DO THROMBOSIS AND RECANALIZATION PRECEDE DILATATION OF CALF-COMMUNICATOR VEINS?

WILLIAM GIRDWOOD, CH.M. (RAND), F.R.C.S., F.R.C.S.E., B.SC. (MED.), *Department of Surgery, University of the Witwatersrand and Johannesburg Hospital*

Doubt as to the truth of the view that the usual cause of abnormal incompetent communicator veins of the calf is due to destruction of valves by thrombosis[1] stems from the common finding of dilated communicator veins of the calf in uncomplicated cases of simple varicose veins.

The incidence of thrombosis and recanalization in dilated communicator veins found in cases with varicose veins is investigated in this study.

METHOD

Sixty-five consecutive cases with varicose veins were studied with a view to subjecting dilated calf-communicator veins to histological examination. The investigation was divided into 2 series.

Series I consisted of 30 cases of varicose veins in which the exposure was designed to assess the sites of all the communicator veins traversing the posterior compartment of the calf. There were 148 communicator veins, and 131 were subjected to histological investigation.

Series II studied 35 further cases in which often a smaller exposure was employed over known sites of expected dilated communicator veins. In others the exposure was adapted to the case, being sometimes more extensive, and in many the communicator veins were followed to their terminations by incising the deep fascia from the inside.

Experience has shown the differences between normal and abnormal vessels. Only definitely dilated communicator veins were excised and subjected to histological examination.

The criteria of thrombosis required some consideration, and certain instances of intimal thickening were considered as non-thrombotic (Figs. 1 and 2). In these a high palisade intimal arrangement was present. In the lower leg a contracted vein appears to have a thickened intima (Fig. 3).

Thickening of the intima may be found in the neighbourhood of normal valves (Fig. 4) and over the thickened muscular portions of a 'thick-thin' phenomenon seen frequently in varicose veins (Fig. 5). In some instances the intimal thickening is unexplained but appears to be non-thrombotic, filling the area where an apparent defect is found in muscle (Fig. 7).

Any obvious intimal thickening in the first series of cases, where thrombosis could conceivably have been the cause of the thickening, was labelled 'thrombosis' (Fig. 6). Thus, in the first series of cases, a most lenient analysis favoured the number of cases regarded as post-thrombotic.

In the second series the analysis concentrated on the differentiation of degrees of eccentric thickening of intima which were regarded as non-thrombotic, and definite thrombosis and recanalization.

The histological opinions on all cases were those of Prof. J. F. Murray of the South African Institute for Medical Research.

RESULTS

Sites of Calf-Communicator Veins

In the first series of cases the sites of the dilated calf-communicator veins are shown in the accompanying diagram (Fig. 10). Of the 148 communicator veins the highest percentage, 27%, occurred in the postero-medial calf region.

