
DISCUSSION.—Dr. Roy D. McClure, Detroit: Through the years it seems that the deepest scars that have been left on most of us as surgeons have been the results of postoperative deaths that should not have happened. I felt in 1911, while working with Dr. Eugene Poole at the New York Hospital, that massage and early exercise in bed would reduce the number of thromboses and fatal embolisms. Then, while Doctor Halsted's resident at Johns Hopkins Hospital, I saw in five weeks on his service and that of Dr. Howard Kelly five deaths from embolism. We talked with Dr. J. J. Abel and Dr. William Howell about the possible use of hirudin but that proved to be too dangerous. When Doctor McLean, working on anticoagulants in Howell's laboratory, brought out heparin in 1916, we used it immediately after that in Detroit. The reactions, however, due to impurities, were so terrible in three cases that we never even reported it. It was not until about 1937 that the group in Toronto put out a purified heparin which was safe to use. We started using that in all cases which showed beginning pain in the calf of the leg. In spite of that we had in 1938 five deaths from embolism, four in 1940, five in 1941, three in 1942, five in 1943, eight in 1944 and 1945. However, that was in about 900 operations a month so it was not a large percentage, but we felt that these deaths should not have occurred. With the exception of two, not one of these fatal cases had specific treatment; the fatal cases came just as a bolt out of the blue.

The question is, how can we determine which patients will have thrombosis and pulmonary embolism. First of all, we agree absolutely with Doctor Parsons that age helps somewhat in predicting. In our series the occurrence is almost unknown under the age of 30. Certain types of operations, as he said, seem more prone to be followed by embolism. Gastrectomy, for example, led our list with 1.66 per cent deaths from fatal embolism, followed by colon and gallbladder operations and prostate operations, etc.

At the present time we have our residents watch very carefully the temperature chart and the calves of the patient's legs, during the postoperative period. An unexplained fever may lead to the giving of anticoagulants, on suspicion. The finding of a positive Homan's sign indicates to us immediately either a femoral ligation at the indicated site, or anticoagulant therapy, or possibly both. We give heparin immediately after operation. We used to give it preoperatively, but we had a few hemorrhages in the wound and gave that up. It takes about two days to get a positive effect from dicumarol. I speak knowingly because I myself have been taking it daily for ten months. What we have all hoped for was that someone would show us a test which would indicate to us an impending thrombosis and which would anticipate all the clinical signs just mentioned. Such a test was hopefully reported at the meeting of the American Surgical Association in Quebec last May by Mahoney and his associates.* Perhaps Doctor Morton can tell us of their subsequent experience.

They believe they have demonstrated the value of daily prothrombin times during the postoperative period, and if that time went down they would start anticoagulants. When I returned to Detroit from that meeting I was very enthusiastic about this and asked Doctor Barron and Doctor Lam of our staff to start this method immediately to see if we could anticipate which patients would have thrombosis and embolism. To date we have studied 179 cases. Each patient has eight prothrombin tests; one the day before operation, two the day of operation, and then one each day following operation for five consecutive days. Of the 179 cases, six have shown leg thrombosis without infarction, four have shown high prothrombin levels, but in two there was no elevated prothrombin as a warning. I feel sure our prothrombin estimations are well done. Seven patients had clinical phlebothrombosis of the lower extremity and also non-fatal pulmonary infarcts.

WIGGINTON, PARSONS AND PURKS

In only two of these did the prothrombin curve give a warning; the other five showed perfectly straight prothrombin curves. One patient had a coronary occlusion during the study. His prothrombin curve gave no warning. As you know, the American Heart Association feels that in coronary thrombosis dicumarol has been a great aid in cutting down the number of deaths.

Of the patients who had had no clinical evidence of thrombosis, 64 had so-called elevation of prothrombin level and 98 were negative. We cannot say for certain that the 64 positives had no thrombosis, but they were not treated, and we have no reason to regret that we did not start anticoagulant therapy on them.

In summary, we have not been able to corroborate the findings of Mahoney and his associates that daily prothrombin determinations are of great value in predicting thrombotic complications, but further studies along this line are strongly indicated.

DR. GEORGE T. McCUTCHEON, Columbia, S. C.: I would like to recite an experience that lends emphasis to Doctor Parson's idea that it is well in some cases, particularly in those in whom you suspect embolism is likely to occur, to give some type of prophylactic medication. A little more than a year ago I encountered a patient who started with a primary disease in his chest. He was seen by his physician, X-ray films were taken, his clinical manifestations were similar to those of a small pulmonary embolus. He had pain in the chest and coughing of blood, but embolus was not suspected at the time. About two days later, on further questioning, it was found that he had had some soreness of his leg, in the calf, for three or four days prior to onset of symptoms.

