CORONARY HEART DISEASE IN BRITISH SOLDIERS

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SUMMARY

The aim of this thesis is to present evidence that young soldiers in the British army have a higher mortality from coronary heart disease (CHD) than the civil population from which they derive, and to investigate the causes of this higher mortality, with particular reference to the role of strenuous physical exercise.

Chapter One, the introduction, outlines the historical perspective of exercise in populations and individuals. It goes on to describe the attitude of the British army to exercise, both voluntary and compulsory, and to place this in the context of expert and lay opinions on the dangers of exercise. Next follows a historical review of CHD, its risk factors, and its pathology. The body's response to exercise in health and disease is discussed, and finally, the present state of our knowledge on the benefits and risks of exercise.

Using data on CHD mortality in the British army culled from the 5 year period 1973-7, and published data from the Office of Population Census and Surveys, the Scottish Home and Health Department, and the Northern Ireland Office, for the same period, Chapter Two compares

age specific mortality ratios and concludes that British soldiers under the age of 40 years have a significantly higher, and British officers of all ages a significantly lower mortality from CHD than United Kingdom civilians irrespective of social class. It discovers an inverse relation between CHD mortality and army rank similar to that seen in the civilian social classes, and a trend of diminishing difference in CHD mortality with age between officers and men also similar to that seen in the civilian social classes. The gist of the data was published in the British Medical Journal in 1981.[1]

By detailed analysis of all deaths in the British army between 1968 and 1977 the author was able to discover 56 sudden and unexpected deaths which occurred within 6 hours of strenuous physical exercise. In Chapter Three he uses these data to define the incidence of sudden unexpected death during exercise, and the spectrum of diseases responsible. He concludes that the incidence, at 3.5 per 100 000, is very low, and that such deaths are caused by a small number of conditions viz: coronary heart disease (63%), subarachnoid hemorrhage (18%), congenital cardiac anomalies (9%), infection (5%), and unknown causes (5%). He concludes, by dint of the severity of CHD found at post mortem examination, and by the fact that those who died during exercise were no

younger than those who died at rest, that strenuous physical exercise does not precipitate death from CHD, except in cases of congenital coronary artery abnormalities, such cases being at particularly high risk of death during sport. The incidence of unexplained death during strenuous exercise is no greater than the incidence of unexplained death overall. In view of the small numbers of deaths involved, and of the severity of the antecedent disease, it is concluded that strenuous physical exercise is not responsible, at least in its immediate effects, for the difference in CHD mortality between soldiers and civilians. These data were published in the Lancet in 1980.[2]

Such is the quality of medical records in the army that the author was able to examine in detail all instances of non-fatal coronary events in the period 1973-7 and assess the accuracy of the diagnostic label, and, in a high proportion, whether or not strenuous exercise occurred in relation to the events. In Chapter Four the juxtaposition of these data with those on fatal coronary events described in Chapter Two allows an assessment of the relative mortality risk of a coronary event occuring during strenuous exercise with one occuring at rest. Whether diagnostic criteria are strict or lax, the mortality from coronary events is not increased when these events occur in relation to strenuous exercise.

It thus seems clear that strenuous exercise is not the cause of the soldiers' higher mortality from CHD. Chapter Five now examines the conventional coronary risk profile of soldiers under 40 years of age with CHD. Obesity, hypercholesterolemia, and cigarette smoking appear to be the main risk factors in this group. The obesity may parallel cigarette smoking through the medium of social drinking. Hypercholesterolemia seems to be a function of all young coronary patients, military and civil alike, and this may stem from an undue proportion of cases of familial hypercholesterolemia presenting in this age group. The factor setting young affected soldiers apart from other soldiers, civilians in general, and young affected civilians, appears to be their very much greater consumption of cigarettes. Thus the major determinant of the higher mortality from CHD seen in soldiers seems to be cigarette smoking. Reasons for this phenomenon are discussed.

The last Chapter summarises the findings of the thesis and indicates the means by which the situation may be remedied.

The author confirms that this work is original and, apart from substantial statistical help with Chapter Two, entirely his own.

CHAPTER ONE

EXERCISE AND THE ARMY

HISTORICAL PERSPECTIVE

1.1. Social. It was typical of Churchill to remark "Sometimes I feel the need for violent physical exercise, so I go and lie down and the feeling soon passes." He was expressing the resentment of the unfit against the social pressures to exercise as surely as the car sticker which proclaimed "I ran away from the London marathon." Not everybody wants to exercise. Unfortunately, most people feel they should, and therein lies the nub of why the subject arouses so much passion. Whether or not exercise is beneficial to bodily health is the main theme of this thesis, but its effect on mental health can scarcely be in doubt. Ancient Greek society was bonded together by exercise just as surely as was the Third Reich, and army commanders from as far back as Thermopilae to Alamein knew the value of exercise in promoting morale and regimental spirit. The SAS, the Parachute Regiment, the Marines, are all welded together by appalling requirements of physical fitness. Jesse Owen's redeeming success in the 1936 Olympics only

served to underline the depth of the hinterland of athleticism in the German nation which, only a decade before, had been the effete Weimar rump, with 10% unemployment and rampant inflation, and which was now about to annex "inferior" nations with such cohesive efficiency that Chamberlain's pacifism must surely have been tinged with awe. Sport may be the opiate of the masses (in more ways than one), but it is also a cement.

1.2. Individual. At the level of the individual the picture is similar. Cicero[3] said of exercise "It can preserve something of our strength even in old age." Addison, the essayist, perhaps paraphrasing him, held that without exercise "the body cannot subsist in its vigour, nor the soul act with cheerfulness."[4] Thomas Jefferson advocated walking as the best form of exercise,[5] Heberden advocated exercise in the treatment of angina,[6] and Paul White, the cardiologist, maintained that work alone never killed a man, and was often heard describing exercise as "the best tranqiliser there is."[7] Much closer to home, Thomas Bassler claimed cast iron immunity from coronary heart disease (CHD) for his marathon runners.[8-10] His debunking took no time at all,[11-14] and with it came the report of the death of a marathon runner sporting a T-shirt with the apocryphal legend "You haven't really run a good marathon until you drop

dead at the finish line - Phaedippides."[15] Whether Phaedippides actually did drop dead after his marathon we can never be sure[16] but he was certainly upholding a fine military tradition when he ran from Marathon to Athens on an important mission for his commander.

EXERCISE IN THE ARMY

1.3. Voluntary Exercise. The soldier's role has scarcely changed since the days of ancient Greece. Despite the intricacies of modern computerised battles the soldier is still required on the ground, holding territory or acquiring it. His strength, endurance, and morale are important factors in the equation of combat and it is in his own interests, and those of his leaders, that he maintain a level of fitness commensurate with these requirements. The British army goes to great lengths to ensure the fitness of its troops. Even the most minor units maintain football, rugby, hockey, tennis and squash teams, and great stress is laid upon inter-unit competition. Even the smallest garrisons are not without football fields, squash and tennis courts. Wednesday afternoon is officially designated as sports afternoon, and early morning running is a daily occurrence among many of the teeth arms. All units have sports stores where specialist kit is issued free

eg: skis, rackets, strips etc. Free courses in official time are provided for coaching and refereeing in all recognised sports, travel to and from matches is free, and rents are waived in many instances. Table 1.1. is a list of the sports the army sponsors in this way.

1.4. Compulsory Exercise. Despite the strong social pressure, all that has been said so far is of a voluntary nature. But the army does not rest there. A compulsory minimum level of physical fitness is laid down and all ranks in the army, up to the age of 50 years, must prove themselves above this level every six months. This minimum level, the Basic Fitness Test, consists of a group run of 1½ miles on the flat, within 15 minutes, followed immediately by a further 1½ miles of individual best effort in not more than 11 minutes (an extra half minute is added for every half decade over the age of 25 years). Defaulters may have several goes but in the end those who fail are medically downgraded and their promotion prospects reduced. Senior officers in their annual Fitness For Role inspections may call upon a unit to demonstrate its fitness by running the Basic Fitness Test as part of the inspection.

1.5. Anecdotal Caveats. Thus the army is perhaps the largest institution in Britain to encourage and demand frequent participation in strenuous exercise. While epidemiologic studies continue to confirm that exercise

is a good thing, at least as far as CHD is concerned,[17-21] these demands are reasonable and the occasional death from trauma acceptable, as is the constant trickle of orthopedic and other comparatively minor exercise induced ailments. However, exercise deaths for no apparent reason continue to occur in sporadic and dramatic fashion, devastate morale, and give rise to caveats from eminent sources which surround exercise with an aura of danger. Thus Barlow[22] says it is bad to run with a fever, Opie[23] cautions middle aged smokers, Fletcher[7] insists on prior medical examination, Mead[229] would like to see ectopic beats treated before exercise, and Noakes[25] warns against ignoring chest pain and undue breathlessness. The list is long, the names impressive, and the experience almost entirely anecdotal. Above all is the overriding feeling that exercise is intrinsically dangerous and we should all be wary. This feeling communicates readily to soldiers and gives rise to macabre humour and a great deal of false alarm hospital referrals. It is important to know therefore, if the army's insistence on strenuous exercise is putting young soldiers ar risk, or if, on the other hand, soldiers are living longer lives by virtue of a cardioprotective effect from strenuous exercise.

CORONARY HEART DISEASE

THE YOUNG EPIDEMIC

1.6. History. Perhaps the earliest account of death from acute myocardial infarction dates back more than 4 000 years,[26] but it was not until about 1859 that the first causal link with coronary artery occlusion was established.[27-8] Thereafter a number of papers confirmed the association,[29-32] but as late as 1915 the then professor of medicine at Cambridge discarded the idea of coronary artery disease producing angina pectoris.[157] It was not until 1918, when electrocardiographic changes began to be accepted as diagnostic of CHD,[33] that the disease became generally recognised. By 1950 it represented 20% of the total male deaths in the United Kingdom[34] and by 1973 30-40%.[35] Today it is the most common cause of death, and 150 000 deaths annually in England and Wales are attributed to it.[36]

1.7. Risk Factors. With the epidemic a mere two generations old there is no answer at present to its cause, but a number of epidemiologic studies have allowed the identification of particular sub-groups in the population at particularly high risk. The disease mainly affects middle aged men, initially of high social class, but

latterly the brunt has been borne by social classes IV and V. Three major risk factors in the general population have been identified so frequently that there is general agreement about their significance viz: hypertension, hypercholesterolemia, and cigarette smoking. A whole host of subsidiary risk factors have been identified, though in some there is less than general agreement. Diabetes mellitus, obesity, small stature, type A personality, family history, raised serum triglyceride, reduced level of serum high density liporotein, western diet and physical inactivity have all been incriminated, and the whole question of stress remains as protean as ever. This has led to the concept of the coronary risk profile whereby the score of risks in individuals or populations is assessed.

