THE VALIDITY OF MORTALITY FIGURES WITH REGARD TO CARDIO-VASCULAR DISEASE

Based on 4,430 Death Certificates

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From a recent report of the British Medical Association on the training of a doctor, it would appear that in many medical schools today the clinician seldom, if ever, attends a *post-mortem* examination or sees a biopsy specimen of the patient who has been under his care in the wards.

The practice of remedial medicine is primarily and chiefly concerned with the clinical and pathological state of the individual patient. Prophylaxis, based on investigation of selected groups of the population, e.g. recruits, sportsmen, factory employees, life insurance candidates, school children and so on, should aim at detection at the earliest possible stage of the least deviation from normal health. The prevention and control of communicable disease, which is mainly within the domain of the public health authorities, is based on

comprehensive data secured by such compulsory measures as notification, supervision, hospitalization and quarantine. The accumulation of clinical facts thus obtained and the study of the effects of various environmental factors, occupational, economic, nutritional and so on, furnish the material necessary for research in social medicine.

Accuracy and completeness of figures relating to morbidity, mortality, and environmental factors are essential to advance in our knowledge of social medicine. Unfortunately, however, the morbidity and mortality data, as at present available, are neither accurate nor complete. In the following investigation the author has attempted to analyse the details in relation to cardio-vascular disease available from a study of 4,430 death certificates in an English borough for the years 1935, 1946 and

TABLE 1

YEAR	1935	1946	1947	Total
Total deaths from all causes	1,368	1,526	1,536	4,430
Total deaths from cardiovascular disease	529 (38%)	669 (43.8%)	694 (45.1%)	1,892
Myocarditis as sole or contributory cause of death	39	59	49	147
Syncope	31, including 4 cardiac	6, including 4 cardiac	10	47
Valvular disease of the heart as sole cause of death, or combined	10	11	15	36
Morbus cordis and heart disease	4	1	0	5
Congenital heart disease	5	4	5	14
Myocardial degeneration with arteriosclerosis or with cardiac dilatation, or with cardiac failure	102	131	148	381
Myocardial degeneration as sole cause of death	7	50	47	104
Arteriosclerosis as sole cause of death	6	29	1	36
Angina pectoris	12	8	8	28

Deaths in an English municipal borough.

1947. The total deaths in these years were 1,368, 1,526 and 1,536, the figures referring to death from cardio-vascular disease being 529, 669 and 694 respectively. The certified diagnoses are shown in Table 1.

Myocarditis

Myocarditis may be defined as inflammation of the muscular walls of the heart. Inflammation is the defensive reaction of single organs or tissues and of the total organism to physical, chemical or bacteriological irritants. It must be remembered that the local tissue changes in bacterial inflammation are accompanied by constitutional disturbance of the whole organism and by mobilization of its forces of resistance. In order to comprehend inflammation at the bedside or in the *post-mortem* room one has to call up a distinct mental picture of the inflammatory process, its causative agent, its state (acute or chronic) and the type of lesion (serous, fibrinous, purulent, haemorrhagic or necrotic).

Conditions such as hypertension, coronary arteriosclerosis, chronic pulmonary disease, endocrine and metabolic disorders, and chronic renal disease give rise to more or less severe affections of the myocardium by reason of functional and/or metabolic derangement, or degenerative processes. Degeneration means biologically a reversion to lower type; pathologically it connotes morbid disintegration of tissue or change in its structure. The expression myocarditis should not be used in reference to primarily degenerative alterations of the myocardium, nor is it appropriate to indicate the end-results of acute or chronic inflammation (i.e. scars consisting mainly of fibrous tissue). The term chronic myocarditis is in common use, but it indicates little or nothing; myocardial fibrosis, on the other hand, indicates what is actually found in the heart, viz. a condition of scarring. Eminent authorities have for some considerable time recommended, for the sake of more accurate diagnosis, the abandonment of the term myocarditis except in conditions in which the histological criteria of inflammation are fulfilled. Whereas some authorities reserve the term myocarditis for the myocardial involvement incidental to rheumatic fever and diphtheria, other writers, relying on extensive *post-mortem* studies, categorically state that true myocarditis occurs in various pyrexial and infective conditions.