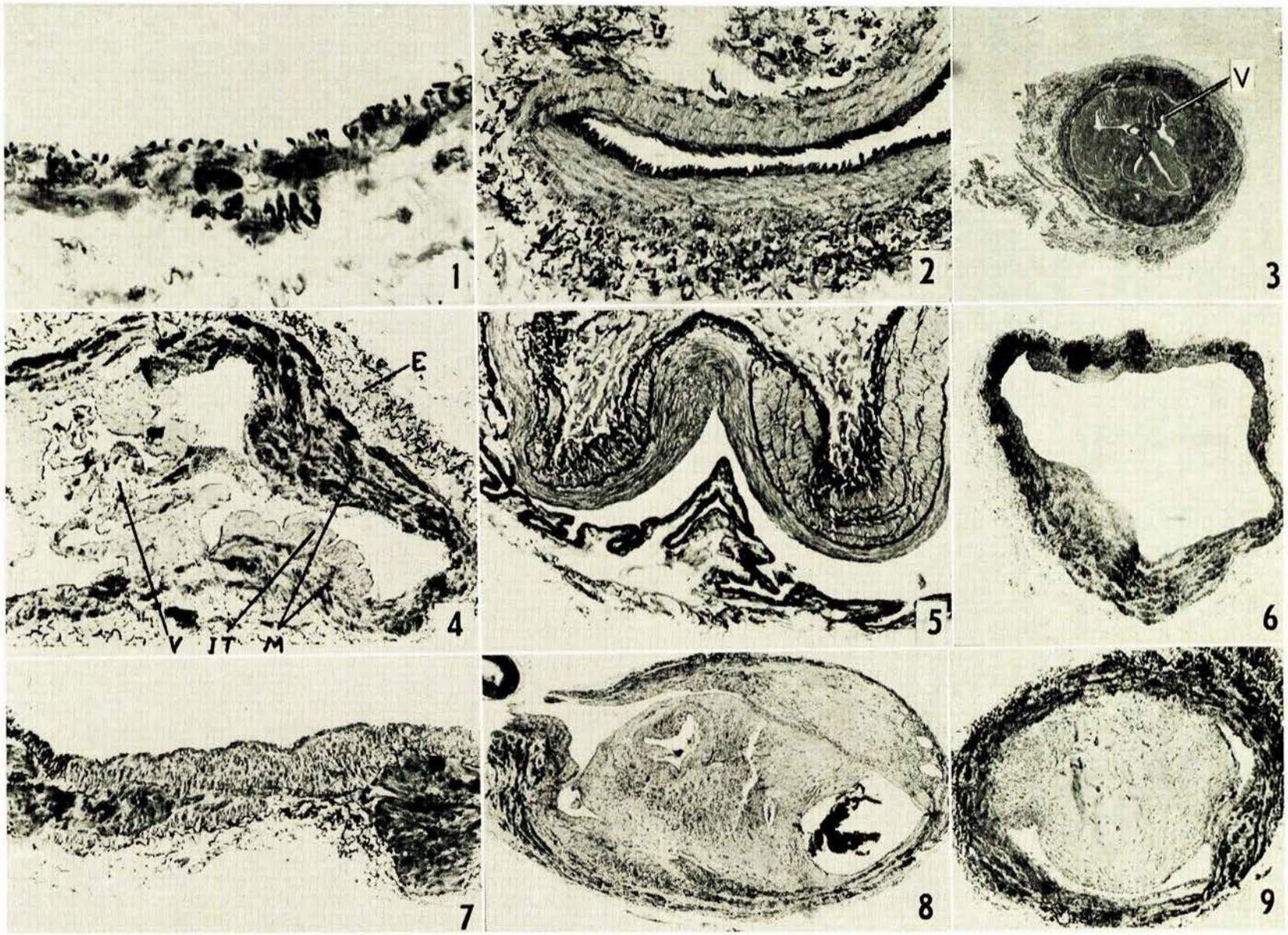

Fig. 1. High endothelial lining in dilated communicator vein. *Fig. 2.* Palisade high intima in dilated communicator vein. *Fig. 3.* Clinically abnormal dilated communicator vein in lower leg now in contracted state showing thickened intima and normal valve. *Fig. 4.* Intimal thickening in relation to normal valve. IT = normal intimal thickening, M = muscle, E = elastic fibres, V = valve. *Fig. 5.* Thick-thin phenomenon. *Fig. 6* Intimal thickening called thrombosis. *Fig. 7.* Focal intimal thickening in defect of muscle. *Fig. 8.* Thrombosis and recanalization. *Fig. 9.* True occlusive thrombosis.

A limited medial incision[2] would expose only 34% of these communicators. The study of the sites of dilated communicator veins in this first series indicated the wide exposure needed to find them, but it also allowed a histological study of dilated perforators of the posterior calf wherever they were to be found.

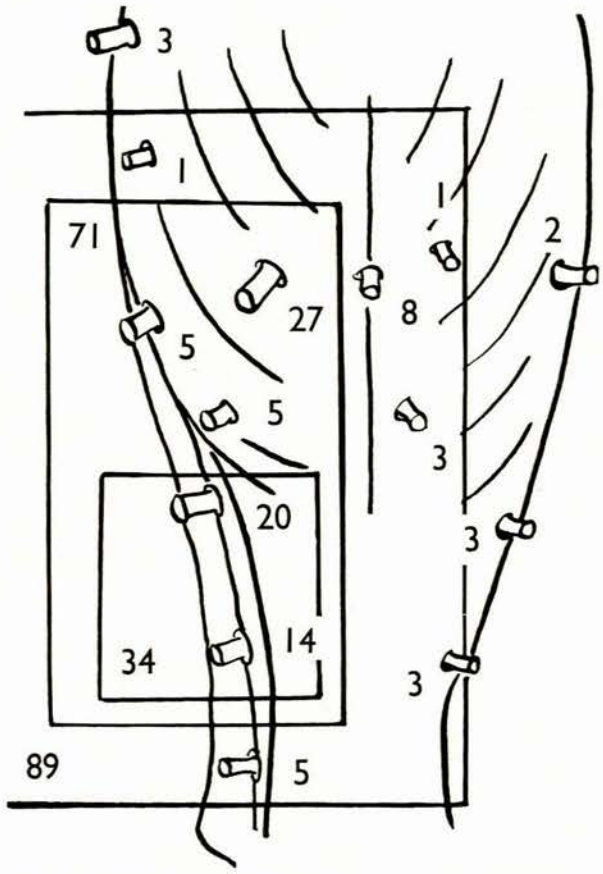

Fig. 10. Percentage sites of 148 dilated abnormal communicator veins; 27% postero-medial, 34% exposed in limited medial incision.

