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Introduction

The clarification of the problem of sudden cardiac death associated with physical exertion represents the most important single contribution made by clinical sportsmedicine to internal medicine so far. It is now possible to differentiate between the natural history of heart disease in patients who during their illness seek medical advice, as contrasted to that of persons in whom the terminal collapse represents the first manifestation of the cardiac affliction. The evidence presented in this volume relates to the latter category and its clinical implications. It also draws attention to the fact that impairment of exercise capacity is not a reliable prognostic criterion in cardiology as shown by the description of cases in which death occurred following outstanding athletic performances. Furthermore, the data under review demonstrate that the discovery post mortem even of gross pathological changes in the heart does not necessarily suffice to explain the fatal seizures. Supplementary factors are invariably involved, such as intercurrent infections (e.g. those discussed in VI and X), trauma to the chest, e.g. those discussed in V, VII, and VIII, or thermic stress, e.g. those discussed in XII and XIII [4]. That the timing of cardiac seizures and their particular clinical characteristics do not occur fortuitously is shown by observations of simultaneously developing acute circulatory failure in identical twins and in several members of the same family, e.g. those discussed in XVI and XVII [3, 7].

The contents of the volume have been arranged as follows: The opening section entitled ‘Asymptomatic Cardiac Diseases Causing Sudden Death in Association with Physical Activity’ summarizes 4 pathological syndromes that have been discovered during autopsy in cases of sudden unexpected death associated with physical exertion, namely coronary arterio- and atherosclerosis, mostly associated with myocardial changes; congenital
anomalies of the coronary arteries; myocarditis; and cardiac tumors. In Section II we report on 64 autopsies conducted after fatal seizures of the kind under study. Invariably, morphological manifestations of diseases of the circulatory system were found. Section III is devoted to a discussion of trigger mechanisms presumably responsible for the onset of the terminal syncope. By far the most frequent post-mortem findings in cases of sudden cardiac death associated with exercise is coronary athero- and arteriosclerosis. In Sections IV, V, and VIII the problem is elaborated in connection with observations of sudden cardiac deaths of pilots in flight, of a case of sudden death of a boy of 10 years, and of a case of sudden death of a middle-aged wrestler. Sections V, VI, and VII have been included because they throw light upon the significance of blunt trauma to the chest. Section IX deals with the problem of congenital anomalies of the coronary arteries, another potential cause of death of seemingly healthy persons. Section X contains the description of autopsy findings of a young man who after a 12-mile cross-country race died from myocarditis. Although myocarditis is a frequent clinical occurrence, sudden death associated with physical exertion due to inflammatory involvement of the myocardium is less frequent than would be expected considering the overall incidence of inflammatory involvement of the heart, primary or secondary. The probable explanation is that most subjects thus afflicted feel ill and are disinclined to participate in athletic activities. Section XI by T. N. James, P. Froggatt, and T. K. Marshall focuses attention to the fact that cardiac lesions, even if they are very small in extent, may be fatal if they cause damage to the conduction system, a finding in agreement with observations by Wolf and Bing [6] and by Schmidt et al. [5]. The accounts by Pugh of ‘Deaths from Exposure on Four Inns Walking Competition,’ and by Schrier et al. on ‘Nephropathy Associated with Heat Stress and Exercise’ (Sections XII and XIII) emphasize the role played by environmental influences as well as by the involvement of extra-cardiac systems. The scope of adjustibility to training of thermo-regulation in normal subjects has been demonstrated by S. Robinson [4]. Pugh et al. [2] presented evidence on the remarkable ability of Channel swimmers to maintain core temperatures during prolonged immersion in cold water. Sections XIV, XV, and XVI deal with 3 rare post-mortem findings in cases of the kind discussed in this monograph: one of subaortic stenosis, one of rupture of the ascending aorta, and one of aortic hypoplasia. That the latter condition may be related to the occurrence of sudden death of athletes was

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pointed out by us 30 years ago (XVI). The validity of our assertion was
Congenital anomalies of the heart do not invariably cause trouble.
J. H. Currens and Paul D. White [1] reported on the findings at the autopsy
of the famous marathon runner Clarence DeMar who died from a malignant
abdominal tumor at the age of 71. The caliber of DeMar’s coronary arteries
was exceptionally large. The authors suggest that this great athlete’s endowment
for long-distance running may have been due in part to the good blood
supply of his myocardium thus engendered.
Sections XVII and XVIII emphasize the role that genetic factors may
play in the timing as well as in the establishing of the pathological bases of
cardiac seizures [3,7].

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XI, XIII, XVII and XVIII.

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