In one series of 5,626 *post-mortem* dissections 240 cases of myocarditis were encountered, of which 186 were non-rheumatic. Among 1,402 autopsies of myocarditis only 130 cases of rheumatic carditis were found, so that the heart condition in more than 90 per cent. of the series was non-rheumatic. The following diseases were im-

plicated:—scrub typhus, epidemic typhus, Rocky Mountain spotted fever, rickettsial diseases, diphtheria, bacterial endocarditis, meningo-coccaemia, scarlet fever, Weil's disease, syphilis, malaria, trichinosis, acute encephalitis, poliomyelitis, infectious mononucleosis, mumps, virus pneumonia, tuberculosis, Boeck's sarcoid, coccidiomycosis, streptococcal, staphylococcal and pneumococcal septicaemia, acute tonsillitis, acute nasopharyngitis, cellulitis, etc. The same authors collected 36 instances of fatal myocarditis attributable to acute nasopharyngitis and tonsillitis. In only three cases was the myocardial involvement suspected clinically. Myocarditis from epidemic parotitis was definitely established on dissection in 1932. The first known case of myocarditis complicating mumps, in which the diagnosis was established during life, was described in 1944, and two years later myocarditis complicating mumps was found in 15.4 per cent. of a series of 104 patients.

Suppurative myocarditis occurs as a usual complication of bacterial endocarditis or acute pericarditis through contiguity, and as a complication of pyaemia through the haematogenous spread of minute bacterial or thrombotic emboli. Fiedler's isolated myocarditis, a foetal myocarditis with consecutive congenital cardiac hypertrophy, and the myocarditis with heart enlargement leading to rapidly progressive congestive failure are rare conditions of obscure aetiology.

Recent literature is studded with reports on cases of severe myocardial involvement, the aetiology of which is not yet clearly understood e.g. that associated with lupus erythematosus disseminatus, Boeck's sarcoid, amyloidosis, acute haemorrhagic nephritis, toxæmia of pregnancy, emetine, sulphonamides, allergy, burns, xanthomatosis, scleroderma, exfoliative dermatitis. Pathological alterations of the myocardium caused in the experimental animal by digitalis, acetylcholine, iodine, or vagal stimulation are essentially of necrotic and degenerative character, and should not be classified as myocarditis in the strict sense of the word.

Heed should be paid to the age-distribution among those alleged to have succumbed to myocarditis in this series. In 1935 death was attributed to myocarditis 39 times among 529 deaths from cardio-vascular disease. Only three were under the age of 50, the youngest being 29; in the latter myocarditis was certified as the only cause of death. Only two were in the sixth decade, one of them having been certified as having also syphilis and tabes dorsalis. The ages of the remainder dying from myocarditis ranged from 65 to 87.

In 1946 there were 59 diagnoses of myocarditis among 669 deaths from cardio-vascular disease.

Only two of these 59 occurred in persons under 50. In 1947 among 694 deaths from cardiovascular disease there were 49 diagnoses of myocarditis. Two of these were really accurate, one certified as a case of Adams-Stokes syndrome due to syphilitic myocarditis, the other as a case of myocarditis and myocardial failure due to rheumatic carditis (mitral stenosis and aortic incompetence). It may be of interest to analyse two of the death certificates from 1946, and two from 1947.

Case 1 (1946), female, aged 28

Immediate cause of death :—

- i. (a) Syncope ; (b) myocarditis ; (c) status asthmaticus.
- ii. Other morbid conditions contributing to death—pregnancy, four months.

Case 2 (1946), female, aged 15 months

Immediate cause of death :—

- i. (a) Myocarditis ; (b) whooping cough.

Case 3 (1947), male, aged 50.

Immediate cause of death :—

- i. (a) Syncope ; (b) myocarditis ; (c) alcoholism.
- ii. Other morbid conditions contributing to death—acute gastritis and haematemesis.

Case 4 (1947), female, aged 50.

Immediate cause of death :—

- i. (a) Cardiac failure ; (b) myocarditis ; (c) carcinoma uteri.

