predominantly "carbohydrate" defect, cortisone alone was used for maintenance therapy.

As soon as the hypoglycaemia was recognized the patient was restored to consciousness with the intravenous administration of glucose. After this, oral glucose feeding sufficed to maintain adequate blood-sugar levels.

It is suggested that the possibility should be borne in mind that hypoglycaemia may be contributing to loss of consciousness in the Addisonian patient. It would therefore seem worth while to estimate the blood-sugar level as a routine in this type of case.

Summary

The case of a woman with Addison's disease presenting in hypoglycaemic coma is described. The coma responded immediately to the administration of intravenous glucose. The differential diagnosis is briefly discussed.

I wish to thank Dr. D. H. Makinson for his interest and help in the preparation of this paper and for permission to publish, and Professor H. L. Sheehan for valuable criticism.

REFERENCES

Banks, H. S., and McCartney, J. E. (1943). *Lancet*, **1**, 771.
Maddock, W. O., Leach, R. B., Klein, S. P., and Myers, G. B. (1953). *Amer. J. med. Sci.*, **226**, 509.
Magnusson, J. H. (1934). *Acta. paediat. (Stockh.)*, **15**, 396.
Rushton, J. G., Cragg, R. W., and Stalker, L. K. (1940). *Arch. intern. Med.*, **66**, 531.
Simpson, S. L. (1932). *Quart. J. Med.*, **1**, 99.
—— (1953a). *British Medical Journal*, **1**, 1110.
—— (1953b). *Proc. roy. Soc. Med.*, **46**, 566.
Thorn, G. W., Forsham, P. H., and Emerson, K. (1951). *The Diagnosis and Treatment of Adrenal Insufficiency*. Thomas, Springfield, Illinois.
—— Koepf, G. F., Lewis, R. A., and Olsen, E. F. (1940). *J. clin. Invest.*, **19**, 813.
Welty, J. W., and Robertson, H. F. (1936). *Amer. J. med. Sci.*, **192**, 760.

LOW TERMINATION OF THE INTERNAL SAPHENOUS VEIN

BY

P. G. BEVAN, M.B., F.R.C.S.
Lecturer in Surgery

S. H. GREEN, M.B., Ch.B.
Lecturer in Anatomy

AND

F. A. R. STAMMERS, C.B.E., M.B., Ch.M., B.Sc. F.R.C.S.
Professor of Surgery

From the Department of Surgery, the Queen Elizabeth Hospital, and the Department of Anatomy, the Medical School, Birmingham

The Trendelenburg* operation for varicose veins, usually in combination with ligation of the main communicating vessels above and below the knee, is one of the most commonly performed in surgery. In terms of alleviation of symptoms, both annoying and incapacitating, a countless multitude of patients must have derived benefit from its effects. In all centres where the operation is performed carefully in the classical method surgical complications are few. When such a complication does occur, however, it is often a tragedy, and may result in the crippling of a young and healthy person undergoing a relatively minor operation for a non-urgent condition.

Accidental ligation of the femoral vein would be expected as a recognized hazard of the operation in view of its relation to the internal saphenous vein, but has rarely been reported. Daseler *et al.* (1946) record one case in which the femoral vein was ligated and severed, but give no details of the anatomy or subsequent procedure. Wells (1948) describes the immediate repair of a femoral vein tied off in the course of a high ligation operation for varicose veins, with no deleterious effects. McPheeters (1945) reports a femoral vein and artery ligated together. Luke and Miller (1948), in their paper entitled "Disasters following the ligation and retrograde injection of varicose veins," review 21 cases with 4 fatalities; these are divided into two groups, operative and thrombotic. In the former no femoral vein ligation is recorded and no abnormal anatomy mentioned. Homans (1949) stresses that interruption of the femoral vein tends to result in chronic oedema, venous engorgement, and post-phlebitic induration and ulceration.

This report presents four cases, all of which have been admitted on or transferred to the same unit during the past three years.

Case 1

A man aged 23 gave a history of bilateral varicose veins for four years. He underwent operation on December 27, 1952. An uncomplicated Trendelenburg procedure was carried out on the left side with venous ligations in the thigh and leg. On the right side the internal saphenous vein entered the femoral at a lower level than normal, at the apex of Scarpa's triangle, and the femoral vein was accidentally tied. In the post-operative period the superficial veins remained tightly distended and the leg became oedematous. However, the patient was discharged on January 10, 1953, with the wounds healed and the swelling controlled by a blue-line bandage.