PATHOLOGY

1.8. Atheroma. The basic process underlying CHD is the development of atheroma which causes patchy stenosis of the coronary arteries. This process, in severe degree is found in association with almost all cases of myocardial infarction and, in about half the fatal cases, there is also occlusive fresh luminal thrombosis. The condition is both degenerative and proliferative,[37] these two processes being epitomised by intimal cell necrosis

and accumulation of lipid on the one hand, and by smooth muscle hyperplasia on the other.

1.9. The Fibrolipoid Plaque. The fibrolipoid plaque is regarded as the archetypal lesion of atherosclerosis. It consists in essence of a lipid base covered by connective tissue.[38] The lipid found in the plaque is essentially cholesterol and it appears to originate in the plasma and be transported across intimal cell membranes as lipoprotein by pinocytosis. Recent studies suggest that high density lipoprotein (HDL) may inhibit the net effect of accumulation of lipid within the intima.[39-41]

1.10. Thrombosis. The key event in the life history of a plaque is thrombosis, and events preceding this appear to be the splitting of the covering layer with expsoure of the underlying lipid.[42-3] Early research concentrated on the cholesterol nature of the plaque despite the obviously important part played by the clotting mechanism, and the early warnings by Duguid[44] that these plaques might well have developed from an initial intramural thrombus. Ross and Glomset[45], and Rutherford and Ross[46] demonstrated that aggregated platelets release a low molecular weight basic protein which has the capacity to trigger proliferation of arterial smooth muscle cells in culture, and Fischer-Dzogga[47] found a similar effect for low density lipoprotein (LDL). In even more recent years the

recognition of the importance of prostacycline in vessel wall smoothness, and thromboxane in platelet stickiness, has not only improved our knowledge of these basic mechanisms, but may also have given us our first major weapon against the disease.[48]

1.11. Wall Shear. Caro[49-50] proposes that wall shear is a major controlling factor in relation to the arterial branches, with most lipid accumulating proximal to the divides of the branch in that region of the wall where shear is lowest.

1.12. The Monoclonal Hypothesis. The monoclonal hypothesis put forward by Benditt[51] in 1973, on the basis of glucose-6-phospate dehydrogenase isoenzymes to explain the smooth muscle proliferation as neoplastic, stimulated Burch[52-3] to proliferate in print a series of cogently argued theories to explain the genetic component of CHD, and to inculcate the so-called risk factors as triggers to allow the escape of "forbidden clones" which, after a latent period, would result in clinical disease at an earlier age than might otherwise occur.

1.13. Coronary Artery Spasm. Maseri's work[54-7] on coronary artery spasm is doing much to change the

traditional static theories of angina as resulting from imbalance of supply and demand.[58] His findings do much to explain the anomaly that many deaths from CHD occur in severely diseased but unquestionably patent coronary arteries.

THE BODY'S RESPONSE TO EXERCISE IN HEALTH AND DISEASE

1.14. Physiological Responses. Physiological responses to exercise include improvement in strength, flexibility and work capacity.[59] The improvement in work capacity appeasrs to be achieved by a reduction in pulse rate, blood pressure, cardiac output, and muscle blood flow for a given work load, and an increase in arteriovenous oxygen extraction.[60] Prolonged exercise increases muscle myoglobin[61] and may affect the athlete's coronary risk profile by increasing the blood levels of HDL,[62] improving glucose tolerance,[63-5] and augmenting the rise in fibrinolytic activity induced by venous occlusion.[63] Athletes also tend to have fewer ectopics, low plasma triglycerides, low adiposity, and to smoke less.[66-7] Folkin and Amsterdam[68] reviewed a large number of stress studies and concluded that strenuous exercise resulted in a consistent and significant reduction in stress.

1.15. Response in Disease. There is some controversy

as to whether the improved work capacity results from central or peripheral adjustments.[60,69-74] Increasing efficiency of the mitochondria seems to be the most likely means,[70,75] since hearts damaged by infarction do not improve their contractility.[69] There have been reports (again anecdotal) of large bore coronary arteries with little or no atheroma in athletes[76-7] but little evidence that myocardial perfusion is increased, at least in hearts which have suffered infarction. There is however, abundant evidence from animal studies that exercise improves perfusion in ischemic, as distinct from infarcted myocardium[78-82] and while the whole subject of whether exercise can reduce the area of myocardium at risk in angina pectoris has been much neglected in humans, papers are beginning to suggest that it may indeed reduce the size of the ischemic area.[83-5] Why such an important field should be so fallow is a mystery.

THE BENEFITS OF EXERCISE

1.16. The Early Works. The benefits of exercise, at least in relation to CHD, have been defined epidemiologically in the main. Most reviewers agree that benefit was first suggested in Morris' comparison of mortality of London bus drivers and conductors[86] which showed that, in men under 50 years, the incidence of sudden death in

conductors was only one third that of drivers. Notwithstanding, Morris admits[87] that "stress" and not idleness was the prime suspect at the time. It was only after further studies on London transport[88-9] and on civil service clerks and psotmen,[90] together with similar studies from across the atlantic by Paffenbarger and his associates on longshoremen[91] and Harvard graduates[92] that firm weight could be placed on strenuous exercise. Nevertheless, these studies were haunted by the twin ghosts of self-selection - men who chose to become fit may have personalities, genetics or proclivities which lower their risk profile rather than the exercise itself - and ignorance of leisure time activity, which was surely as important as work activity.

1.17. Firming-up the Data. The latter was successfully dealt with in 1973 when Morris showed that leisure time activity was also protective from CHD when it was strenuous, and he was careful to define strenuous as "a calorific expenditure of greater than 7.5 kilocalories per minute, equivalent to heavy industrial work." This was found to include digging, swimming, tennis, hill climbing, running, squash, vigorous walking over 4 miles per hour, cycling and tree felling.[93] Both Morris and Paffenbarger found the protective effect to hold good for all ages, and for high risk sub-groups as well.[91,93]

The Framingham study came to the same conclusion even, surprisingly, when exercise was minimal.[94] Despite subtlties by both workers though, the question of self-selection has not been fully resolved. Paffenbarger goes some way to combat this in his longshoreman study,[91] although the fact that all workers entering the trade had to hold strenuous jobs initially and for 5 years thereafter does not entirely free the study from self-selection as Thomas suggests.[95]

1.8. Quality of Exercise. While the literature abounds with details of the quantity of exercise undertaken, few authors separate exercise into isometric and isotonic, despite there being considerable evidence that the effects of the two types on the cardiovascular system are importantly different.[7,96] The lumping together of these two different forms of exercise seems to be an important source of error in assessing the true value of exercise in the prevention of CHD and, perhaps more importantly, in assessing the risks of exercise.

1.19. Confirmation of Benefit. Notwithstanding, these findings on the apparent protective effect of exercise in CHD have been found in North Dakota farmers,[97] kibbutzim workers,[98] the Frammingham cohort,[99] and many others.[94,100-5] Several studies have shown no

effect on CHD mortality,[106-8] and one suggests that exercise had a deleterious effect.[109]

1.20. Mechanisms of Protection. The mechanism by which physical activity exerts its proposed effect is not known. Wood and his colleagues[110-1] found runners had a high level of high density lipoprotein cholesterol (HDL) compared with non-runners, and Castelli's group has shown this to afford protection from CHD.[112] The facility to supersaturate the plasma with cholesterol appears to reside in the HDL fraction, and it has been postulated that HDL exerts its protective effect by its ability to extract cholesterol from intimal cells into an already saturated plasma. Exercise also seems to lower traditional risk factors such as plasma triglyceride,[113-7] plasma cholesterol,[111,113,114,116,118] blood pressure,[119-23] anxiety,[115-6,126] obesity,[124-5] smoking,[124-5] and to promote a more healthy life style.[124,127]

1.21. Protection is Indirect. Since most animal and clinical and pathological studies of physical inactivity have not shown exercise to be directly related to the atheromatous process one must conclude that physical inactivity does not have a direct effect on atherosclerosis. Rather the effect seems to be to enable the body to tolerate ischemia better, and to alter other risk factors for

atherosclerosis.[128] None of the studies has shown a direct cause and effect relationship however, between exercise and reduction in CHD[95,129] and, in any case, as Kannel pointed out,[94] the fact that East Finland workers have both high physical activity and, at least until recently, the highest rates for CHD in the world, suggests that any such protective effect may be easily overwhelmed.

THE RISKS OF STRENUOUS EXERCISE

1.22. Causes of Death. Phaedippides may have been the first recorded case of sudden unexpected death during strenuous exercise but he certainly was not the last. Koskenvuo[130] reported 45 such cases in Finnish soldiers, Maron,[131] on 19 GI's, and Opie[23] on rugby players and referees. Cheitlin,[132] who did much of the work in establishing the link between anomalous coronary artery origin and sudden death on the sportsfield, has recently reviewed the causes of sudden death during exercise. He says "the known causes of sudden death in young people are few, and include congenital coronary artery anomalies such as anomalous origin of the left coronary artery from the anterior sinus of Valsalva, hypertrophic cardiomyopathy, aortic stenosis, and atherosclerotic coronary artery disease. Even rarer causes are prolapsed mitral valve and dissection of the aorta with rupture into the pericardium, coronary emboli from atrial myxoma, and

occlusion of the coronary ostium by vegetations, and benign papillary fibroelastosis of the aortic valve. Electrophysiological problems include accelerated atrioventricular conduction with atrial fibrillation causing ventricular fibrillation, and prolonged QT syndromes." He also considers whether bridging of the left anterior descending coronary artery by a band of myocardium may be another rare cause. Rather surprisingly, he fails to mention the well recognised association of hypoplastic coronary arteries with sudden death.