Case 1. Syncope is merely a clinical syndrome or mode of death and will be dealt with later. This is said to be due to myocarditis which in turn is ascribed to status asthmaticus in a pregnant woman aged 28. In the first place status asthmaticus in itself is a disorder of the function of respiration which may lead to haemo-dynamic alterations and thus to cardiac involvement through increase of pressure in the pulmonary circuit, ventricular strain, dilatation and hypertrophy of the right heart ; but it is difficult to imagine how a status asthmaticus could cause true myocarditis, in other words an inflammatory process. In the second place, we are completely in the dark about the aetiology of the said status asthmaticus. Did it occur in an allergic woman in the course of bronchial asthma ? Was it a pulmonary oedema in the course of an acute left ventricular failure resulting from arterial hypertension in a pregnant woman ? Was it a pulmonary oedema due to toxæmia of pregnancy, or to acute left auricular failure in association with mitral stenosis ? Was the alleged myocarditis acute or chronic, pyrexial

or apyrexial ? None of these questions can be answered by the reader and therefore the certificate is not acceptable to the Registrar General.

Case 2. According to the present state of our knowledge whooping cough or its causative agent, haemophilus pertussis, never gives rise to inflammation of the heart (myocarditis). However, the following complications have been described in association with the disease, viz. bronchopneumonia, pulmonary atelectasis, vesicular and interstitial emphysema, subcutaneous emphysema, pneumothorax, convulsions and encephalitis. In view of these facts this certificate would not appear to be valid.

Case 3. Alcohol, like morphia, methylsulphonal, etc., acts specifically upon the central nervous system ; it does not act upon the myocardium as an inflammatory agent. Consequently there is no such thing as myocarditis due to alcoholism. Even the best known cardiac poison, digitalis, does not cause inflammation of the myocardium when exhibited in toxic doses, for only albuminous, fatty and necrotic lesions of the myocardium have been described. This certificate, therefore, is of no value.

Case 4. This sounds rather like an enigma. Was there any real myocardial involvement due to cancer ? If so, then it was a metastasis, not an inflammation, for cancer is a neoplastic not an inflammatory process. Was there myocardial involvement due to some aetiological factor other than cancer ? If so, then it was merely coincidental with the carcinoma uteri. Was there any myocardial involvement, inflammatory or neoplastic, at all ? One feels inclined to assume that there was no cardiac involvement and that the cancer was the only cause of death, but the medical attendant was apparently unable to discern and localize the deleterious effects of metastases and/or malignant cachexia.

Among 147 deaths from myocarditis covering three years, 130, i.e. over 90 per cent. ranged from 63 to 95 years of age. It is a common clinical aphorism that the cardio-vascular pathological alterations incidental to old age are essentially degenerative and not inflammatory in nature. They are caused by coronary sclerosis, arterial hypertension, chronic pulmonary disease, metabolic and hormonal disorders, anaemias and so on. Thus, quite apart from all the evidence previously adduced, the misuse of the term myocarditis becomes the more obvious when considered in relation to the age-distribution among the alleged sufferers from that condition. There are, doubtless, some less frequent inflammatory and infective cardiac complications that terminate the lives of the aged, but these should be carefully checked by modern scientific tests. The routine

employment of these has thrown much light on cases of myocardial involvement which was formerly obscure. Systematic electrocardiographic studies in a series of 72 cases of scarlet fever have shown toxic myocardial alterations in 14 (i.e. 20 per cent.).

In conclusion it would seem clear that the term myocarditis as applied in the series under study is, with very few exceptions, a misnomer, and does not furnish the positive information required by the Registrar General.

Syncope

Syncope was certified as the cause of death 47 times during 1935, 1946 and 1947. The term denotes a functional disorder of circulation occurring in persons with unstable vaso-motor centres. The commonest form of syncope has been described by the late Sir Thomas Lewis as a vaso-vagal attack. The expression vaso-vagal implies that the afferent and efferent limbs of the reflex arc are in the vagus. Syncope due to vagal reflexes which originate in the carotid sinus may occur in persons with perfectly normal hearts. The provocative stimuli are optic, acoustic, olfactory or painful sensations or emotions or long standing. There are three types of vaso-vagal attack, all having in common disturbances of sensation which vary from mere dimness of vision to complete unconsciousness together with pallor and sweating:—(a) slowing of the heart and fall of blood pressure; (b) fall of blood pressure only; (c) cerebral symptoms only.