He was readmitted on May 29, 1954, with chronic oedema of the leg and small ulcers on the anterior and medial aspects of the ankle. Varicosities were present in the distribution of the internal saphenous vein. With rest in bed and eusol dressings the ulcers healed and he was discharged on June 15. On his third admission on February 5, 1955, the areas of ulceration were again present, the varicosities more obvious although less tense, and the swelling of the leg had become less over the previous year. Trendelenburg tests showed that the deep venous return was functioning. A further operation was performed on February 14 in which the varicosities in the calf and above the knee were ligated without post-operative increase in the oedema. He was discharged on February 23 with the ulcers healed.

Case 2

A housewife aged 38 was admitted on May 27, 1955, from another hospital where on the previous day the left femoral vein had been accidentally ligated following a haemorrhage in the course of a bilateral Trendelenburg operation. The foot was warm but pulseless, a little cyanosed, and anaesthetic. The patient was mentally confused. The wound was immediately opened under general anaesthesia, when it was confirmed that the femoral vein had been ligated and divided, and it was seen that the saphenous vein entered the femoral vein a good 5 cm. below the groin. The femoral artery was in marked spasm, but there was no sign of damage to its wall. After this second operation the patient developed oliguria, albuminuria, and uraemia, which was felt to be due to lower nephron nephrosis. The possible causative factors of this condition could have been one or more of the following: (1) prolonged hypotension following the severe haemorrhage of the first operation, leading to renal anoxia; (2) trauma to the limb and ligation of the femoral vein causing spasm of the femoral artery, which extended proximally to involve the renal arteries (Trueta, 1947); and (3) congestion and necrosis of the muscles consequent on ligation of the femoral vein with effects similar to the crush syndrome. The blood urea had risen to 430 mg. per 100 ml. on June 3.

*The high ligation of the internal saphenous vein at the sapheno-femoral junction was first described by Homans in 1916. The operation actually recorded by Trendelenburg (1890) was simple ligation of the internal saphenous in the mid-thigh. The term "Trendelenburg operation" as denoting the high ligation is retained in this paper as it still receives widespread acceptance. The internal saphenous vein of this paper is the vena saphena magna of the *Nomina Anatomica* (1955 revision).

Because of increased tension in the leg a further operation was performed on June 4 when the deep fascia was divided by longitudinal incisions extending from knee to ankle. The anterior tibial muscles were found to be yellow, bloodless, and insensitive to mechanical stimulus. Diuresis followed two days later, and by June 18 the blood urea was down to 70 mg. per 100 ml. A final operation was carried out on the leg on July 4, consisting of excision of the necrotic anterior compartment muscles. The permanent effect on function was a severe degree of foot-drop but full power in the posterior and peroneal muscles. The patient was discharged on July 31; her general condition was completely satisfactory, renal function having returned to normal, and she was walking well with a calliper fitted with a spring elevator to aid dorsiflexion.

Case 3

A stores assistant aged 15 was admitted to hospital on June 26, 1955, with a history of varicose veins in the left leg for the past two years. On examination the main incompetent communication in the thigh was found at the level of the apex of Scarpa's triangle. At operation on June 30 an anatomical abnormality was found in the groin. The internal saphenous vein was seen to pass laterally, curving near the inner border of sartorius to enter the lateral aspect of the femoral vein at a much lower level than normal. It was ligated together with its tributaries: the two upper tributaries entered the femoral vein directly. He was discharged without complication on July 4.

Case 4

A machine viewer aged 22 was admitted to hospital on September 24, 1955, with a history of aching in the left calf for the previous 18 months. Examination showed varicosity of the internal saphenous vein and its tributaries. At operation there was no sign of the internal saphenous vein at a point 4 cm. below and outside the pubic tubercle. Instead it was found to enter the femoral vein 4 cm. below this point. The incision was extended downwards and the operation completed without complication. He was discharged on September 29 with no post-operative troubles.