1.23. Unsubstantiated Dogma. The approach to the subject is unfortunately bedevilled by anecdotal reports and ex-cathedra statements by people who ought to know better.[22-3,133-4] There is, for example, little evidence[135] to support the contention that sportsmen are at risk of sudden death if they exercise with a fever, or that myocarditis, occult or not, is more likely to result in death if the patient exercises. And yet one sports cardiologist infers from "several anedotal reports of athletes collapsing while out for a run 'to sweat out' a cold or a fever" that "they presumably die from a lethal cardiac arrhythmia precipitated by a low grade myocarditis and potentiated by exercise" so that "any severe exertion, competition, or training during the course of, or in the immediate convalescent period, of an

acute pyrexial illness is therefore hazardous and should be severely discouraged."[133] Behind statements like these lies a deep and as yet untested universal feeling among even the specialists that strenuous exercise is intrinsicallly dangerous.

1.24. The Ventricular Ectopic. The role of the ventricular ectopic is also difficult to evaluate. Several studies have shown that arrhythmias provoked by exercise are predictive of CHD and sudden death,[136-8] particularly when they occur at low heart rates and are complex.[136-42] But, as Halpern[143] points out, these inferences depend on the likely prevalence of CHD in the population being tested.

1.25. Prediction of Sudden Death. Most studies of the total experience with death that occurs suddenly and unexpectedly, unassociated with trauma, conclude that about 90% are associated with pathologically significant coronary artery disease.[144-8] When such deaths occur in relation to sport the incidence is nearer 50%.[2] Prodromal symptoms are common[23] but tend to be ignored.[149-50] Wether prodromal symptoms are more common during exercise than at rest is not known. Superior physical fitness does not seem to guarantee protection from exercise deaths[149] and preliminary medical examination, in its present form, seems to be of little help in detecting

those at risk.[134,149,151]

1.26. The Bassler Hypothesis. Thomas Bassler exemplifies the prosyletising zeal which tends to characterise joggers. He stated, on doubtful grounds as it turned out, that " a search of the literature by the American Medical Joggers Association failed to document a single death due to coronary atherosclerosis among marathon finishers"[152] and "when the level of vigorous exercise is raised high enough the protection appears to be absolute."[153] Opie and Noakes, his principle antagonists in this, then produced evidence of coronary deaths in marathon runners.[154] Alas, this evidence turned to water when examined closely[155] and the South Africans retired in embarassment to retrench and, eventually, come up with the inevitable, proven, well-documented cases of coronary deaths in marathon runners,[25,156] as did others.[158] The Bassler hypothesis had had its day.[159] But at least it had helped to crystalise in people's minds the large body of evidence suggesting that sport helps prevent death from CHD.

1.27. Post Coronary Exercise. While successful marathon running is no guarantee of the absence of CHD[160] it would appear that the presence of CHD is no bar to marathon running either,[161] indeed it seems difficult to

stop some coronary patients from running marathons.[158,162] Shephard, whose Toronto group has considerable experience of post coronary exercise, quotes one of cardiac arrest in more than 250 000 hours of supervised exercise, which is not importantly different from the 364 000 patient hours reqired for one random re-infarction in the general non-exercising Toronto population as a whole.[134](Not everyone agrees with his figures though[163]) All in all there is abundant evidence that Glasser's remarks about sport being a "positive addiction" are accurate.[164]

1.1. Games and Sports in the Army

The following games and sports qualify for free travel concessions and the waiving of rents.*

Association football	Athletics
Badminton	Basketball
Billards	Birdwatching
Boxing	Cricket
Cycling	Fencing
Flying	Game Shooting
Gliding	Golf
Gymnastics	Hockey
Lawn Tennis	Modern Pentathlon
Motor Cycling	Mountaineering
Rackets	Riding
Rowing	Rugby
Sailing	Ski-ing
Squash Rackets	Swimming
Table Tennis	Target Shooting
Trampoline	

* See Games and Sports in the Army. Army code 61021 Amdt 3/Feb/1979.

CHAPTER TWO

MORTALITY FROM CORONARY HEART DISEASE IN THE BRITISH ARMY COMPARED WITH THE CIVIL POPULATION

INTRODUCTION

2.1. Soldiers exemplify the "healthy outdoor life" in that, not only do they often work out of doors, but they undertake strenuous physical exercise as an integral part of their daily lives. They might therefore be expected to have a reduced incidence of coronary heart disease (CHD).[63,74,86-8] On the other hand, soldiers are known to be heavier smokers than civilians,[165] and military duties may impose considerable amounts of stress. Because the population is so young, many coronary events come to cardiac catheterisation. Because this is done under civilian auspices and cardiac meetings mingle military and civilian angiograms, the clinical impression arose that CHD was more severe in soldiers than in civilians. This study is an attempt to evaluate this clinical impression.

AIM

2.2. The aim of this paper is to compare the mortality in the British army from CHD with that in the civil population of the United Kingdom.

SUBJECTS AND METHODS

2.3. The Army Population. Only British army males were considered (thus excluding the Brigade of Gurkhas and the Women's Services) who had died from CHD between 1 January 1973 and 31 December 1977. There were 156 such deaths but only the 148 cases under the age of 55 years were considered. Over this age the army population is too small for meaningful comparison.

2.4. The Civil Population. Civilian statistics were obtained from published data from the Office of Population, Census, and Surveys, the Scottish Home and Health Department, and the Northern Ireland Office.

2.5. Army Records. The medical statistics branch of the Ministry of Defence had recorded the deaths of 1258 soldiers between 1973 and 1977. That this was the total number of deaths in the period was confirmed by reference to the independently compiled Casualty List held centrally

in the Ministry of Defence.

2.6. Diagnosis. The recorded cause of death in every case was examined together with the clinical and pathological data on which the diagnosis was made, in an attempt to assess the firmness of the diagnsosis.

2.7. Accuracy of Diagnosis (Overall). Only 177 of the 1258 deaths had no record of post mortem,either because no post mortem was carried out, or because post mortem data were not available. In 45% of the deaths unconfirmed by post mortem there was clear ante mortem histological or radiological evidence of cancer, and a further 37% were clearly due to trauma. The rest (32 cases) were due to medical conditions for which the clinical data left no reason to doubt the diagnosis. Post mortems were carried out in the main by coroners and civilian pathologists in the United Kingdom, but also by military patholgists both in the United Kingdom and abroad.

2.8. Accuracy of Diagnosis (CHD). The diagnosis of death from CHD (as coded 410-14 in the eight edition of the International Classification of Diseases) was accepted in 156 cases. In 137 of these the diagnosis was made or confirmed at post mortem. Of the 19 deaths not so confirmed 9 had clinical and elctrocardiographic

evidence of acute infarction at demise, and a further 9 had a history of previous infarction of whom 7 had chest pain as a terminal symptom. Eight cases were over the age of 55 years and so excluded, leaving the study population of 148 cases.

RESULTS

2.9. Differences between Officers and Men. The Standardised Mortality ratio (SMR) for British army males compared with the civilian male population of the United Kingdom was 100. This, however, was made up from an excess number of deaths in the age group 20-39 years and a reduced number in the age group 40-54 years. Since most of the young men in the army are soldiers, there being relatively more officers with increasing age, SMRs were calculated separately for officers and soldiers. As shown in Table 2.1. the SMR for soldiers compared with male civilians was 148, and for officers 54 ($p < 0.001$).

2.10. Difference from Civilians. The SMR for soldiers under 40 years was significantly higher ($p < 0.01$) than the highest ratio among the civilian social classes (social class V), and that for officers of all ages significantly lower ($p < 0.01$) than the lowest ratio among these classes (social class I). Figure 2.2. is a chart

of age specific mortality ratios by social class in United Kingdom male civilians (1971), and by rank for British army males, showing this straddling of the civilian figures by the military, and suggesting a trend of diminishing difference in mortality with age between soldiers and officers, similar to that among the civilian social classes.

2.11. Gradation of Mortality with Rank. When officers were separated into direct entry officers and those promoted from the ranks, and soldiers divided into senior non-commissioned officers (SNCO's) on the one hand, and junior non-commissioned officers (JNCO's) and privates on the other, there was a gradation of increasing mortality with decreasing rank (Table 2.3.).

2.12. Gradation of Mortality Difference with Age. There was a gradient of decreasing difference in mortality rate with increasing age (Figure 2.2.).

2.13. Sudden Death is Commoner in the Young. Only 38 (26%) of the 148 fatalities survived long enough to reach hospital, but of the 78 under the age of 40 years, only 7(9%) survived. Death before reaching hospital seems commoner in the young.

POSSIBLE SOURCES OF BIAS

2.14. Regional Selection in Recruiting. The army is recruited disproportionately from areas of the country with a high incidence of CHD as shown in Table 2.4. However, standardisation show this bias to account for less than 2% of the observed difference between the army and the civil population.

2.15. Social Class Selection. Recruiting also tends to polarise social class, some officers being recruited from public schools and universities, and some soldiers enlisting by default of local employment. This social selection may account for part of the observed differences between civilian social classes and army ranks.

2.16. Personality Selection. Possibly recruits to the army may be disproportionately of the time-urgent type A personality, a possible risk factor for CHD.[166] If this were an important determinant however, it might be expected to increase mortality among officers too, since offficer recruits would be expected to show the same bias, and this is manifestly not the case.

2.17. Medical Care before the Event. The following factors, common to all ranks, might have been expected

to lower military mortality:

a. Induction Medicals. Potential recruits are screened on entry into the Service. Those with hypertension, cardiomegaly, renal disease, Cushing's syndrome, diabetes, or gross obesity are likely to be detected and excluded, although hyperlipidemia is not actively sought, and a positive family history of CHD is no bar to enlistment.

b. Routine Medicals. During the period studied it was policy to carry out medical examinations every 4 years on the fully fit, and annually on the unfit. The thoroughness of the examination varied, but measurement of weight and blood pressure, and urine examination for protein, blood and sugar is likely to have been carried out in most cases. Thus the diseases mentioned in the previous paragraph are likely to be detected if they develop later in life and, while not all lead to discharge from the Service, treatment, at least, is likely to be stated earlier.

c. Obesity Measures. Obesity is actively discouraged and in the grosser cases, oe 25% or more above optimal weight, downgrading to a lower medical category occurs which may result in delayed promotion.