Histological Findings

The main findings relative to the two series of cases are summarized in Table I.

TABLE I. ANALYSIS OF HISTOLOGICAL STUDY

	First series	*Second series*
Cases	30	35
Communicator veins	148	266
Histology done	131	175
Thrombosis and recanalization	8	21
Normal valves	8	34
Eccentric thickening: all degrees	37	64
Mild	—	23
Moderate to severe	—	41

(a) *In the first series* of cases thrombosis and recanalization was seen in 8 veins in 131 examined. Normal valves were present in dilated abnormal communicator veins in 8 veins. Eccentric thickening of any appreciable degree was found in 37 veins.

Additional findings. Other facts discovered were as follows. In the same leg thrombosis could be present in communicator veins, and yet other communicator veins could be unaffected (17 veins).

With a history suggestive of thrombosis, such as swelling and tenderness after an illness or pregnancy, there was no evidence of thrombosis in any of the dilated communicator veins in 8 cases.

With ulceration or induration present, there was no evidence histologically of thrombosis in dilated communicator veins in 5 cases.

Unsuspected thrombosis in communicator veins, when no history or physical signs indicated the possibility of this, was observed in 5 cases.

Cases with thrombosed communicator veins had previous surgery such as operative treatment of the varicose veins (8 cases), injection treatment of varicose veins (7 cases), a suggestive history of thrombosis with pain and swelling (9 cases), and induration or ulcer present in the past was found in 11 cases.

(b) *In the second series* histology was done in 175 veins (35 cases with 266 veins).

Thrombosis with organization or recanalization (Figs. 8 and 9) was present in 21 dilated communicator veins (12%).

Moderate to severe eccentric intimal fibrous thickening (Murray) was diagnosed in 41 veins (25%). Slight eccentric intimal thickening was found in 23 veins (21%).

Normal valves were seen in 34 veins (20%). This greater frequency of observed valves corresponds to the more liberal use of serial sections but did not alter the findings with regard to the incidence of thrombosis in veins.

DISCUSSION

Dilated incompetent communicator veins in this study are not due to thrombosis and recanalization.

Dilatation is undoubtedly present, normal valves are often present, and there are different varieties of intimal thickening.

Even in thrombosis and organization and intimal thickening, an ingrowth of elastic and muscle cells into the thickened area is a prominent feature, presumably to retain contractability and distensibility.

The concept of fixed, permanently dilated, fibrosed 'post-recanalized' communicator veins with destroyed valves is not valid.

Cockett[1] states: 'The organization and recanalization of such a thrombosis will involve destruction of its valves and production of an incompetent perforator vein in this situation. This actual occurrence has been seen by us at postmortem dissection, and we believe that this is probably the usual mechanism whereby the valves of the perforator veins may be destroyed.'

In the case of dilated communicator veins, the evidence is strong that it is not thrombosis and recanalization that produces the abnormality. It is suggested that the physiological responses on straining and relaxing in the standing position (in the presence of veins with a greater or lesser quantity of blood circulating through them), veins which are abnormal in structure or altered in structure by pressure, hormones, thrombosis and spasm, and obstructions and dilatations are important causative factors.

Continued evidence of tendencies to dilate and contract is given by the elastic and muscle tissue ingrowth into the damaged thickened intima, even when thrombosis has been the cause. It is more important to discover the reasons for dilatation and contractions and their effects on veins, than to blame thrombosis for abnormality and dilatation of the communicator veins.

SUMMARY

The sites of dilated communicator veins in 30 consecutive operative cases of varicose veins have been studied using a 'seam' exposure.

Limited medial incisions only expose about ⅓ of the dilated communicator veins.

In 131 dilated communicator veins examined histologically in the first series only 8 were thrombosed and recanalized, and 37 had eccentric intimal thickening.

Normal valves were found in as many instances as thrombosis in the first series.

In another 266 dilated communicator veins, 175 examined histologically, 12% showed thrombosis with organization and 20% were seen to have normal valves.

Dilated abnormal communicator veins excised at operation in cases with varicose veins are not due to thrombosis and recanalization with destruction of valves.

I wish to thank Prof. D. J. du Plessis for his stimulation and advice in this study and Prof. James Murray of the SAIMR for the many hours of laborious effort he has put into the interpretation of the histology.

REFERENCES

1. Cockett, F. B. (1955): Brit. J. Surg., **43**, 260.
2. Cockett, F. B. and Elgar Jones, D. E. (1953): Lancet, **1**, 17.