for them?" The last question aids in determining the severity of the case.

The attempt should next be made to produce pain by active and passive movement, the patient's appearance being noted the while.

Then ask: (18) "Is the pain sharp or dull?" If he has had rubbing or massage, ask: (19) "Is massage painful?" (20) "Does it do you good?" Proceed to inquire about stiffness by asking, (21) "Does rest make the stiffness greater or less?" (22) Does the stiffness get greater or less when you are up and moving about?" Conclude with some such phrase as (23) "When you move *quickly* you get a sharp stabbing pain. Can you do as much as anybody else if you go slow?"

The last question is a valuable one for testing the patient's mental outlook. It puts him on his mettle. A genuine case answers in the affirmative or with a negative that is obviously reluctant. Cases in which there is unwillingness of the spirit as well as weakness of the flesh are not ashamed to give a negative the tone of which is whining in "war-worn" cases, and impressive, indignant, or sanctimonious in patients who are knowingly exaggerating their symptoms.

If the answers seem to coincide with the definition of myalgia given above, the diagnosis is made and a note registered for future reference as to the degree of the condition, and as to the degree, if any, of exaggeration employed.

TREATMENT OF THROMBOSIS.

BY

H. FAIRLEY MARRIS, CAPTAIN R.A.M.C.

VENOUS THROMBOSIS such as occurs in the typhoid group of fevers may be speedily aborted by the intravenous injection of sodium citrate.

During the past two years seventeen cases of venous thrombosis have been under my care, the majority having arisen during the course of an infection bacteriologically proven to be by a member of the enteric group, the remainder occurring during the course of other fevers of obscure origin.

This complication has long been recognized as prolonging convalescence and leading to permanent mischief, such as troublesome swollen limb or the formation of intractable varix.

The signs and symptoms as seen in my series may be briefly described. Rigors were noted in four of the cases. Fever was always present; pain invariably so, on two occasions being so severe that the question of intestinal perforation arose. Swelling of the limb was always present, usually appearing on the third day from onset of the other symptoms. In most of the cases the thrombus could be felt.

Among the enteric cases the distribution was as follows: Left femoral 9, right femoral 1, right and left femorals 1, left iliac 2, right and left iliacs 1.

The remaining three cases occurring during the course of other fevers were: Left femoral 2, right and left popliteals 1.

The earlier cases were treated on the usual lines—namely, locally with anodyne liniments, and internally by the liberal administration of citric acid and a milk-free diet.

The following is the first case treated by the intravenous injection of sodium citrate:

A soldier (Case 1 in the table), aged 19, admitted to hospital on the twenty-third day of his illness, which was subsequently shown to be typhoid fever. The temperature was 101° F., and the pulse 106 per minute. By the thirty-second day the temperature had regained the normal by lysis; the pulse rate, however, was still about 100 per minute.

On the thirty-fourth day the temperature rose to 100° F., and the patient complained of considerable pain in the lower part of the left abdomen. A thrombus was suspected, but nothing could be felt, and measurements round the two lower limbs at the calves gave the same result—namely, 10½ in.

By the thirty-fifth day the left leg was swollen, and on the thirty-seventh day oedema was observed in the right ankle. The swelling in both legs increased rapidly, and by the thirty-ninth day the man was in such agony and the abdomen so rigid that the possibility of an intestinal perforation was considered.

By the fortieth day the thighs resembled bolsters, and the superficial veins on the lower part of the abdomen became so conspicuous that the inferior vena cava appeared to be involved.

Thereupon resolved to try the effect of a 0.5 per cent. solution of sodium citrate in normal saline. Half a pint of this

solution warmed and sterilized was slowly run into the median basilic vein of the left arm. Twelve hours later the pain had almost gone. The following day a second injection of 8 oz. was given, and by the next morning the temperature had fallen, the patient was free from pain, and the abdomen was quite soft. A speedy recovery took place.

Imperial drink was given before the first injection; thereafter this was suspended. Below are shown a few of the calf measurements taken on the days stated:

Day of Disease.	Right Calf.	Left Calf.
34th	10½ in.	10½ in.
35th	10½ "	12½ "
37th	12½ "	12½ "
40th	13½ "	13½ "
45th	12½ "	12½ "
57th	11 "	11 "
60th. Got up for first time.		
61st	10½ "	10½ "
73rd. No oedema	10½ "	10½ "
74th. Evacuated as a walking case.		