2.18. Medical Care After the Event. Variations in factors

relating to circumstances following a coronary event may have introduced bias, thus:

a. Speed of Admission. No means could be devised to measure the relative speed of admission to hospital following a coronary event. Although soldiers often work in out of the way places they are seldom far from wheeled transport, and emergency helicopters are immediately available. The concept of high morale demands that soldiers be aware that high quality medical facilities are immediately available and the army gives this high priority.

b. Hospital Treatment. Treatment in hospital is much less important since only 38 of those who eventually died survived long enough to get there. However, since 18 of these were admitted under civilian auspices in the United Kingdom bias seems unlikely.

2.19. Medical Discharge. The process of medical discharge from the Service is an important source of bias, especially since it affects officers more than soldiers. In the period studied 19 officers and 20 soldiers were medically discharged with a diagnosis of CHD. On the unlikely assumption that all of these would have died had they remained in the Service, the SMRs for officers and soldiers become 79 and 178 respectively, still

appreciably different from the civil population.

CONCLUSIONS

2.20. This study suggests that:

a. British soldiers under the age of 40 years have a significantly higher mortality from CHD than their civilian counterparts irrespective of the civilians' social class.

b. British officers of all ages have a significantly lower mortality from CHD than civilians of any social class.

c. There is an inverse relation between CHD mortality and rank in the army similar to that seen is the civilian social classes.

d. There is a trend of diminishing difference in CHD mortality with age between officers and men similar to that seen in the civilian social classes.

e. Death before reaching hospital is commoner in the young.

DISCUSSION

2.21. This thesis is concerned hereafter in attempting to explain this mortality difference between soldiers and civilians, particularly with reference to the role of strenuous exercise, but also by examing the conventional risk profile for coronary heart disease. There are a number of ways in which strenuous exercise could influence mortality from CHD.

2.22. Accepting that soldiers exercise more, the amount of exercise taken is likely to be age related, with younger soldiers taking more. This could explain why the difference in mortality rates in maximal in the young. Nor is this postulate negated by the officers' trend in the opposite direction, since exercise could be precipitating a risk factor (eg, cigarette smoking) present in soldiers but not in officers.[167] According to Burch,[52-3] exercise, alone or in combination with another factor could facilitate the emergence of a forbidden clone, or shorten the latent period such that the disease would present earlier. Any such mechanism would have its maximal effect in younger age groups. Notwithstanding, civilians show the same age related mortality difference, making exercise less likely to be the culprit.

2.23. Alternatively, exercise might directly increase mortality in the following ways:

a. Precipitating Death. Exercise could, in itself, actually cause the death of a healthy soldier, or precipitate it in one with mild CHD. Alternatively, the presence of a mild upper respiratory or enterovirus infection might result in exercise associated death through the mechanism of occult myopericarditis, as suggested by Barlow.[22]

b. Affecting Infarct Size. The anoxia associated with exercise could result in the enlargement of any infarct that might fortuitously occur, and so increase mortality.

c. Affecting Speed of Demise. Exercise could, by inducing earlier dysrhythmia increase the proportion of sudden deaths, and so raise mortality.

2.24. The next chapters examine these and other concepts in greater detail. But before leaving this chapter brief mention will be made of the major points of difference between the life of a British soldier and that of a civilian.

THE SOLDIER'S LIFE

STRENUOUS EXERCISE

2.25. Mention has been made of the amount of exercise the soldier is required to perform, and of the steps the army takes to encourage sport. That this does indeed make soldiers fitter than civilians is shown by Amor [169] who, in a survey in 1975, found 45% of soldiers to be fit, in that they had a maximal oxygen uptake (VO_2 max) of at least 45 millilitres/kilogram/minute (mls/kg/min), and 16% were definitely unfit, with a VO_2 max of less than 35 mls/kg/min. These findings are similar to those in the United States army[170] but a study of Canadian armed forces found only officers and infantry soldiers to be fitter than civilians.[171]

DIETARY HABITS

2.26. Married Soldiers. 53% of soldiers are married and, while they are in the United Kingdom, can be expected to have the same dietary habits as any other British family. When serving abroad for more than a month or two the British soldier travels accompanied by his family and, by virtue of NAAFI, is able to, and normally does buy, and eat, normal British food. Thus the dietary habits

of married soldiers in quarters is unlikely to differ importantly from normal United Kingdom dietary habits.

2.27. Single Soldiers. Single soldiers in the main eat food prepared by the Army Catering Corps but also have access to shops and cafes. Menus in regimental dining rooms offer such a wide variety of dishes than, essentially, a soldier choses his own diet, and supplants it some 15-20% with outside purchases.[172]

2.28. Soldiers in the Field. The composition of soldiers' food both in barracks and in the field has been the subject of much research.[173-85] A recent study showed the fat content of soldiers' food in barracks is 42%, of which a high proportion is saturated fat, despite the use of polyunsaturated cooking oils.[185] On exercise, and these may last a few weeks to a few months several times a year, soldiers eat composite rations ("Compo") or other specialised diets where the fat content is of the order of 35%. A feature common to these arduous duty diets is the drive to weight reduction, and to this end fat content has been increased, experimentally at least, to 52%. Apart from the conversion to cooking with polyunsaturated oils, the army has interfered little in the traditional eating habits of the soldier.

SOCIAL STRESS

2.29. War. The Northern Ireland campaign was at its height during the periods under study in this thesis. From the beginnings in 1969 until the present, soldiers are required to perform a 4 month tour of duty in the province roughly every year and during these tours soldiers experience considerable stress. Responsability devolves onto section commanders of urban patrols of 5-6 men, at constant readiness to cope with sniping and bombing. In the heat of an incident the most junior soldier often has to decide for himself whether or not he has sufficient political justification to retaliate. Whatever the effect of this stress there is no change in the trend of CHD mortality during the first 10 years of the campaign.

2.30. Military Discipline. If anything, the effect of military discipline is to reduce stress in that it forces the soldier, in all aspects of his life, to conform to a set of rules which he not only understands but is also trained to apply. Breaking of the rules produces clearly defined punishments which are not unduly severe. Soldiers who might be disadvantaged in civilian life are protected by the regimental system.

2.31. Separation. Periods of separation from spouse

and family seldom last longer than 4 months but they may be frequent. A week or more on a course or exercise 3-4 times a year is common, and this on top of Northern Ireland tours. Such separation is the cause of much marital stress, on the one hand by fear of death or injury to the husband, and on the other by the very real worry that the wife will not be able to cope on her own.

2.32. Moving House. Soldiers move house every 2-3 years in the main but, while the interval is seldom longer than this, it may be much shorter. However, the stress of moving house may be less in the army than in civilian life since solders often translocate en masse, using well established procedures, into societies whose infrastructure he already understands. Nevertheless this is a major source of stress to soldiers wives, especially when combined with separation.

2.33. New Environment. Soldiers enlist on average at age 19 years and are likely to marry around the age of 23 years, as civilians do. During these 4 years of batchelorhood the soldier exchanges his parents' home for the barrack block. Stress from overcrowding is long since a thing of the past, billets are bright and spacious with 6 or so men to a room. Nevertheless there may be

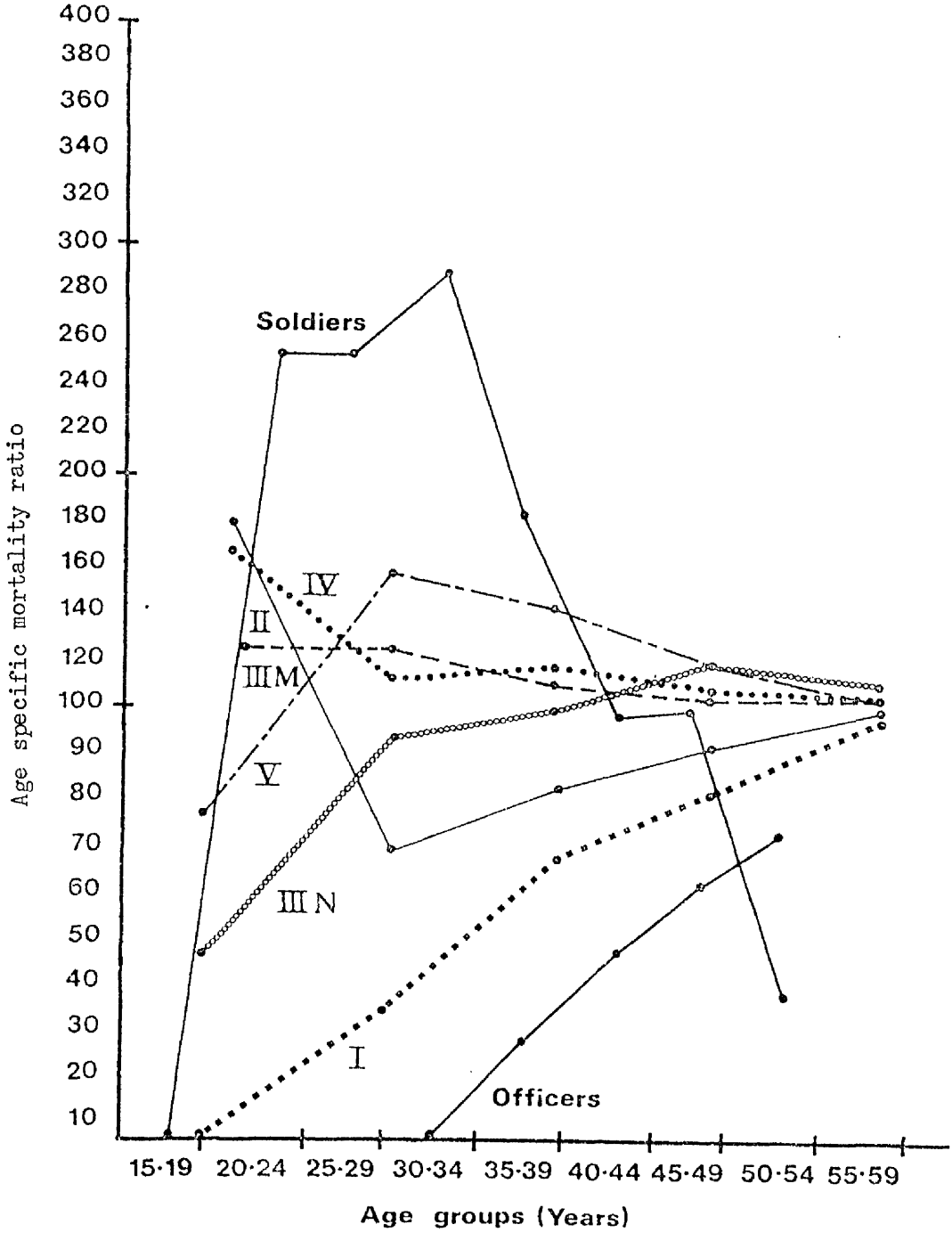
difficulty in escaping social contact.

