appeared competent, and we identified three incompetent perforating veins. This is a year and a half after SEPS. We then did an ascending venogram. We were able to identify three incompetent perforators (Fig. 1). You can see the clips from the SEPS, and you can see that one of those perforators communicates with an incompetent segment of the greater saphenous vein below the knee. So that's not good. There were recanalization changes of the superficial femoral vein (Fig. 2), and an incompetent perforator in the thigh are seen. Then as you come up in the groin here we identify some portion of the profunda, and the remainder of the iliac veins appear to be normal. So we identified three incompetent perforators in the calf, at least one incompetent perforator in the thigh, and recanalization changes of the superficial femoral vein. We haven't done anything further at this point.

III. PRIMARY VENOUS DISEASE: DEEP AND SUPERFICIAL REFLUX

TREATMENT OF PRIMARY VENOUS INSUFFICIENCY

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Recognition that a chronic venous problem is due solely to primary venous insufficiency (PVI) defines that the entire problem is due to reflux in the veins, and that obstruction is absent. PVI cases can present with exactly the same clinical appearance as post-thrombotic cases, but the treatment implications are quite different because the venous system is entirely patent and the luminal surfaces of the veins are normal. Successful treatment is possible by surgical means in all segments of the lower extremity veins afflicted with PVI, including superficial (saphenous), perforator, and deep veins, and the results in all of these divisions of the venous tree are highly favorable.

PVI has distinctive histologic findings that have been published in the past but are not frequently appreciated. The findings in the endothelial layer consist of hyperplasia, which produces the white streaks often seen on the luminal surface of the opened vein. The real pathology is in the subendothelial and medial layers where the most striking change is an increase of the collagen which appears to become aggressive by wrapping around muscle bundles and actually fragmenting the syncitial continuity of the muscles in PVI. This is accompanied by fragmentation of elastic layers and areas of either hyper- or hypoplasia of the muscular layers. This process logically results in dilation of the venous wall, and dilation of the wall leads to valve incompetence.

The striking difference between primary and secondary disease is that the signs of prior acute thrombosis and inflammation or hemorrhage seen in post-thrombotic disease are absent in primary disease. These signs are hemosiderin deposition, neovascularization in the old thrombus and in the vein wall, and leukocyte infiltration of the wall.

The gross changes of PVI are strikingly different than those found in post-thrombotic disease (PTD). In PVI, the lumen is smooth and the wall is pliant and of relatively normal thickness. Valve sites are fewer in the saphenous vein of PVI than in the normal state, probably due to atrophy and ultimate disappearance. All stages of atrophy of valve cusps can be seen in these veins. In the deep veins, the valve cusps are normal in appearance, but are stretched and elongated. These findings are strikingly different than in post-thrombotic veins where the luminal surface is irregular, contains synechiae and random webs, and sometimes endoluminal masses are present. The valves are disfigured, scarred, and often entirely destroyed. The wall is thickened, non-pliant, and there is usually a peri-phlebitis with adhesions to surrounding tissues.

Given these differences, it is not surprising that there are excellent opportunities for surgical repair in PVI and little reason for limiting treatment to external support and change of life-style in the otherwise healthy person. With care in diagnosis, and adherence to the CEAP requirements for definition of the etiologic basis for the clinical problem between primary, secondary, and congenital causes, and the pathogenetic mechanisms of reflux and obstruction segment by segment, opportunities for correction of the abnormal physiology abound in primary disease.

Treatment of superficial primary disease in the saphenous system, and of the perforator veins, is widely practiced and is all that is needed in 30-50% of the cases of ulceration, and in the vast majority of non-ulcer cases. The deep system is implicated in 60%+ of primary ulcer cases, and requires surgical repair in a so-far unknown percentage of these to provide long-term relief of the Class 4, 5, and 6 problems. The long-term success of valve repair, coupled with control of saphenous and perforator incompetence in PVI cases of classes 4, 5, and 6 up to 4 years and beyond, is well-demonstrated in the literature to fall in the range of 65-80% in every published series of significant size.

Given the present ability to diagnose primary venous disease accurately and by non-invasive affordable tests, the appropriate management of all primary disease should be by surgical correction in the active patient. This includes saphenous, perforator, and deep vein correction. The major question is to define which patients do, and which patients do not, require correction in the deep veins to provide a long-lasting favorable result. The answer to this will require comparative prospective treatment groups.

(Scientific Articles continue on next page)