Discussion

All four cases illustrate a venous abnormality at the fossa ovalis that has not received adequate description or emphasis—namely, entrance of the internal saphenous into the femoral vein at an abnormally low level. In Cases 1 and 2 the femoral vein was ligated by mistake (Case 2 at an outside hospital), and it is felt that the anatomical abnormality was mainly responsible for the error, although in Case 2 severe haemorrhage contributed.

The practical importance and medico-legal implications of such low terminations are obvious. When the proximal tributaries from the abdominal wall enter the femoral vein directly, and when this arrangement is combined with a low opening of the saphenous vein into the femoral, a real danger exists if the surgeon does not visualize the condition. In the "classical Trendelenburg" operation the incision, centred on a point 4 cm. outside and below the pubic tubercle, will give direct exposure to the femoral vein (see Fig. 1). This may be ligated before it is realized that there is no overlying internal saphenous vein. The illusion is more complete if one or more of the tributaries usually entering the internal saphenous outside the saphenous opening is found to enter the femoral vein directly above the sapheno-femoral junction (as in Case 3).

Edwards (1934), Daseler *et al.* (1946), Mansberger *et al.* (1950), and Allan (1951) have described the tributaries and collaterals of the saphenous vein, and the last three authors have attempted classification. In all these series of dissections all the tributaries are described and depicted as ending in the internal saphenous vein and arranged into groups according to the patterns of their termination. None of these authors record an actual instance of low termination of the internal saphenous vein, but all describe or figure

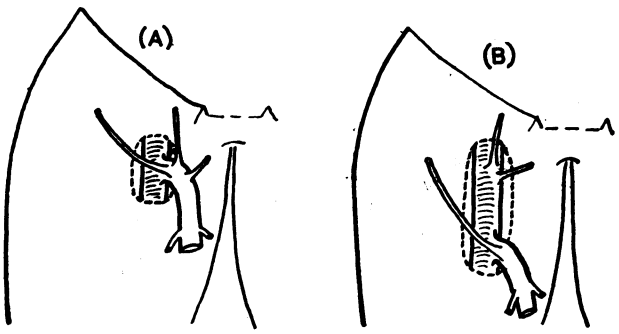

FIG. 1.—(*a*) The normal arrangement at the sapheno-femoral junction. (*b*) Example of a low termination of the internal saphenous vein. The superficial external pudendal and superficial epigastric veins are seen to be entering the femoral vein directly; the superficial circumflex iliac vein and the internal and external accessory saphenous veins are entering the internal saphenous vein. The fossa ovalis is long and narrow.

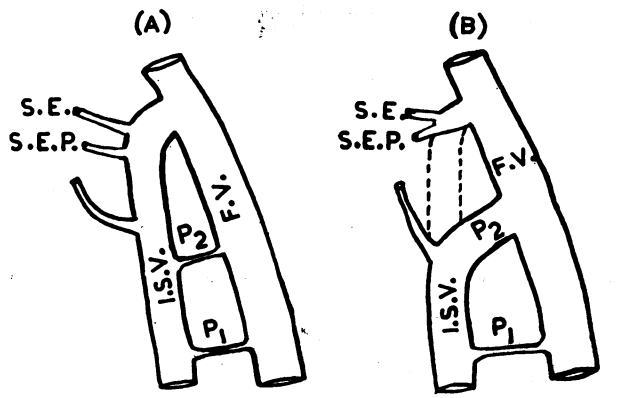

FIG. 2.—(*a*) Diagram (after Sherman) to show normal relationship of internal saphenous vein (I.S.V.) and femoral vein (F.V.). The two highest perforating veins are shown—the mid-Hunter perforator (P1), and the perforator 6–8 cm. below the sapheno-femoral junction (P2). (*b*) Low termination of internal saphenous vein due to overdevelopment of the high perforating vein (P2). The superficial external pudendal (S.E.P.) and superficial epigastric (S.E.) veins enter the femoral vein directly.

entrance of the superior saphenous tributaries into the femoral vein. Foote (1954) also gives brief references to this condition. Cathro (1954) records a variation of this low termination of the internal saphenous vein revealed in the course of routine laboratory dissection. In the case he describes the vein winds around the lateral aspect of the femoral artery, and the point of entry into the femoral vein is 7 cm. below the mid-inguinal point. He clearly indicates what the practical importance of this anomaly might be.