2.34. Cigarette Consumption. A major difference in the soldier's life style, in relation to CHD, is cigarette smoking. Long hallowed by tradition the British army pays neither local nor British tax on cigarettes in certain parts of the world. In effect this means that in the British Army of the Rhine, which comprises one third of all British troops, the cost of cigarettes is about half that in the United Kingdom. As will be discussed later, this is reflected in soldiers' smoking habits, which are very different from civilians. (A similar trend may exist in relation to alcohol, but since this seems uniformly to be regarded as unconnected with coronary risk, this aspect has not been pursued.)

AGE	CIVILIAN	OFFICERS			SOLDIERS			ALL RANKS		
in years	per 1000 male popn	popn in 1000's	expected deaths in 5 years	observed deaths 1973-7	popn in 1000's	expected deaths in 5 years	observed deaths 1973-7	popn in 1000's	expected deaths in 5 years	observed deaths 1973-7
1-19	0.002	0.1	0	0	25.2	0.3	0	25.4	0.3	0
20-24	0.007	1.8	0	0	45.2	1.7	4	47.0	1.7	4
25-29	0.029	2.6	0.3	0	32.9	4.0	10	35.6	4.3	10
30-34	0.102	2.3	1.2	0	17.6	9.0	26	20.0	10.2	26
35-39	0.335	2.7	4.3	2	12.1	20.3	36	14.7	24.7	38
40-44	0.942	3.0	14.4	6	4.3	20.4	19	7.4	34.8	25
45-49	2.043	2.7	26.8	14	1.1	11.0	11	3.7	37.8	25
50-54	3.719	1.5	28.6	19	0.3	6.3	2	1.9	35.0	21
TOTAL		16.8	76	41	139.9	72.9	108	155.6	149	149
STANDARDISED MORTALITY RATIO			54			148			100	

2.1. Mortality from CHD by age and rank for British army males (1973-7) compared with the United Kingdom civilian male population (1973-7)

2.2. Age specific mortality ratios by social class by UK male civilians (1971) and by rank group for British Army Males (1973-77)



	Popn in 1000's	Expected deaths in 5 years	Observed deaths 1973-77	Standardised mortality Ratio
Direct Entry Officers	13.5	49	17	33
Officers promoted from the ranks	3.3	25	24	100
Senior NCO's	31.4	56	69	123
Junior NCO's & Privates	107.4	19	39	205
Total	155.6	149	149	100

2.3. Standardised mortality ratio of Army ranks compared with the UK male population

2.4. Recruiting by region of British Army males compared with population and incidence IHD (1973-76)

Region	Percentage of Population aged 15-19 years	Percentage of recruits	Civilian regional SMR for IHD
North	5.9	7.5	113
Yorkshire and Humberside	8.8	10.6	107
East Midlands	6.5	7.9	91
East Anglia	3.0	2.5	77
South East	28.7	19.6	87
South West	7.3	9.0	90
West Midlands	9.3	9.7	97
North West	11.6	14.9	115
Wales	4.8	5.0	117
Scotland	10.7	11.1	120
Northern Ireland	3.4	2.2	123

CHAPTER THREE

STRENUOUS PHYSICAL EXERCISE AND SUDDEN DEATH: INCIDENCE AND CAUSES

INTRODUCTION

3.1. Sudden unexpected death in relation to strenuous physical exercise has been widely documented[23-4,186-206] but there is no concensus that the relation is more than fortuitous.[187,190] Several authors have commented on the paucity of data on which to base a judgement.[24,148]

AIM

3.2. The aim of this paper is to define the incidence in soldiers of sudden unexpected death during strenuous physical exercise, the spectrum of diseases responsible, and to determine to what extent, if any, exercise is responsible for, or contributes to, sudden unexpected death in young men.

SUBJECTS AND METHODS

3.3. Population. Cases of sudden unexpected deaths during strenuous exercise were drawn from the population

of British army males during the years 1968-77, the average annual population being 160 000.

3.4. Age. The mean age of a soldier in 1977 was 26 years, and the age at entry 18.8 years. Very few serve beyond the age of 55 years. The age distribution of officers differs from that of other ranks as shown in table 3.1.

3.5. Exercise. During basic training all ranks undergo near maximal exercise for several months. Thereafter the amount of exercise varies considerably, but most soldiers undertake near maximal exercise on most days of the week. The minimum permissable level, until 1977, was an annual 10 mile hike with full kit, followed by an assault course. This has been changed with effect from April 1978 to a three mile run twice a year in 26 minutes (those over 25 years of age have an extra half minute for every half decade).

3.6. Access to Data. The central medical records of the army from 1969 are held at Stats (G)4, Ministry of Defence, Stanmore,Middlesex, and before 1969, at the Central Repository, Hayes, Middlesex.

3.7. Completeness of Data. A check on the completeness

of the fatality records was made by reference to the Casualty List of the Ministry of Defence, which is compiled separately. All deaths had been recorded and a diagnostic label attached, but data other than these bare facts was missing in 80 cases.

3.8. Deaths There were 2401 deaths among British army males in the decade 1968-77. The central medical records of all but 80 of these were examined in detail for evidence of sudden unexpected death having occurred during or within 6 hours of strenuous exercise (see 3.12. Definitions). Fifty-six such cases were found.

3.9. The Post Mortem Rate (Overall). The post mortem rate was 86% and half (47%) of those dying without post mortem had a diagnosis, made ante mortem, of cancer. In the other half, the main reason for lack of post mortem was death in an inaccessible place (20%).

3.10. Diagnosis. Of the 56 cases dying suddenly and unexpectedly within 6 hours of exercise only 6 did not undergo post mortem examination and of these 5 had ante mortem diagnosis of subarachnoid hemorrhage (SAH) confirmed clinically and by lumbar puncture. The sixth had clinical and electrocardiographic evidence of acute myocardial infarction. The post mortems were carried out in the main by United Kingdom coroners and

pathologists but also by military pathologists at home and abroad.

3.11. Other Relevant Deaths. The exercise assocated deaths occurred in a very small number of diagnostic categories. The total number of deaths in these categories and the numbers of deaths in other diagnostic categories which were not associated with exercise, but which might have been expected to have been (eg. cardiomyopathy, aortic stenosis) were also scrutinised.

3.12. Definitions.

a. Sudden Unexpected Death. Sudden unexpected death is death occurring without apparent cause within 6 hours of the onset of symptoms in an apparently healthy individual.[191] Thus death from trauma, heat hyperpyrexia and heat exhaustion were not included.

b. Strenuous Physical Exercise. Strenuous physical exercise is defined as exercise likely to approximate to the maximal aerobic capacity of the subject. The type of exercise undertaken is described in detail in each individual case.

RESULTS

3.13. Incidence. In 1.6 million man years there were 56 sudden unexpected deaths within 6 hours of strenuous physical exercise, giving an annual incidence of 3.5 per 100 000, or 2% of all deaths in the decade.

3.14. Range of Diseases. Sudden unexpected deaths during strenuous physical exercise fell into 5 diagnostic categories (Table 3.2.). Thirty five deaths (63%) were due to CHD, 10(18%) to subarachnoid hemorrhage (SAH),5(9%) to congenital cardiac anomalies, and 3(5%) each to infection and unknown causes.

3.15. Coronary Heart Disease. The 35 deaths from CHD formed 12% of the toal deaths from CHD in the decade. Nineteen of the 35 cases (Table 3.3.) had complete occlusion of a coronary artery, 10 had severe stenosis and 2 had moderate stenosis. Post mortem results were unavailable in 2, and in one case no post mortem was carried out. In 22 of the 23 cases on which information was available atheroma importantly involved more than one coronary artery.

a. Age. The mean age at death from CHD was 41(SD 6) years and that from the exercise associated cases was 36(SD 6) years, the difference being highly significant.

However, this difference disappears when the bias of older age groups exercising less in obviated by considering only deaths under 35 years of age. Thus there were 101 deaths from CHD under 35 years of age of which 14 occurred in relation to exertion. The mean age at death overall was 29.6(SD 4) years, and for the exercise associated cases 30.0 (SD 4).

3.16. Subarachnoid Hemorrhage. Ten (33%) of the 33 deaths from SAH in the decade occurred during strenuous exercise, but the type of exercise undertaken by these cases was very different(Table 3.4.). Only 2 deaths occurred in relation to formal sport, the others occurring during various forms of sudden strenuous isometric work associated with the Valsalva manoeuvre.

a. Age. The age at death was the same whether the subject died during exercise (28(SD 5) years) or not (28(SD 7) years).

3.17. Congenital Abnormalities. Of the 10 deaths due to congenital abnormalities of the coronary and great vessels, half occurred in relation to strenuous exercise (Tables 3.4. and 3.5.)

a. Age. The mean age at death in the 5 cases that were exercise associated (22(SD 6) years) was lower than that in the 5 cases that were not (29(SD 8) years), but not significalntly so. When only deaths due to

coronary artery abnormalities are considered however, despite the small sample size, soldiers who died during strenuous exercise were very significantly ($p < 0.001$) younger than their counterparts(Tables 3.4. and 3.5.)

METHODOLOGICAL OBSERVATIONS

3.18. Checks. While a check could be made on the overall numbers and on the accuracy of the diagnosis, the study depends upon the recording of the association of strenuous exercise and sudden death, and no check could be made on this. However, in view of the dramatic nature of this association and the general wealth of clinical detail in army records, it is likely that the association was recorded in most cases.

3.19. Completeness of Data . In only 80 (3%) of the total of 2401 deaths in the decade were the records unavailable to confirm the recorded diagnosis.

3.20. Definition of Exercise. Strenuous physical exercise is an unsatisfactory term and is as poorly defined here as in the literature generally. The pathophysiological adjustments associated with jogging 100 metres are importantly different from those related

to lifting a sack of potatoes onto a lorry.[96] Clearly there are at least two types of exercise, each associated with different risks, and they should be assessed separately.

3.21. Subversion?. It could be said, and indeed has been, that the paucity of unexplained deaths results from unwillingness by the army to accept the moral and legal consequences of unexplained death on the sportsfield. This implies that clinicians and pathologists subvert their professional honesty on joining the army and become more respectful of adminsitrators! In fact the wealth of clinical and pathological detail to corroborate the diagnosis should put to rest any doubts about this and in any case, civilian coroners made the diagnosis in nearly two thirds of cases.