Coma, syncope and anginal pain are symptoms which, like headache, commonly occur in a variety of morbid states, some of them trivial, some fatal. Epilepsy, hysteria, diabetic or uraemic acidosis, hypoglycaemia, indulgence in alcohol or other narcotics, bacterial toxæmia, brain lesions of traumatic, inflammatory, vascular or neoplastic nature may all be associated at times with a transient or terminal syncope or coma. To describe syncope as cause of death in a case of myocardial infarction is as meaningless as to designate intractable headache as cause of death in a case of cerebral tumour. By syncope we mean loss of consciousness caused by a deficient flow of blood from the heart to the brain. Phrases such as syncope, coma, asphyxia, cardiac syncope, respiratory failure, used to designate the cause of death without any further particulars, merely indicate the cessation of vital functions of the heart or brain or respiration. It is only by stating the pathology, aetiology and contributory factors immediately underlying such a condition as 'congestive heart failure' that a clear meaning of the cause of death will be conveyed; e.g. :—

Immediate cause of death :—

- i. (a) Congestive heart failure due to (b) high degree mitral stenosis due to (c) old rheumatic fever.
 - ii. Other morbid conditions—pregnancy, six months, or arterial hypertension.
- Immediate cause of death :—
- i. (a) Pulmonary oedema, viz. acute left ventricular failure due to (b) recurrent myocardial infarction due to (c) coronary sclerosis and arterial hypertension.
 - ii. Other morbid conditions—hypochromic microcytic anaemia, or obesity or prostatic adenoma with urinary retention.

Cardiac syncope occurs when there is sudden diminution of cardiac output, either because the ventricles beat very slowly, e.g. in complete auriculo-ventricular block (Adams-Stokes syndrome); or very rapidly as in paroxysmal tachycardia, auricular flutter and auricular fibrillation; or because of the sudden onset of left ventricular failure, most commonly occurring as the result of myocardial infarction. With reference to the treatment of cardiac infarction it is recommended that morphia should be given at once, as the sooner the pain is controlled, the less likely is the patient to die from syncope. With regard to prognosis in angina pectoris, it is stated that there is always risk of infarction and that sudden death from syncope, or ventricular fibrillation, may occur any severe attack.

It is to be noted that syncope, asphyxia and coma are the common terminal clinical phenomena which usher in or represent the act of dying. In other words, the ceasing function of heart, respiration or brain. Thus it becomes clear that a syndrome such as syncope or asphyxia in itself cannot explain the cause of death. However, where the practitioner feels inclined to indicate the vital organ such as heart, brain, lungs, liver, kidneys or adrenals primarily ceasing function, he may do so, but he should make reference to the aetiological, clinical and chronological landmarks of the patient's history so that the reader may comprehend the train of pathological events, i.e. the whole morbid process from its origin to its end.

Myocardial Degeneration and Arteriosclerosis

Myocardial degeneration with arteriosclerosis, or with cardiac failure or dilatation was diagnosed 102 times in 1935, 131 times in 1946, and 148 times in 1947. Myocardial degeneration as the sole cause of death was certified seven times in 1935, 50 times in 1946, and 47 times in 1947.

The definition of degeneration in general was given above in discussing myocarditis. The chief causative factor of myocardial degeneration and

fibrosis is coronary atherosclerosis with consecutive impairment of circulation (chronic myocardial ischaemia). Sudden obstruction of coronary vessels especially those of major calibre through atherosclerosis, thrombosis, thrombo-angiitis obliterans or embolism results in local myocardial ischaemia, infarction and necrosis.