From information received it was learnt that this patient was returned to duty 104 days from the onset of his illness—that is, seventy-four days from the beginning of the thrombosis.

This result was so striking when compared with those cases I had already seen that I decided in future to treat such cases by the oral and intravenous

methods alternately. Limb measurements were taken daily. On two occasions an attempt was made to compare the coagulation time of the blood before and after the injections.

The method used was as follows: a long capillary tube was drawn out and filled with the patient's blood from a finger prick; the tube was then quickly broken into inch-long pieces. Every quarter of a minute one of these was taken and the blood expelled by blowing, until, on account of coagulation, this was no longer possible. In Case No. 4 it was found that the blood could be expelled from only eight of the tubes before the injection, whereas after the injection the blood was readily expelled from twenty-four tubes. The coagulation time of the blood had been changed from two to six minutes.

In the earlier cases the injections were followed by rigors occasionally, which occurred as early as half an hour and as late as three hours afterwards; they were never severe, and were quickly relieved by half an ounce of brandy and hot bottles. With more careful preparation of the citrate solution, using recently distilled water and efficient sterilization, the rigors have not been observed. Rigors are not infrequently observed after intravenous injections of other substances, and this is not an important point.

The following table sufficiently explains itself.

CASE 1.—A = Pain in left iliac fossa. B = Injection of 0.5 per cent. sodium citrate.

VENOUS THROMBOSIS.
Cases of the Enteric Group treated by the Intravenous Injection of 0.5 per cent. Solution of Sodium Citrate.