CONCLUSIONS

3.22. The annual incidence of sudden unexpected death during strenuous physical exercise is very low at 3.5 per 100 000 and these deaths account for only 2% of all deaths.

3.23. Sudden unexpected deaths during strenuous exercise are due to a small number of causes. Thus 63% were due to CHD,18% to SAH, 9% to congenital cardiac anomalies,

and 5% each to infection and unknown causes.

3.24. Those who died from CHD had severe and widespread disease and were already at very high risk. The risk for those with lesser degrees of severity appears to be small.

3.25. Strenuous exercise does not appear to precipitate death from CHD since those who died had very severe disease and the age at death was the same whether death occurred in relation to exertion or not.

3.26. Deaths from SAH occurred during heavy work rather than strenuous exercise. The high proportion associated with exertion (30%) suggests a causal relation inspite of there being no difference in age at death between exercisers and non-exercisers.

3.27. Subjects with congenital abnormalities of the coronary arteries are at high risk of sudden deaths during strenuous exercise since half of all such deaths were related to exertion and these cases were significantly younger.

3.28. No deaths occurred from aortic stenosis or from hypertrophic obstructive cardiomyopathy despite their

anecdotal reputation.

3.29. No sport could be identified as particularly dangerous. No deaths occurred in relation to the game of squash rackets although it is a popular game in the army and has a popular reputation as being a dangerous sport.

3.30. Unexplained death was no more common during exercise than at rest. It occurred during exercise only 3 times in 10 years.

3.31. There is no evidence that concomittant minor infection increases the risk of sudden death during exercise.

DISCUSSION OF RESULTS

PAUCITY OF SIMILAR STUDIES

3.32. South African Rugby Players. A search of the literature reveals only one study similar to this one, which devotes itself exclusively to sudden death in relation to strenuous exercise.[23] By searching the South African newspapers Opie found 21 sudden deaths in relation to sport, particularly rugby football, of whom

18 were thought to be due to CHD, and one to SAH. By estimating the length of the game and the number of games the player was likely to have taken part in, he arrived at the estimate of one death per 50 000 rugby hours for players, and one per 3 000 rugby hours for referees - elastic figures, to be sure.

3.33. Finnish Recruits. Koskenvuo,[130] in a more controlled study on Finnish recruits, estimated that the incidence of sudden death from all causes was 6.8 per 100 000, of which "the onset of acute symptoms occurred during strenuous exercise in a third." This incidence is remarkably similar to that in the present study. The causes of death in this third (13 cases, when heat stroke is excluded) were CHD and myocarditis in 3 cases each (one each of which had an associated congenital cardiac abnormality), and one case each of SAH, hypoplastic aorta, medial necrosis, arterial rupture in the neck, and 3 from unknown causes.

3.34. Other Studies. Mortiz[200] attempted a similar study using autopsy reports from the United States army during the Second World War. Unfortunately, lack of a denominator, and of a clear cut numerator in many cases, precludes anything more than general conclusions, at least as far as the present study is concerned.

Notwithstanding, he found that the principal categories of disease responsible for sudden death were "heart disease, intracranial hemorrhage, meningococcemia, miscellaneous diseases, and cause of death not disclosed by autopsy." More specifically Luke[207] reviewed 275 consecutive cases of autopsies of sudden unexpected deaths in young adults aged 20-45 years. His criteria were similar to those in the present study, except that he excluded cases in which the cause of death could not be found (5% in his series, the same as in this one). He found that CHD accounted for 28% of sudden unexpected deaths in young adults, and various forms of intracranial bleeding for a further 20%. Similar conclusions have been reached by Kuller,[145] Murphy,[205] and others,[147,194] suggesting that, with the exception of coronary artery anomalies, the spectrum of causes of sudden death during exercise is similar to that overall.

CORONARY HEART DISEASE

3.35. The Diagnosis. 50% of the deaths from atheromatous CHD undergoing post mortem examination had thrombotic occlusion of a coronary artery and, of the remaining 16, 14 had severe or occlusive atheroma, and 2 moderate atheroma. Myocardial ishcemia is thus very likely to have been the actual cause of death in these 31 cases.

3.36. Severity. It can be seen from Table 3.3. that those who died of CHD had severe and widespread disease. By comaprison only 12% of Korean War casualties, of similar age, had narrowing of 50% in a single coronary artery.[192] Thus those who died were already a high risk group. The paucity of deaths associated with less than severe and widespread disease supports the corollary that those with mild to moderate disease, and those with single vessel disease are at low risk.

3.37. Proportion of CHD Deaths with Exercise. 12% of coronary deaths in the army occurred in relation to strenuous exercise. This compares with 2.1%,[187] 5%,[190] 28%,[193] 65%,[130] and 85%[23] in other series. At first sight the figure of 12% might seem disproportionately large since even solders do not spend 12% of their time (3 hours of every day) exercising. However the figure refers to deaths within 6 hours of exercise and not just to deaths during exercise. Nevertheless there is room for some suspicion since in point of fact very few deaths occurred more than one hour after exercise. A recent study on joggers in Rhode Island comes to a similar conclusion, namely that there may be a very small increase in risk of coronary death with sport.[208] The population was however poorly defined, and the study was much smaller than this one.

3.38. Comparative Age. In an international study of men under 40 years of age who died from myocardial infarction unrelated to exertion, the mean age at death was 35.4 years.[209] In this study, despite the army's bias to younger age groups (Figure 3.1.), the mean age at death for men under 40 years of age whose deaths were related to exercise was 33 (SD 5) years.

SUBARACHNOID HEMORRHAGE

3.39. Type of Exercise. Comparison of Table 3.3. with Table 3.4. reveals a clear dichotomy in the types of exercise related to death from CHD and SAH. SAH appears related to static heavy work while CHD, and the other categories of death, relate to various forms of running in the main. This has been commented on before, and the explanation forwarded is that heavy work, like lifting weight, is associated with breath holding which is in effect a powerful Valsalva manoeuvre which results in intense peripheral vasoconstriction in response to the diminishing cardiac output. On cessation of the exercise and resumption of breathing a high pressure ejection from the left ventricle results in unusually high wall stress and so rupture at a site of weakness.

CONGENITAL ABNORMALITIES

3.40. Anomalous Origin of the Left Coronary Artery. The 2 cases of death from anomalous origin of the left coronary stem from the right coronary cusp occurred during strenuous exercise (Table 3.3.). In both cases the subsequent course of the artery was backwards between the aorta and the pulmonary artery. The association of this anomaly with sudden death, particularly during strenuous exercise, is well known.[195-6] Indeed the risk of death in subjects with such anomalies has even been quantified at 27%.[197] Authors have speculated that the immediate change of direction of the artery on emergence, with close application to the aortic wall results in its compression during ejection of a large cardiac output.

3.41. Hypoplasia of the Coronary Arteries. Hypoplasia and atresia of the coronary arteries are also well recognised as causes of sudden unexpected death.[198-9] In this series the two exercise related cases had coronary arteries free of atheroma, but the 4 cases which were not associated with exercise (Table 3.5.) all had severe stenosis and were older. Mild atheroma may produce severe stenosis in hypoplastic arteries and cause ischemia. Strenuous exercise, by increasing

myocardial oxygen requirements, may have a similar effect and produce death before atheroma develops.

CONSPICUOUS ABSENTEES

3.42. Aortic Stenosis. No deaths occurred from occult aortic stenosis either during exercise or normal activity. Since there is a traditional association between tight aortic stenosis and death on the sportsfield, its absence from the present study is surprising. It may arise from the much reduced incidence of aortic valve disease now that rheumatic heart disease has almost disappeared, and also perhaps from detection in initial and subsequent medical examinations in the army.

3.43. Hypertrophic Obstructive Cardiomyopathy. Routine medical examinations are much less likely to detect hypertrophic obstructive cardiomyopathy and indeed there were 5 deaths from this cause in the present study (Table 3.5.) However, none occurred in relation to exercise although all but one were under the age of 35 years and were likely to have been exercising regularly.

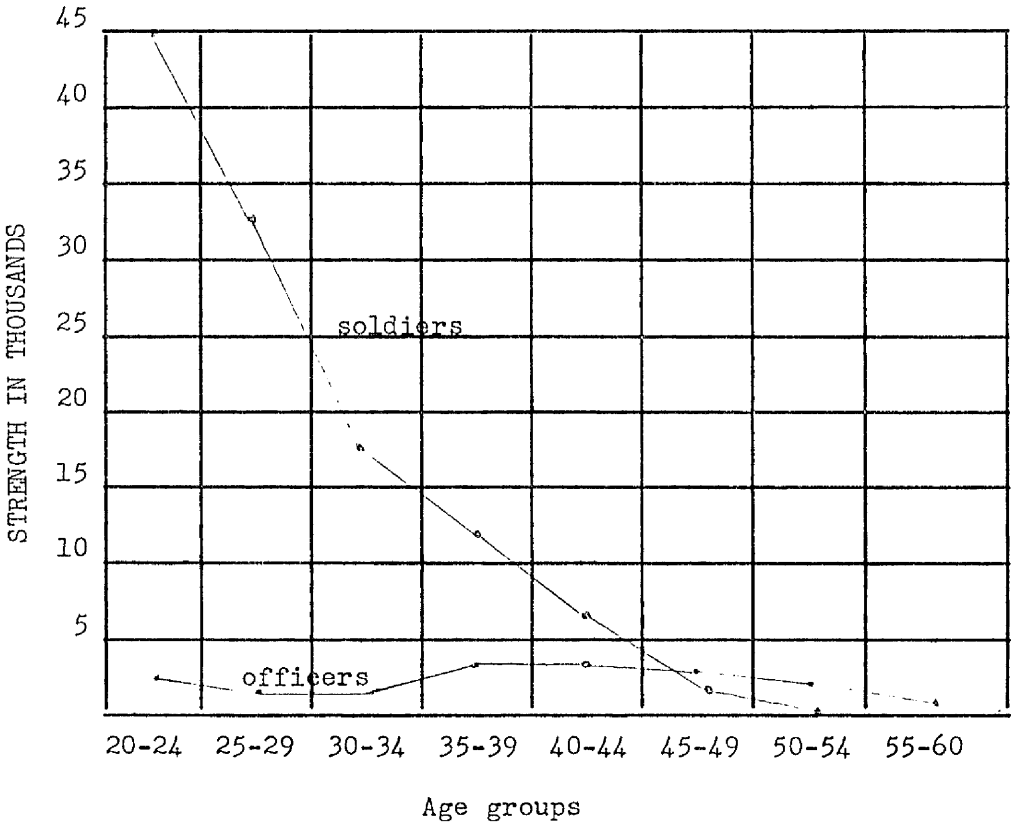
3.44. Occult Myopericarditis. Only one out of 12 deaths from myopericarditis was related to exercise (findings similar to those of Moritz[200]) and, of the 3 unexplained

deaths, only one could possibly have been due to occult myopericarditis. So, in spite of good theoretical reasons to the contrary, [22,193] occult myopericarditis is unlikely to be an important cause of death in young men who undertake strenuous activity while harbouring symptoms of upper respiratory or enterovirus infection.