Yet, natural death from old age alone occurs but rarely, the life of the aged being terminated by inter-current maladies, every aged person being the bearer of healed, latent or chronic disease. Unfortunately, in prevailing circumstances no attempt has been made to unravel the diagnostic problems of the aged either at the bedside or in the post-mortem room prior to certification of the cause of death. 'The main purpose of necropsy is to demonstrate in the dead body the changes resulting from disease which cannot be demonstrated in the living patient.' I venture to suggest that the rule must be hammered into the heads of all hospital workers, whether on the staff of medical schools or on the staff of county hospitals, that no clinical diagnosis of a deceased patient can be considered acceptable unless corroborated by necropsy. 'The chief flaw in a badly organized service, such as that which has evolved in this country during the last century, is lack of time, and both the general practitioner and the consultant, in order to earn a living wage, are frequently obliged to undertake far more work than they can deal with efficiently in the hours at their disposal. . . . This seems to be one of the very reasons why such diagnostic labels as myocardial degeneration and arteriosclerosis are still in vogue. Indeed, the term myocardial degeneration is a generality applied to a whole class of morbid syndromes and anatomical lesions as diverse as rheumatic carditis, myocardial infarction and pernicious anaemia, with fatty degeneration of the myocardium. The term arteriosclerosis denotes a pathological process commencing in early adolescence, clinically latent throughout adult life, giving rise to morbid manifestations chiefly in the aged, or in the younger under special circumstances. The clinical significance of arteriosclerosis depends mainly on the pathogenetic and morphological type of the vascular lesion such as atheroma, Moenckeberg's sclerosis (degeneration and calcification of the middle coat of the artery), endarteritis obliterans, thrombo-angiitis obliterans, arteriosclerosis commonly associated with hypertension and old age, lupus erythematosus disseminatus, periarteritis nodosa, and suppurative, syphilitic, rheumatic or tuberculous arterial inflammation; also on the localization of the lesion, cerebral, myocardial, renal, pulmonary or retinal; and on the calibre of the arterial segment involved, large or medium-sized, arteriolar or capillary.

Arteriosclerosis as such does not kill. The simple senile dilatation and tortuosity of the large vessels, with loss of elasticity, have no serious effect on haemodynamics. 'It is conceivable that all our organs may be diseased, and yet work in harmony. A man of 60 who had diabetes, nephritis, arterial hypertension, arteriosclerosis and pulmonary tuberculosis worked as a wool sorter for eight years, and finally died from pulmonary haemorrhage.' In that case, the certificate of death should read:—

Immediate cause of death:—

- i. (a) Pulmonary haemorrhage due to (b) chronic pulmonary tuberculosis.
- ii. Other morbid conditions contributing—diabetes mellitus, arterial hypertension, nephritis.

In conclusion, the term arteriosclerosis in itself is a generality, and as such is not acceptable as a certified diagnosis unless the pathological type of the vascular lesion, its localization and aetiology are clearly indicated.

Angina Pectoris

'The better the diagnostician, the longer the history' (Walter C. Alvarez).

Angina pectoris was certified as cause of death 12 times in 1935, eight times in 1946, and eight times in 1947. In these 28 cases angina pectoris occurred as the sole cause of death in seven.

The term angina pectoris, originally introduced into clinical usage in 1768, denotes a special type of chest pain precipitated by effort. It is ascribed to myocardial ischaemia resulting from deficient blood supply. The intensity, duration and pathis of reference of anginal pain, its association with general and cardio-vascular symptoms, and its signs vary according to the degree and extent of the underlying myocardial ischaemic lesion, with the threshold of the nervous end organs, and with the psychic sensitiveness of the patient. Thus many cases of infarction have been known to occur without noticeable pain and, conversely, cases occur with protracted, intense pain in which there has been no demonstrable infarction.

Myocardial infarction commonly results from complete arterial occlusion, with underlying coronary atherosclerosis, but infarction may be absent when the occlusion develops very slowly or when there is an ample blood supply to the affected area via collaterals. On the other hand, infarction may result from brief attacks of angina pectoris, viz. transitory coronary insufficiency—its physiological equivalent—in the absence of occlusion, provided the intensity and duration of the transient ischaemia were adequate to produce a myocardial lesion. The anatomical lesions of myocardial infarction without occlusion are