No.	Age.	Bacteriological Diagnosis.	Site of Thrombosis.	Onset of Thrombosis.	Subsequent Pyrexia after First Injection.	Measurements of calf (Inch.s).	Convalescence.*	Treatment.	Subsequent History.
1	19	Typhoid by agglutination	R. and L. iliac. cava	33rd day	3 days	37th day: R.12 $\frac{1}{2}$, L.12 $\frac{1}{2}$ 61st day: R.10 $\frac{3}{4}$, L.10 $\frac{1}{4}$	21 days later	40th day: 10 oz. of 0.5 % sodium citrate intravenously. 41st day: 8 oz. of 0.5 % sodium citrate intravenously. No change in diet. Imperial drink by mouth.	Evacuated to United Kingdom (walking case, no oedema) 35 days after onset of thrombosis; returned to duty 70 days after onset of thrombosis.
2	28	Typhoid; <i>B. typhosus</i> in urine	L. iliac vein	38th day	8 days	38th day: R.11 $\frac{1}{4}$, L.13 49th day: R.11 $\frac{1}{4}$, L.11 $\frac{1}{4}$	19 days	38th day: 8 oz. of 0.5 % sodium citrate intravenously. Mild rigor 4 hour after injection. No change of diet; no citrates by mouth.	Evacuated to United Kingdom (walking case, no oedema) 40 days after onset of thrombosis.
3	27	Typhoid by agglutination	L. femoral	24th day	7 days	25th day: R.10 $\frac{1}{2}$, L.12 $\frac{1}{2}$ 44th day: R.10 $\frac{1}{2}$, L.10 $\frac{1}{2}$	17 days	25th day: 6 oz. of 0.5 % sodium citrate intravenously. No change in diet; no citrates by mouth. Coagulation time before injection 2 minutes; after injection 6 minutes.	Evacuated to United Kingdom (walking case, no oedema) 24 days after onset of thrombosis; returned to duty 80 days after onset of thrombosis.
4	20	Paratyphoid B bacillus in urine	L. femoral vein	22nd day	8 days	25th day: R.11, L.12 $\frac{1}{2}$ 44th day: R.11, L.11 $\frac{1}{2}$	23 days	26th day: 6 oz. of 0.5 % sodium citrate intravenously. Mild rigor 2 hours after injection. No change in diet; no citrates by mouth.	Mental case throughout. Evacuated to United Kingdom (sitting case, no oedema) 32 days after onset of thrombosis.
5	28	Typhoid by agglutination	L. femoral	27th day	11 days	27th day: R.11, L.13 $\frac{1}{2}$ 49th day: R.11, L.11 $\frac{1}{4}$	17 days	27th day: 10 oz. of 0.5 % sodium citrate intravenously. 29th day: 7 oz. of 0.5 % sodium citrate intravenously. Each injection followed by moderately severe rigor 3 hours later. Coagulation time before injection 15 minutes; after injection 6 minutes. No change in diet; no citrates by mouth.	Evacuated to United Kingdom (walking case, no oedema) 31 days after onset of thrombosis; returned to duty 80 days after onset of thrombosis.
6	26	Paratyphoid B by agglutination	L. femoral	43rd day	9 days	44th day: R.10 $\frac{1}{2}$, L.12 $\frac{1}{2}$ 54th day: R.10 $\frac{1}{2}$, L.10 $\frac{1}{2}$	14 days	45th day: 5 oz. of 0.5 % sodium citrate intravenously. 46th day: 5 oz. of 0.5 % sodium citrate intravenously. No change in diet; no citrates by mouth.	Evacuated to United Kingdom (walking case, no oedema) 28 days after onset of thrombosis; returned to duty 10 weeks after onset of thrombosis.
7	24	Paratyphoid B isolated from stools	R. femoral L. femoral	46th day	8 days	49th day: R.14, L.12 $\frac{1}{2}$ 52nd day: R.15 $\frac{1}{2}$, L.12 $\frac{1}{2}$ 57th day: R.14 $\frac{1}{2}$, L.12 $\frac{1}{2}$ 69th day: R.12 $\frac{1}{2}$, L.12 $\frac{1}{2}$	24 days	49th day: 4 oz. of 0.5 % sodium citrate intravenously. 51st day: 10 oz. of 0.5 % sodium citrate intravenously. 55th day: 10 oz. of 0.5 % sodium citrate intravenously.	Evacuated to United Kingdom (sitting case, little oedema) 26 days after onset of thrombosis.
<i>Cases of the Enteric Group treated by the Oral Method.</i>									
8	20	Paratyphoid B isolated from stools	L. femoral	23rd day	10 days	—	19 days	10 gr. citric acid, four times a day; imperial drink ad lib.; no milk	Evacuated to United Kingdom (walking case 29 days after onset of thrombosis; returned to duty 13 weeks after onset of thrombosis. Evacuated to United Kingdom (stretcher case, much oedema) 60 days after onset of thrombosis.
9	27	Paratyphoid B isolated from blood	L. femoral	41st day	10 days	—	60 days	10 gr. citric acid, four times a day; imperial drink ad lib.; no milk	Evacuated to United Kingdom (walking case, no oedema) 35 days after onset of thrombosis; quite fit.
10	25	Paratyphoid B by agglutination	R. femoral	20th day	19 days	20th day: R.13 $\frac{1}{2}$, L.12 $\frac{1}{2}$ 31st day: R.13, L.12 $\frac{1}{2}$	24 days	31 citric acid four times daily; imperial drink ad lib.; no milk	Evacuated to United Kingdom (walking case, no oedema) 31 days after onset of thrombosis.
11	19	Paratyphoid B isolated from stools	L. iliac	16th day	8 days	—	28 days	31 citric acid four times daily; imperial drink ad lib.; no milk	Evacuated to United Kingdom (walking case, no oedema) 40 days after onset of thrombosis.
12	19	Paratyphoid B by agglutination	L. femoral	22nd day	18 days	22nd day: R.10 $\frac{1}{2}$, L.12 $\frac{1}{2}$ 44th day: R.10 $\frac{1}{2}$, L.11 $\frac{1}{2}$	28 days	31 citric acid four times daily; imperial drink ad lib.; no milk	Evacuated to United Kingdom (walking case, no oedema) 18 days after onset of thrombosis.
13	23	Paratyphoid B by agglutination	L. femoral	? 24th day	7 days	—	13 days	10 gr. citric acid four times daily; imperial drink ad lib.; no milk	Evacuated (stretcher case, much oedema) 94 days after onset of thrombosis.
14	23	Paratyphoid A isolated from blood	R. femoral	49th day	15 days	49th day: R.12 $\frac{1}{2}$, L.10 94th day: R.11 $\frac{1}{2}$, L.10	94 days	10 gr. citric acid four times daily; imperial drink ad lib.; no milk	Evacuated to United Kingdom (sitting case, little oedema) 26 days after onset of thrombosis.
<i>Cases of other Fevers treated by the Oral Method.</i>									
15	21	P.U.O.; clinically suggested an enteric infection	L. femoral	25th day	10 days	—	16 days	31 citric acid four times daily; imperial drink ad lib.; no milk	Evacuated to United Kingdom (sitting case, little oedema) 17 days after onset of thrombosis.
16	28	Clinically suggested typhoid	L. femoral	24th day	3 days	27th day: R.13 $\frac{1}{2}$, L.14 $\frac{1}{2}$ 38th day: R.13 $\frac{1}{2}$, L.13 $\frac{1}{2}$	14 days	31 citric acid four times daily; imperial drink ad lib.; no milk	Evacuated as walking case, no oedema, 47 days after onset of thrombosis.
17	23	P.U.O.	Popliteal R. and L.	19th day	15 days	—	23 days	31 citric acid four times daily; imperial drink ad lib.; no milk	