3.45. Congestive Cardiomyopathy . It has been postulated that even if sudden death does not occur from occult myopericarditis, then congestive cardiomyopathy might.[197] There is no evidence from this study to support this association, there being only 2 such deaths in the decade.

3.46. No Cause Found. Only 3(5%) of the 56 deaths were unexplained. This compares with 19(5%) of the 379 deaths unexplained overall in the categories of death in which exercise associated deaths occur (Table 3.2.). Thus while James[188,203] points to purkinje anomalies as a cause of sudden death during exercise and others to a build up of dysrhythmogenic metabolites,[202] the number of cases not open to resolution by routine post mortem would appear to be small overall, and the association with strenuous exercise does not appear to increase their number.

3.1. Army strength by age



cause of death	myocardial ischemia	subarachnoid haemorrhage	congenital cardiac anomaly	infection	unknown	total
exercise deaths	35	10	5	3	3	56
total deaths	295	33	10	26	19	382

3.2. Causes of sudden death during strenuous exercise

3.3. Coronary heart disease and sudden death in sport.

No	Age	Activity	Thrombus	Worst Vessel	Gen State
1	33	rugby	yes	LMS occl at 1.5cm	
2	23	waterskiing	yes	LMS occl	severe
3	35	BFT	yes	LAD occl at 2cms	nil else
4	24	brawling	yes	LAD occl at 2cms	severe in Cx
5	26	route march	yes	LAD occl at 1cms	severe in LAD
6	43	training run	yes	LAD occl	severe
7	29	climbing	yes	LAD occl	severe
8	35	football	yes	LAD occl	RCA occl
9	50	lawnmowing	yes	LAD occl	RCA occl
10	43	basketball	yes	LAD occl	
11	35	circuits	yes	LAD and Cx occl	many 90% sten
12	35	water polo	yes	Cx occl	
13	39	BFT	yes	Cx occl	severe
14	40	football	yes	Cx occl	severe
15	25	football	yes	RCA occl at 1cm	
16	25	brawling	yes(old)	LMS occl	
17	34	rugby	no	LAD occl(ath)	mod in RCA
18	35	climbing	no	LAD occl(ath)	severe
19	41	football	no	LAD occl(ath)	severe
20	42	cycling	no	LMS pinpoint sten	severe in RCA
21	37	marching	no	LMS pinhole sten	severe
22	45	running	no	LAD pinhole sten	mod RCA and Cx
23	31	training run	no	LMS 80% sten	
24	34	running	no	LAD sev ath	severe Cx
25	30	training run	no	LAD sev ath	severe Cx
26	36	BFT	no	LAD sev ath	
27	34	cycling	no	LAD sev ath	severe RCA & Cx
28	36	football	no	gen severe	gen severe
29	32	X-country	no	gen severe	gen severe
30	34	rugby	no	moderate ath	gen moderate
31	36	cycling	no	moderate ath	old scar
32	53	lawnmowing		no PM data	
33	46	pushing car		no PM data	
34	41	weightlifting		no PM data	
35	49	tennis		no PM	

LMS - left main stem coronary artery
LAD - left anterior descending coronary artery
Cx - circumflex branch of left coronary artery
RCA - right coronary artery
PM - post mortem
BFT - basic fitness test (see text)

3.4. Non-atheromatous causes of sudden death in sport

No	Age	Activity	Cause of death
1	22	loading truck	SAH-RMCA aneurysm
2	35	carrying friend	SAH-LACA aneurysm
3	26	brawling	SAH-PCA aneurysm
4	30	marching	SAH-no aneurysm at PM
5	15	brawling	SAH-no aneurysm at PM
6	21	football	SAH-no PM
7	31	football	SAH-no PM
8	53	sexual intercourse	SAH-no PM
9	29	electrocution	SAH-no PM
10	19	pushing vehicle	SAH-no PM
11	20	sprinting	distal coronary artery atresia
12	17	unspecified	hypoplastic coronary arteries
13	20	physical training	ectopic origin of LCA
14	19	three mile run	ectopic origin of LCA
15	35	unspecified	Marfanoid aortic dissection
16	17	7-a-side rugby	myopericarditis
17	19	running	endocarditis
18	21	training run	bronchopneumonia
19	29	hockey	no cause found
20	19	10 mile march	no cause found
21	19	4 mile run	no cause found

SAH - subarachnoid hemorrhage
MCA - middle cerebral artery
ACA - anterior cerebral artery
LCA - left coronary artery
PCA - posterior communicating artery
PM - Post mortem examination

No	Age	Cause of Death
1	49	hypoplastic CA with gross atheroma
2	27	hypoplastic CA with left main stem occluded by atheroma
3	35	hypoplastic CA with circumflex and LAD occluded by atheroma
4	39	hypoplastic CA almost obliterated by atheroma
5	28	hypoplastic aorta 8mm in diameter with left CA occluded by atheroma
6	20	HOCM
7	51	HOCM
8	31	HOCM
9	27	HOCM
10	33	HOCM
11	18	inactive myocarditis
12	28	acute on chronic myopericarditis
13	30	focal myocarditis
14	19	chronic myocarditis
15	16	acute myocarditis
16	15	acute rheumatic carditis
17	17	acute myocarditis
18	20	myocarditis
19	42	tamponade from myopericarditis
20	36	rheumatic carditis with previous valvotomy
21	20	fibrinous pericarditis with bronchopneumonia
22	22	congestive cardiomyopathy
23	15	congestive cardiomyopathy

CA - coronary arteries
HOCM - hypertrophic obstructive cardiomyopathy

3.5. Relevant deaths unassociated with strenuous exercise

CHAPTER FOUR

MORTALITY RISK OF EXERCISE ASSOCIATED CORONARY EVENTS COMPARED WITH THOSE OCCURRING AT REST

INTRODUCTION

4.1. During exercise myocardial oxygen demand is increased. Coronary artery occlusion occurring by chance at this time might be expected to be associated with an increased risk of mortality via, for example, an increase in infarct size. While anecdotes abound, the author is unaware of any work specifically orientated to this topic.

AIM

4.2. The aim of this paper is to show whether or not a coronary event coming on in relation to strenuous exercise carries a higher mortality than one occurring at rest.

SUBJECTS AND METHODS

THE DEAD

4.3. Population. This study covers the years 1973 -7

and uses the same population as that described in Chapter Two (paras 2.3.-2.8.) but includes all ages.

4.4. Activity at Death. Examination of the medical records of the 156 deaths from CHD revealed information on the deceased's activity within 6 hours of demise in 98 cases (62%). In a further 19 cases the patient had a serious illness likely to preclude strenuous exercise (16 had a previous myocardial infarction, 2 occurred in the post-operative period, and one was an asthmatic on treatment). Thus in 117 cases (75%) a clear statement could be made as to whether or not the patient had undertaken strenuous exercise within 6 hours of demise. The actual number who had was 22 (Table 4.1.).

THE LIVING

4.5. Population. Only British army males who had suffered one or more non-fatal coronary events in the 5 years 1973-7 were considered.

4.6. Source of Data. The number of coronary events which occurred, and the number of patients in whom they occurred, were taken from the hospital discharge summaries received by the medical statistics department. It is, and has been, army policy that any suspected

coronary event, of whatever nature, is assessed in hospital. No cases are treated at home. With our youthful population, and a high percentage of youthful, inexperienced army doctors as general practitioners and Regimental Medical Officers, it is likely that this policy was complied with fully. Nevertheless, it can be seen that admission to hospital is inherent in the definition of a coronary event.

4.7. Completeness of Records. The medical statistics branch receives reports of hospital admissions as well as discharges, and these admission figures were used to assess the completeness of the discharge reporting. Hospital discharge reports are received for 89% of all hospital admissions, including those to civilian hospitals, the frequency of reporting varying with the severity of the condition and the length of stay in hospital (by reason of exercise, billet accommodation etc, the army admits many minor cases to hospital). CHD is not separately coded, being lumped under Circulatory Diseases (Chapter VII of the International Classification of Diseases, 8th revision), but the report rate for similarly serious diseases such as neoplasia, psychiatric illness, and acute appendicitis, is 100%.

4.8. Diagnosis. Only cases reportedly due to CHD

were investigated to ascertain the minutiae of the diagnosis. Thus no check was made on all other diagnostic categories to find cases of CHD which had been missed. The diagnosis of CHD was made in all cases by hospital physicians. 426 such episodes (non-fatal) were reported.

4.9. Accuracy of Diagnosis. Such was the quality of the records that the liklihood of the physician's diagnosis being accurate could be assessed. Six categories of probablity were used (Table 4.2.):-

a. Definite Myocardial Infarction. The diagnosis of myocardial infarction could be verified in 195 cases by a history of typical chest pain and unequivocal electrocardiographic evidence of acute transmural infarction in all cases, and by supporting evidence (usually cardiac enzyme levels, but also thallium scan or angiography) in all but 43 cases.

b. Definite Angina Pectoris. The typical history of angina in 50 cases was supported by a positive maximal exercise test in 15, angiography in 11, ischemic electrocardiogram in 5, previous infarction in 8 and subsequent infarction in 11.

c. Probable Coronary Event. Probable CHD was

diagnosed when there was typical chest pain (typical of myocardial ischemia) with other abnormalities suggestive of CHD but not amounting to transmural infarction (Table 4.3.).

d. Doubtful Coronary Event. The criteria used to diagnose doubtful CHD are detailed in Table 4.4.

e. Not Coronary Heart Disease. In 16 cases labelled as CHD there were data to confirm conditions associated with, or likely to mimic CHD, but none to incriminate CHD itself (Table 4.5.).

f. Others. Thirty-five admissions were for further investigations and not for the coronary event, and in 9 cases the central medical file could not be obtained.