described as focal disseminated myomalacia localized in the sub-endocardium and papillary muscles. Acute myocardial infarction without coronary arterial occlusion is intermediate between the ordinary anginal attack and the complete acute obstruction of a coronary vessel. At necropsy of such a case in a man of 37, innumerable microscopical areas of necrosis and fibrosis were found. Brief attacks of angina pectoris at rest designated by some authors as 'premonitory pain' are often associated with evidence of myocardial necrosis. 'A thorough history is of paramount importance in the recognition of progressive coronary insufficiency which may early result in the formation of small areas of necrosis. One should not be satisfied with the plain diagnosis of angina of effort.' Though coronary atherosclerosis is the most constant and predominant pathology underlying the anginal syndrome, there are various other causative factors worthy of note, such as (a) anaemia by virtue of decreased oxygen content of blood, (b) syphilitic aortitis or bacterial aortic valvulitis implicating coronary ostia, (c) free aortic regurgitation, syphilitic or rheumatic, with a low diastolic pressure, (d) rapid paroxysmal heart rhythms without compensatory increase in coronary flow, (e) rarely, mitral stenosis, it is said, through diminished cardiac output or pressure from an enlarged left auricle.

The intricate differential diagnostic problem of acute protracted coronary insufficiency, i.e. the physiological mechanism of myocardial infarction, was already appreciated by James B. Herrick in his first case of acute coronary occlusion in 1910. On that occasion he and his surgical colleague considered as alternatives a pneumothorax, acute pancreatitis or strangulated diaphragmatic hernia. Indeed, gastrointestinal symptoms are not uncommonly associated with acute or chronic myocardial disease. It is noticeable in patient's histories how frequently complaints of indigestion, flatulence and eructation occur in both the ambulatory middle-aged man with transitory coronary insufficiency, and in the elderly, acutely ill patient with recent infarction. The close inter-relationship of cardiac and gastric symptomatology deserves particular attention to avoid diagnostic errors. A man of 58 with chronic coronary insufficiency, followed-up over a period of two years, manifested anginal pain typically related to effort, and inextricably inter-mingled with symptoms of gastric ulcer. He succumbed to a perforated gastric ulcer proved at necropsy, together with numerous patches of myocardial fibrosis visible on cross-section of the apex of the left ventricle.

Apart from abdominal complications, a dissecting aneurysm of the aorta, pericarditis or

pulmonary embolism may be easily mistaken for acute coronary occlusion. In addition the following conditions, if presenting precordial pain, must be differentiated from coronary insufficiency:—spondylitis, intercostal neuralgia, intercostal myositis, herpes zoster, pleurisy, spontaneous pneumothorax, tabetic crises, cardiospasm, mediastinal or pulmonary disease, neurocirculatory neurasthenia, hyperventilation with consecutive alkalosis and diaphragmatic flutter.

In view of the aforesaid it becomes obvious that there is a perplexing complexity of clinical, physiological and anatomical features pertaining to the anginal syndrome which, for that very reason, cannot in itself explain the cause of death without qualification of the underlying pathogenesis. 'Stenocardia, or the breast pang, described by Heberden, is not an independent affection, but a symptom associated with a number of morbid conditions of the heart and vessels.' Therefore, the term angina pectoris cannot be applied as certified diagnosis. Likewise we do not certify the cause of death from cerebral tumour as 'headache from increased intracranial pressure,' or the cause of death from a perforated hollow viscus (stomach, gall bladder, appendix) as 'abdominal colic.'

To conclude, the term angina pectoris denotes a clinical syndrome due to various pathological conditions of the myocardium, but also not infrequently associated with affections of supra- and infra-diaphragmatic structures adjacent to the heart. Thus, the certified diagnosis 'angina pectoris' does not interpret the real cause of death and, therefore, should be abandoned.

Valvular Disease of the Heart, Congenital Heart Disease, Morbus Cordis Heart Disease

Valvular disease of the heart was certified as immediate cause of death 10 times in 1935, 11 times in 1946 and 15 times in 1947. Congenital heart disease as immediate cause of death was certified five times in 1935, four times in 1946 and five times in 1947. Morbus cordis and heart disease as immediate cause of death was certified four times in 1935, once in 1946 and did not appear in 1947.

The terms 'heart disease,' 'valvular disease of the heart,' 'congenital heart disease' are commonly employed as titles of medical textbooks, and chapters thereof, but they do not classify a clearly established malady. It is suggested, therefore, that such terms as heart disease, valvular disease of the heart and congenital heart disease should be altogether banned from medical certificates of the cause of death.