On examination of the leg it was found that he had a positive Homan's sign, some swelling and tenseness of the calf. Ligation of the femoral vein was done and a clot was found at the point of ligation. During the next three or four months I encountered two other cases of a similar nature and was completely puzzled by the problem. I wrote to Dr. Mims Gage and Dr. Alton Ochsner, and they told me that so far as they knew this particular thing was unique; that they had never heard of it. Since that time there has been a total of seven patients with a similar syndrome; all in patients who had had no bed rest and in whom the leg had not been subject to trauma. We had to call in a term for it, and picked out one that rolled off the tongue very easily—spontaneous unprovoked phlebothrombosis. The last patient we saw might be questionable to some extent; a boy aged 15 who had had an appendectomy some five weeks before. He was ambulated immediately and had a completely uneventful convalescence. Nine weeks later, as he was entering an elevator, he was suddenly seized with pain in the chest and severe cough, and it was discovered that he had physical and X-ray indications of a pulmonary embolism, with pain, tenderness and swelling of the calf. That was the only one of the number who had, in recent weeks at least, been confined to bed.

It brings up an important point in diagnosis for our fellow internists as to some obscure conditions of the chest, and I wonder how often it might happen in cases of sudden death. None of these patients died; all had emboli of rather mild nature. We are completely at a loss as to the explanation of the cause of phlebothrombosis, I believe. Certainly we have to scratch our heads or stretch a point to believe that bed rest or trauma has much to do with it. So far as I am concerned it is still an imponderable problem, and one which is a long way from solution, and I believe it falls within the realm of the biochemist and the physiologist to produce the answer. I don't believe surgeons can. All these patients were subjected to elaborate study of the clotting mechanism and no abnormalities could be found. It remains for more delicate tests to be developed, or to find factors other than those we now understand as being causative in phlebothrombosis.

DR. FREDERIC W. BANCROFT, New York: Our experience with the tests for phlebothrombosis has been a good deal different from that of Doctor McClure. Doctor Stanley-Brown and I started this study in 1928, and at that time we made more than 4000 tests routinely on our surgical cases. It may be that the reason our results showed more was because at that time we did both prothrombin and fibrinogen tests, but in the series

794
we studied we had only one case, a breast amputation, done at another hospital, where there was no elevation of the clotting factors. The others had either high prothrombin or fibrinogen, and in the cases that developed accidents on routine study they had had warning signs before. So I do feel that we may find some method to ascertain which are the dangerous cases.

I was interested in Doctor Parsons’ statement about the high percentage in hysterectomies. I think we have here possibly a different picture than in operations above the pelvis. I wish to report one case that illustrates what I mean. A woman had had a cesarean operation; a perfectly normal postoperative temperature; she had gone home, and on the tenth day afterward she had pain in the chest. She was brought back to the hospital and on X-ray it was a question as to whether it was an infarct or a virus pneumonia. Her legs were carefully measured; there was no tenderness and no swelling. However, seven days after onset of the pain in the chest, she had evidence of phlebothrombosis in the right thigh. I operated on her and took out the thrombus, but did not have a satisfactory suction apparatus, so did not feel that I had gotten entirely above the thrombosis. However, she apparently improved, but at the end of eight days after this she had pain in the chest again, and the X-ray showed an infarct in the opposite chest; seven days later she showed obvious thrombosis on the right. I operated on that side and she was given spinal anesthesia which I think was a mistake, because in flexing her forward I think they liberated another thrombus; anyway she died. It was obvious from the postmortem that the thrombosis had occurred primarily in the pelvic vein and had proceeded in a retrograde manner down her thigh.

A similar case is that of a woman who had had a normal delivery, and on the seventh postoperative day had pain in the chest, and X-ray evidence of pulmonary infarct—no evidence in the thigh. We went in simultaneously on both femoral veins; they were in spasm and did not contain a clot, but in passing a suction tube up to near the bifurcation of the vena cava we got clots from both sides and with anticoagulant therapy she went home five days later.