Anson and McVay (1938) describe 200 dissections to show the fossa ovalis and its related blood vessels. In 90% of their cases the fossae measured between 3 and 6.4 cm. in length, the longest being 8.5 cm. and the average 4.6 cm. Unfortunately no records of the exact position of the sapheno-femoral junction are included, although (as pointed out by Cathro) their Figure 5 is very similar to the case Cathro reported. Anson and McVay state that the fossa may be long and narrow, thus presumably allowing a low entrance of the internal saphenous into the femoral vein. On rare occasions, however, the internal saphenous vein may terminate below the level of the fossa. Glasser (1943) records one instance (out of 100 dissections of the groin) in which the internal saphenous pierced the deep fascia and joined the femoral 2.5 cm. below the fossa ovalis. He further states that the frequency of superior saphenous tributaries opening independently into the femoral vein is 16% of all specimens.

The long saphenous vein has well-recognized connexions with the deep veins. Sherman (1944) illustrated the constant mid-Hunter perforator and a less frequent perforator

6–8 cm. below the sapheno-femoral junction. At the lower end of the adductor canal tributaries of the highest genicular veins often connect with the saphenous veins (Daseler *et al.*, 1946; de Takats and Quillin, 1933). In addition, deep perforating veins are not infrequently present in the region of the fossa ovalis. If any one of those connexions is over-developed the internal saphenous vein will terminate short of the fossa ovalis (Kopsch, 1955) and the usual upper segment entering the femoral vein 4 cm. below the pubic tubercle will be missing (see Fig 2).

It is thought that the risk of ligating the femoral vein in cases where such an abnormality is present would be minimized by adopting the following measures as a routine: (1) Operations to be performed on in-patients under general anaesthesia, thus facilitating unhurried dissection and exposure of the vessels. (2) To realize the existence of the above anatomical variation from normal. (3) Dissection to be carried out of all the expected tributaries of the internal saphenous vein to make sure of their mode of termination before dividing and ligating the main vein. Although one or more of these tributaries may enter the femoral vein above the level of the internal saphenous, some of these tributaries always enter the internal saphenous vein. (4) In cases of suspected abnormality there should be no hesitation in extending the incision distally to expose the highest existing communicating vein between femoral and internal saphenous systems. (5) When the supposed internal saphenous is dissected free, palpation of the femoral artery lateral to it prior to ligation. The artery should be on a plane deep to the veins. (This measure saved the femoral vein from ligation in Case 4.)

Three of these four patients were aged 15–23. When patients of such a young age group present with varicose veins causing symptoms, an anatomical abnormality such as low termination of the internal saphenous vein should be considered.

Summary

Four cases of entrance of the internal saphenous into the femoral vein at an abnormally low level are recorded.

In two of these cases the femoral vein was ligated. The anatomical abnormality was considered to be mainly responsible for the error.

Measures are suggested as an aid to preserve the femoral vein in similar cases.

We should like to thank Mr. A. J. H. Rains for supplying details of Case 3.

REFERENCES

Allan, J. C. (1951). *S. Afr. J. med. Sci.*, **16**, 105.
Anson, B. J., and McVay, C. B. (1938). *Anat. Rec.*, **72**, 399.
Cathro, A. J. M. (1954). *British Medical Journal*, **1**, 918.
Daseler, E. H., Anson, B. J., Reimann, A. F., and Beaton, L. E. (1946). *Surg. Gynec. Obstet.*, **82**, 53.
de Takats, G., and Quillin, L. (1933). *Arch. Surg. (Chicago)*, **26**, 72.
Edwards, E. A. (1934). *Surg. Gynec. Obstet.*, **59**, 916.
Foote, R. R. (1954). *Varicose Veins*, 2nd ed., p. 142. Butterworth, London.
Glasser, S. T. (1943). *Arch. Surg. (Chicago)*, **46**, 289.
Homans, J. (1916). *Surg. Gynec. Obstet.*, **22**, 143.
—— (1949). *Surgery*, **26**, 8.
Kopsch, Fr. (1955). *Rauber-Kopsch Lehrbuch und Atlas der Anatomie des Menschen*, 19th ed. Thieme, Stuttgart.
Luke, J. C., and Miller, G. G. (1948). *Ann. Surg.*, **127**, 426.
McPheeters, H. O. (1945). *Surg. Gynec. Obstet.*, **81**, 355.
Mansberger, A. R., jun., Yeager, G. H., Smelser, R. M., and Brumback, F. M. (1950). Ibid., **91**, 533.
Sherman, R. S. (1944). *Ann. Surg.*, **120**, 772.
Trendelenburg, F. (1890). *Beitr. klin. Chir.*, **7**, 195.
Trueta, J. (1947). *Studies of the Renal Circulation*. Blackwell, Oxford.
Wells, C. (1948). *Brit. J. Surg.*, **36**, 97.

