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New Trends in Lymphology

Introduction

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Physiologists throughout the world are increasingly aware of the fact that the final goal of the cardiovascular system, i.e. supplying tissues with oxygen and nutritients through transcapillary exchange processes, can not be understood without considering lymph vascular system. It is evident that a) blood capillaries, b) connective tissue with its ground substance, cells, fibres and prelymphatics, and c) lymph capillaries form a structural and functional unit. How this unit, according to present knowledge, looks and functions, will be described by CASLEY-SMITH in Section II.

Lymphatics are vital in removing from the tissues plasma proteins which continually escape blood capillaries; even in regions with fenestrated blood capillaries, where according to CASLEY-SMITH, most of the escaped proteins are retransported via the blood stream, radical lymphatic blockage will induce protein molecules to stagnate in the tissues. From the view-point of the pathologist and the clinician, this is the only fact which really matters. For physicians confronted with patients and diseases, calculations with a lot of integrals are hocus-pocus; moreover, even for minds with some theoretical training it often becomes difficult to distinguish between calculations based on actually measured data or on assumptions. The well-known formula based on STARLING's hypothesis

$$F = k[(P_c - P_i) + (\pi_i - \pi_c)]^1$$

continually misleads physiologists to feed assumed, nonmeasured data into computers. It would be fine if *pericapillary* interstitital fluid were representative for the *entire* interstitial fluid – we can obtain neither the one nor the other – and if it had the same protein concentration and composition as lymph which we can obtain from collectors – but let us 'assume' that these concentrations are identical!

If lymphatics fail to remove plasma proteins ('Low (nil)-lymph-flow-failure'), monocytes will immediately invade the area in order to save tissues from suffocation in lymphedema through uptake and proteolytic breakdown of extravascular plasma proteins. (I have coined the term *'Extralymphatic mastering of plasma proteins'*.) It is both of theoretical and practical importance that *benzopyrone* compounds are increasing extralymphatic mastering of plasma proteins. On the other hand, *lymphedema should be defined as a combined failure of canalicular lymphatic drainage plus cellular extralymphatic mastering of plasma proteins in combination with a normal lymphatic load.*

The overwhelming majority of physicians associate the words lymphatics or lymphology with malignancies and with X-ray and isotope lymphography.

Now, ALTORFER and CLODIUS produce evidence in Section 5 for the fact that absolute and definitive blockage of lymphatic drainage of only one extremity suffices to cause *death* (*'Malignant lymphedema'*). In Section 4 I shall try to demonstrate that even an incomplete and more or less reversible lymphostasis will initiate various *'Diseases of lymphostasis'*. Only by studying these experimental syndromes, can lymphatic participation in various primary diseases of non-lymphatic origin (*'safetyvalve insufficiency of lymph drainage'*) be understood. In contrast to protein drainage, transport of water and crystalloids via lymphatics is, as long as STARLING's equilibrium prevails, negligible. Any disturbance of this equilibrium, on the other hand, will immediately and drastically increase lymph flow. MISLIN, in Section 3, describes how this important compensatory mechanism works by increasing performance according to demand.

1952 we have pointed out that if, in an organ experimental lymphatic blockage leads to lymphedema, this automatically implies the necessity to search, in any edema of unknown origin arizing in this organ, which form of lymph-drainage-insufficiency (mechanical, dynamic or safety-valve) was responsible as a causative factor. Twenty years had to elapse until the idea seems to have become a trend in modern lymphology.

[1] k, Filtration coefficient; P_c, blood capillary pressure; P_i, pericapillary interstitial pressure; π_i, pericapillary colloid osmotic pressure; π_c, plasma colloid osmotic pressure.