That is when patient first left bed after onset of thrombosis.

* 0.5 per cent. sodium citrate in normal saline.

Conclusions.

From the above series of cases it would appear justifiable to conclude that the treatment of thrombosis by this method does quickly arrest the process. From the patient's point of view the almost immediate release from pain was most gratifying.

The comparison of a series of cases is always a difficult matter. To obviate this difficulty cases were treated alternately by the two methods, and so may be considered unselected. In reality the cases treated by the intravenous method were, on the whole, the more serious.

Analysis of the two groups shows that in those treated by the intravenous method the subsequent period of pyrexia was shorter by an average of four days and the onset of convalescence by an average of ten days than in those cases treated by the oral method.

Reference to the table also shows that the average number of days from the onset of thrombosis to evacuation in the cases treated by the intravenous method was thirty days, while in those cases treated by the oral method it was thirty-nine. It will, moreover, be noted that, whereas in the series of seven cases treated by the intravenous method five were evacuated as walking and two as sitting cases, and in one only was there oedema present on discharge; in the oral series of ten cases, only five were evacuated as walking and three as sitting cases, while two had to be sent to England as stretcher cases, and in no fewer than five cases there was a considerable amount of oedema present on discharge.

The known facts concerning the action of citrates on the blood as worked out by Sir Almroth Wright would lead one to expect that it would lengthen the coagulation time, and in my own series this is confirmed by the experiments in the coagulation time before and after the administration of sodium citrate in the course of treatment recorded above.

Moreover, not only is the coagulation time increased, but the tendency of the blood to coagulate is diminished. This, as is commonly known, can be demonstrated *in vitro*. With the application of citrate to the blood in circulation, in which as in the case of thrombosis a clot has formed, the formation of further clot is apparently prevented, and such clot as is already formed would then seem to behave in the same way as a clot may be seen to behave outside the body—that is to say, it rapidly shrinks, with the expression of serum, and so opens up the venous channels.

The circulation is then speedily restored, the clot being ultimately absorbed. The better results obtained by the administration of the citrate intravenously than orally would seem to be dependent upon the more immediate and intimate manner in which the drug is brought into relation with the blood by the former than by the latter method.

Lectures

ON

THE ANATOMICAL AND PHYSIOLOGICAL PRINCIPLES UNDERLYING THE TREATMENT OF INJURIES TO MUSCLES, BONES, AND JOINTS.

GIVEN AT THE ROYAL COLLEGE OF SURGEONS OF ENGLAND,
NOVEMBER–DECEMBER, 1917.

BY

PROFESSOR ARTHUR KEITH, M.D., F.R.S.,
CONSERVATOR OF THE MUSEUM.

