrene occurred below the knee. I subsequently amputated just above the condyles, and the patient made a good recovery.

It has been argued (*BRITISH MEDICAL JOURNAL*, November 28th, 1925, p. 1018) that the lower end of the popliteal should then have been exposed and the clot removed, a procedure which may present considerable difficulties. In the *Clinical Journal* I argued that immediate massage or pétrissage, as described and advocated by M. Paul Claisse (*Bulletins et Mémoires de la Société Médicale des Hôpitaux de Paris*, June 10th, 1910, p. 766) in reporting a successful case, might have been