I think it is a difficult thing to know whether or not to use anticoagulants, or operation. I have done 35 thrombectomies, where the thrombus has extended above the femoral ligament. That one case is the only case of death; the rest have recovered satisfactorily. I have also given anticoagulants. I have not operated on any patient who had had a thrombosis for about ten days before being seen by me because I felt if they had thrown no embolus by that time, they were not going to, and that they could be treated by lumbar sympathetic block. I would like to say one word about our concept of early ambulation. Many of us have thought that if we got patients up and let them sit in a chair we were giving early ambulation. As a matter of fact that is the worst position we could possibly put them in for venostasis. The feet are much lower than they are in a Gatch bed, the knees are over the edge of the chair where there is compression on the popliteal veins, and there is sharp flexion of Poupart’s ligament—all factors, I think, that aid venostasis. It is my idea that early ambulation is to get a patient out of bed, make him walk around the bed, get back into bed and lie flat, and not sit part of the time in a chair in as poor a position, probably, as we can get.

I wish to thank Doctor Parsons, and I enjoyed his paper very much.

DR. HUGH A. GAMBLE, Greenville, Miss.: In the discussion of Doctor Parsons’ paper there has been little said about the prophylaxis of postoperative phlebitis or thrombophlebitis. In 1934 I published in the American Journal of Surgery a description of what I called a bedcycle and later, in discussing a paper before this Association, described our method of preventing thrombophlebitis. Up to that time we were losing about one case out of every 1500 operations from pulmonary embolism. We had what seemed to be an epidemic of thrombophlebitis. A large proportion, at least one-third or one-fourth of the operative cases, would develop the condition and this began to be so much the case that patients in coming to the hospital would inquire as to what caused it and if there was any way to prevent it.

795
We went over the medical literature for the preceding 40 years. We found that Aschoff gave the most lucid description of the pathogenesis of this condition that we had seen. Adopting Aschoff's theory as to the causation of thrombophlebitis we had built what has been called a bedcycle. It is simply two bicycle pedals mounted upon a frame so that it can be placed upon the bed, and the patient is required to exercise his lower extremities on it for a period of five minutes twice a day, beginning the day of operation. While we felt that this was of extreme importance, we realized that during and following operation there is marked concentration of the blood, there is marked increase in the clotting elements present, and that it is at this time that the venous flow is at its lowest level. Therefore, we made it a practice of giving intravenous glucose in 5 per cent solution continuously through operations in an endeavor to maintain blood water levels. In order to promote the flow of venous blood we have routinely, for the first 24 hours following operation, required that these patients be given inhalations of carbon dioxide and oxygen every three hours for three minutes. Later on we began to get them up early, usually, where practicable, within the first 24 hours, requiring them to walk around the room. However, regardless of this we have maintained the use of the bedcycle throughout the whole period of hospitalization.

Since 1933 my brother and I have had something over 52,000 major operative cases. We have had one death from pulmonary embolism during this time. We have had seven cases of thrombophlebitis and I cannot but feel that these preventive measures have been effective in lowering the incidence of this condition.

The one death I speak of occurred in a man on the 21st day following operation. He had had a resection of the large bowel for carcinoma of the ascending colon and an end-to-side ileo-colostomy. He had had no symptoms suggestive of phlebitis of the lower extremities, but on that day he complained of pain in his lower abdomen and thought that he had eaten something that had disagreed with him. He was given an enema but was not entirely relieved of discomfort. At four o'clock in the morning of the 21st day I was called to see him and when I reached the hospital found him dead. An autopsy showed a large embolus in the right pulmonary artery, which had originated in the veins draining the transverse colon.

I feel that there is not enough attention paid to the fact that these embolic processes begin immediately after operation when the blood is at its highest concentration and the venous flow is at its lowest level and that, if you employ measures that will keep the water level of the blood up to normal and promote the flow of blood from the outset and keep it up from day to day, you will be convinced of the efficaciousness of this method of preventing these complications, which in some sections of the country seem to be so prevalent.

Dr. Roy C. Wigginton, Vicksburg (closing): We would like to thank those who have so kindly and so generously discussed our paper. We, of course, do not believe that either anticoagulant therapy or vein interruption represent necessarily the final answer as to the therapy of venous thrombosis. We do believe, however, that perhaps careful evaluation of the risk in each surgical patient, determined by the criteria based upon the experiences of many who have studied the problem, will indicate those patients who should have dicoumarol as a prophylactic measure. We believe that the intelligent use of dicoumarol in this fashion might perhaps greatly reduce the mortality and morbidity incident to venous thrombosis, and might do so without posing impossible laboratory problems or unduly endangering lives.

At the Tulane University, and I believe at other places both in America and abroad, a great deal of work is now being done on the mechanism of blood clotting. At least two previously unknown factors have been discovered. As a result of this study many of the previous theories pertaining to blood coagulation may perhaps be discarded. It is further possible that those now investigating this phase of the subject may eventually solve the problem. Certainly, at the moment, its solution is by no means complete.