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CC/NUMBER 27
JULY 7, 1986

Buck R C. Intimal thickening after ligature of arteries: an electron-microscopic study. *Circ. Res.* 9:418-26, 1961.
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This paper showed that the cells that come to obliterate the lumen of doubly ligated arteries arise from smooth muscle of the tunica media. The tissue in the lumen contains many elastic fibers. Endothelium does not participate; in fact, it becomes necrotic. [The *SCI*® indicates that this paper has been cited in over 170 publications.]

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May 25, 1986

My interest in vascular disease arose from association with a teacher in pathology, J.C. Paterson, one of the mavericks in the long-defunct American Society for the Study of Arteriosclerosis. My wish to study the question of the origin of cells of subendothelial hyperplasia arose from the work of another Canadian friend, the anatomist, Rudolph Altschul, whose careful light-microscopic observations on these cells were begging for their electron-microscopic interpretation. The model used in my paper of subendothelial hyperplasia, the doubly ligated artery, came to mind because one of my fellow PhD students at the University of London, R.M.L. Merhrotra, had used it earlier in light-microscopic studies.

Although for several years I had been in Murray Barr's department, where important work was in progress on sex chromatin, I resisted letting myself be drawn away from my interest in vascular disease. This stubborn demonstration of independence was never resented by Murray.

My laboratory was formerly the morgue in an old building that was then the medical school. Animal handling, tissue processing, and sectioning were all carried out in this subterranean room, where I wrestled for a year with polymerization damage in methacrylate-embedded arteries. As polymerization proceeded, the fine structure of the specimens was literally subjected to microscopic explosions.

In the summer of 1960, when I visited the laboratory of E. Kellenberger in Geneva and learned how to use a new embedding material, Vestopal, the polymerization problems vanished. Humidity, and not the ghosts of unclaimed bodies embalmed in my old room, had been the cause of the trouble.

I think that this paper attracted attention because it provided a demonstration of the role of the medial smooth muscle cell in subendothelial hyperplasia. Its role is not only in hyperplasia but also in migration through fenestrations in the internal elastic lamina and in the synthesis of elastic lamellae in the new intima. The morphology of these cells was a blend of smooth muscle and fibroblastic features, and to distinguish them from medial smooth muscle cells, I suggested the name "myo-intimal" cell. There has since been much interest in the synthetic capabilities of the vascular smooth muscle cell, principally in culture (reviewed by Chamley-Campbell and colleagues in 1979[1]), by which biochemical techniques have confirmed the hypotheses suggested by these morphological observations.

1. **Chamley-Campbell J, Campbell G R & Ross R.** The smooth muscle cell in culture. *Physiol. Rev.* 59:1-61, 1979. (Cited 195 times.)