The Philips organization at Eindhoven, Netherlands, has begun publication of a quarterly journal, *Medicamundi*, devoted to material on radiology, "electromedicine," electrotherapy, and related subjects. The first three issues have already appeared; these include papers on radioisotopes, colposcopy, *x*-ray film, "small-volume irradiation," ultraviolet treatment of psoriasis, etc. *Medicamundi* is obtainable in Britain from Cleaver-Hume Press, Ltd., 31, Wright's Lane, London, W.8, price 21s. per annum.

A QUANTITATIVE CHEMICAL TEST FOR "MICROSCOPIC HAEMATURIA"

BY

H. WYKEHAM BALME, M.D., M.R.C.P.

A. ERIC DORMER, M.B., M.R.C.P.

AND

LIONEL RAWLINGS

From the Professorial Medical Unit, St. Bartholomew's Hospital, London

The detection of very small quantities of blood in urine is best performed by microscopical examination of the centrifuged deposit, from which a rough estimate of the amount of blood present can be made. This is usually expressed in such terms as "5–10 red cells per 1/6-in. field," but often there is no consistent usage of descriptive terminology. Unfortunately, great inaccuracies arise unless a highly standardized technique is used (Addis, 1925, 1926; Goldring and Wyckoff, 1930; Lyttle, 1933), and these techniques are inevitably complex, with the major discrepancy occurring in the estimation of the red blood cells. One of the principal difficulties, apart from errors of centrifugation and sampling, is the variable appearance of phosphates, round uric acid crystals and other particles which may be difficult to distinguish from red cells or by diluting the red-cell content of the deposit lead to very large errors in estimating the amount of blood present.

Chemical methods are largely immune from this difficulty, and it is the purpose of this paper to show that if applied to centrifuged urine they are not only delicate enough for most purposes but can at the same time give a satisfactorily quantitative answer very simply.

The method used was the amidopyrine test for blood, in which iron-containing blood derivatives catalyse the oxidation of amidopyrine by hydrogen peroxide to produce a lilac colour. As a result of a number of preliminary experiments in which varying amounts of blood of known characteristics were added to normal saline or normal urine and tested as described below, it was found that a urine containing 5,000 red blood cells per millilitre would just give a positive reaction.

Details of Method

Five and 10 ml. of urine are pipetted respectively into separate conical centrifuge tubes and spun. Centrifugation is carried out for 10 minutes at 3,000 r.p.m. From each specimen all but 0.2 ml. of the supernatant fluid is carefully removed and the volume made up to 1 ml. with distilled water. Then 0.2 ml. of 30% acetic acid is added, followed by 1 ml. of alcoholic amidopyrine, and, finally, by 3 drops of 20-volume hydrogen peroxide.

The tubes are well shaken and the production of a lilac or mauve colour is noted up to three minutes. If neither is positive the urine is reported as containing fewer than 5,000 red cells per ml. If the precipitate from the 10-ml. tube is positive and that from the 5 ml. negative, report as approximately 5,000 red cells per ml. If both are positive, proceed by making serial dilutions as follows.

Five-millilitre samples of normal urine—that is, urine containing no detectable red cells—or normal saline if this is preferred, are pipetted into each of six graduated conical centrifuge tubes, and into the first tube is also pipetted 5 ml. of the urine to be tested. Mix well, and from this tube pipette 5 ml. into the next tube and so on, the final 5 ml. being discarded. These tubes now contain serial dilutions from 1:2