**III. THE PRINCIPLES AND PRACTICE OF
HUGH OWEN THOMAS.**

MEN could not have approached a common objective by more diverse routes than did Hunter and Hilton on the one hand and H. O. Thomas on the other. Hunter and Hilton worked long and hard in the dissecting and *post-mortem* rooms in the search of facts to guide them in formulating rational means of treatment; they observed the efficacy of their methods in regulated hospital wards and on closely tended private patients. The circumstances amidst which Thomas matured his experience were very different: his field of observation was the steady stream of accident cases drawn from the dockland of Liverpool, which passed through his surgery; his field of experiment lay in his workroom, where, in workman's

attire, and with the hand of an expert, he wrought the exact form of splint or machine which he desired for the treatment of each particular case that came under his care. Here, then, is a surgeon of a new kind, one who could and did use his knife, but it was his final and fixed opinion, founded on thirty-three years crowded with experiments on orthopaedic cases, that the blacksmith's hammer, deftly used, was, in most cases, a more powerful reparative instrument than the surgeon's knife.

Thomas was born and bred an orthopaedic surgeon. His father, Evan Thomas, had his home and surgery, so Professor Rushton Parker has informed me, at 72, Great Crosshall Street, Liverpool, where, although unqualified, he was the referee of certain workmen's clubs—dock gatesmen, ship's carpenters, boilermakers, and several others. He had five sons: Hugh Owen was the eldest; two others besides him—Richard and Evan—studied and qualified in medicine; as the boys grew up they had to help in the surgery and could not fail to gain a first-hand knowledge of injury and disease. Indeed, it was proposed at one time that Hugh should become his father's apprentice, but the ill state of his health—he never at any period of his life was robust—led to a change of plan. In 1851, at the age of 17, he was apprenticed to his maternal uncle, Dr. Owen Roberts of St. Asaph. In 1855, at the age of 21, he went to Edinburgh to study medicine. Syme and Spence and Simpson were in the heyday of their fame; Goodsir was teaching anatomy; there was then a demonstrator of anatomy newly arrived from London—William Turner. Lister was also there commencing the Edinburgh phase of his great career. But in Thomas's writings there is never an allusion to his life in Edinburgh; only one man seems to have impressed him there, Hughes Bennett. He apparently spent little more than a year in Edinburgh, for in 1857 he was studying at University College, London; of his life and experience in London he never gives us the slightest hint. In 1857, in his 23rd year, he became a Member of this College, and in the following year, still in poor health, he returned to Liverpool, where he spent the thirty-three years of his professional life amongst the accident cases of Liverpool dockland. He began in easy circumstances so far as finance was concerned. Professor Rushton Parker informs me that he had at first a very hard struggle, but ultimately acquired the surgeonships of the various clubs which had been held by his father, and that thus an annual income of at least £500 was assured him. His cases supplied him with all his interests—his cases and his workroom where he designed, modified, and perfected the methods he applied for treatment. He knew and sympathized with the working people; there were many poor and ailing men round him who could ill afford to leave their work and seek treatment on week-days; Thomas established a Sunday clinic for them—he eased his poor patients and enriched his own store of knowledge.

Thus he lived from his twenty-fourth to his fortieth years, his fame during that period being still confined to dockland. In 1874 an event occurred which led to his labours becoming better known. Mr. Parker, police surgeon, had reason to visit Mr. Thomas, who then had his surgery and hospital in Greta Street, in connexion with a case—that of an inspector of police—who was then under Mr. Thomas's care on account of a compound fracture of the leg. Mr. Parker had taken with him his son, Rushton, a young surgeon of 28, with an assured future in the Liverpool School of Medicine. We can learn the effect of that visit from the preface which Thomas wrote for his first important publication on *Diseases of the Hip, Knee, and Ankle Joints, with their Deformities: Treated by a New and Efficient Method* (London: H. K. Lewis. First edition 1875; third edition 1878). It was not actually his first publication, for in 1873 he described a method of using silver wire ligatures for the treatment of compound fractures of the mandible. The ligatures were fixed by a terminal spiral which could be conveniently tightened as the mandibular fragments yielded. He also advocated then a practice adopted in the present war, of removing any tooth situated on the line of fracture, as its presence there always retarded union. Once started as a writer he continued to issue *Contributions to Surgery and Medicine* almost annually until death brought an end to a laborious career, handicapped by indifferent health. He died in 1891 at the age of 57. Even in the manner of publication he emphasized his individuality. His writings