4.10. Activity at the Onset of Symptoms. Of 426 non-fatal events, 16 were wrongly diagnosed and 35 were investigative readmissions. Of the remaining 375 episodes, 74 were second or subsequent episodes and the subjects were therefore unlikely to have been taking strenuous exercise. Of the remaining 301 in whom a single event occurred during the period of study, 23 had had a coronary event prior to this time, and these too

were unlikely to have been exercising. There remains 278, in whom a history of exercise was available in 125 and a history of rest in 95, leaving only 58 (15%) in whom no such data were available (Table 4.6.). Of the 125 in whom a history of exercise was given, only 60 were indulging in strenuous exercise (Table 4.7.). In 65 cases the patient was said to have exercise associated chest pain without the type of exercise being specified. Almost all of these fell into the angina or doubtful CHD diagnostic categories and carried a bald statement such as "the patient complained of chest pain on exertion", often with a remark on duration eg:"for 6 weeks". Under these circumstances it was thought likely that the exercise was an exaggeration of normal activity rather than strenuous. Also it was felt that the severity of exertion would influence the patient's statements and so those of the recorder. Finally, since exercise associated non-fatal events are the denominator, the use of the smaller figure ensures than any error will exaggerate the mortality rate from exercise related events.

RESULTS

4.11. Mortality rate for all Coronary Events. Of 582 episodes diagnosed as coronary events, data were available to disprove the diagnosis in 51 cases, all non-

fatal (Table 4.2.). Of the remaining 531 coronary events, 156 had a fatal outcome(29%). Twenty-two fatal (Table 4.1.) and 60 non-fatal (Table 4.7.) coronary events were associated with strenuous exercise, giving a mortality rate for exercise related events of 27%, which is not significantly different.

4.12. Mortality rate for Proven Myocardial Infarction. When only proven transmural infarction is considered, and assuming that all deaths from CHD had, or would have had, transmural infarction, then there were 156 deaths from transmural infarction and 195 survivors, giving an overall death rate of 44%. Twenty-two fatal(Table 4.1.) and 26 non-fatal(Table 4.7.) transmural infarctions were related to strenuous exercise, giving an exercise associated mortality of 46%.

4.13. Mortality Rate for Proven and Probable Coronary Events. There were 292 non-fatal events in the proven and probable categories(Table 4.2.) giving an overall mortality for those likely to have had a coronary event at 35%(Table 4.8.). Forty-eight of those non-fatal events were exercise related (Table 4.7.), giving an exercise associated mortality of 33%(Table 4.8.).

CONCLUSION

4.14. Whether diagnostic criteria are strict or lax, the mortality rate for coronary events is not increased when these events occur in relation to strenuous exercise.

DISCUSSION

4.15. In 85% of non-fatal events it was possible to say whether or not the patient had been exercising. The effect of any further exercise associated cases in the unrecorded 15% would have been to lower mortality from exercise associated events.

4.16. Similarly, in 65 cases where the record merely stated "chest pain on exertion" the exercise was assumed not to be strenuous. Any error resulting from this assumption would lower the exercise mortality still further.

4.17. No exercise data were available in 39 of the 156 fatalities. A plausible reason for this is that no connection with exercise existed. In any case it is likely that the proportion of exercise deaths in this group was smaller than in the main group. But even if it were the same, this would add another 4 deaths to

the 22 already discovered and raise the mortality to 30% which is still similar to the overall mortality of 29%.

4.1. Deaths from coronary heart disease in relation to exercise.

NO	AGE	ACTIVITY	DOMINENT LESION
1	25	football	thrombotic occlusion
2	40	football	thrombotic occlusion
3	34	rugby	thrombotic occlusion
4	39	three mile run	thrombotic occlusion
5	25	brawling	thrombotic occlusion
6	29	climbing	thrombotic occlusion
7	35	circuit training	thrombotic occlusion
8	35	football	thrombotic occlusion
9	43	training run	thrombotic occlusion
10	41	football	atheromatous occlusion
11	35	climbing	atheromatous occlusion
12	36	football	severe stenosis
13	34	three mile run	severe stenosis
14	31	training run	severe stenosis
15	45	running	severe stenosis
16	32	cross country run	severe stenosis
17	37	marching	severe stenosis
18	42	cycling	severe stenosis
19	30	running	severe stenosis
20	34	rugby	moderate stenosis
21	41	lifting weights	necropsy data not available
22	49	tennis	necropsy not carried out

4.2. Diagnostic probability of CHD in survivors

Diagnosis	Exercise associated	Non-exercise associated
Myocardial infarction	33	162
Angina	50	-
Probable CHD	8	39
Doubtful CHD	10	64
Not CHD	-	16
Missing data	-	9
Investigative admissions	-	35
Total	101	325

4.3. Criteria for probable coronary event.

T wave flat or inverted in SI lead of ECG	17
Inferior MI diagnosed on inferior Q waves or transient T changes on ECG with no rise in enzymes	7
Transient ST elevation or depression	2
Positive exercise test	5
Left ventricular failure with past proven CHD	4
Left ventricular failure with CHD proven at post mortem	3
Ventricular tachycardia or ventricular fibrillation with previously proven myocardial infarction	4
Intermittent atrial fibrillation with previously proven myocardial infarction	1
Q waves in lead SI of the ECG with elevated enzymes	1
Chest pains only with acute myocardial infarction two months later	1
Coronary bypass surgery as the only record	1
Equivocal ECG and elevated enzymes	1
	47

4.4. Doubtful cases of Coronary Heart Disease.

Previous infarction. Little evidence of a new event	13
ECG changes during non-CHD illness or surgery	8
Chest pain unsupported by other evidence of an event	19
Chest pain with equivocal ECG or exercise test	18
Chest pain with normal ECG but raised cardiac enzymes	7
Chest pain with positive ECG or exercise test but with negative thallium scan or angiogram	8
Abnormal ECG only	1
	74

4.5. Conditions wrongly diagnosed as Coronary Heart Disease

1. Mitral leaflet prolapse
2. Hypertension
3. Atypical chest pain. Bad family history
4. Hypertension. Intermittent claudication
5. Chest pain. Psychiatric discharge
6. Femoral aneurysm
7. Hypertrophic cardiomyopathy
8. Computer error
9. Intermittent atrial fibrillation
10. Abnormal ECG. Normal angiogram
11. Pneumonia and myopericarditis
12. Exercise associated chest pain
13. Renal artery stenosis. Hypertension. Left ventricular failure
14. Hypertension. Diabetes. CVA. Claudication
15. Chest pain. Equivocal ECG. Positive exercise test. Hypoplastic circumflex coronary artery without atheroma.
16. Unable to pass basic fitness test

4.6. Breakdown of activity data

Total events	582	
Deaths	156	
	History of strenuous exercise	22
	History of normal activity	76
	History of serious illness	19
	No data available	39
Survivors	426	
	Wrong diagnosis	16
	Investigative readmissions	35
	History of unspecified exercise	125
	(History of strenuous exercise 60)	
	History of normal activity	95
	History of previous coronary event within the study period	74
	History of previous coronary event outwith the study period	23
	No data available	58

4.7. Survivors from a coronary event during strenuous exercise.

A. Myocardial Infarction

No	Age	Activity
1	33	circuit training
2	47	squash
3	39	hockey
4	38	6-a-side football
5	31	12 mile route march
6	47	walking the dog
7	34	3 mile run
8	43	three mile run
9	42	one mile run
10	34	3 mile run
11	43	hockey
12	42	mowing the lawn
13	36	mountaineering
14	40	squash
15	36	4 mile run
16	25	swimming
17	41	lifting a weight
18	43	stoking a boiler
19	26	3 mile run
20	42	moving house
21	35	football
22	29	3 mile run
23	51	golf
24	47	tennis
25	43	walking steep hill
26	44	lifting boxes

B. Angina

No	Age	Activity
1	34	road running
2	39	walking uphill
3	37	3 mile run
4	44	lifting boxes
5	33	playing football
6	35	stoking boiler
7	37	3 mile run
8	38	cross country run
9	38	coitus
10	33	dancing
11	37	swimming
12	49	climbing stairs
13	41	40 step-ups
14	46	walking uphill

C. Probable CHD

No	Age	Activity
1	33	running
2	39	running
3	43	tennis
4	45	gardening
5	36	5-a-side football
6	47	running
7	41	gardening
8	45	climbing stairs

D. Doubtful CHD

No	Age	Activity
1	42	walking uphill
2	35	swimming
3	33	running
4	39	running training
5	40	hill walking
6	28	rugby
7	35	3 mile run
8	40	marching
9	36	walking uphill
10	43	cycling
11	29	one mile run
12	35	half mile run

4.8. Mortality rate for coronary events.

	OVERALL	STRENUOUS EXERCISE
CRUDE MORTALITY	$\frac{156}{582} = 27\%$	$\frac{22}{82} = 27\%$
CORRECTED MORTALITY (excluding wrong diagnosis and investigative admissions)	$\frac{156}{531} = 29\%$	$\frac{22}{82} = 27\%$
MORTALITY FROM PROVEN TRANSMURAL INFARCTION	$\frac{156}{351} = 44\%$	$\frac{22}{48} = 46\%$
MORTALITY FROM PROVEN AND PROBABLE CORONARY EVENTS	$\frac{156}{448} = 35\%$	$\frac{22}{70} = 31\%$

CHAPTER FIVE

CORONARY RISK PROFILE OF YOUNG SOLDIERS WITH CORONARY HEART DISEASE

INTRODUCTION

5.1. It would seem clear from the foregoing chapters that strenuous exercise is not an important cause of the increased mortality from CHD seen in soldiers. This chapter now examines the more conventional risk factors for CHD.

SUBJECTS

5.2. The subjects of this paper are 164 British army males with CHD under the age of 40 years (mean age 34.6 ±3 years) diagnosed between 1976 and 1981.

METHODS

DIAGNOSIS

5.3. The diagnosis of CHD was established as follows:-

a. Coronary Arteriogram. In 48 cases there was a report of a coronary arteriogram showing luminal obstruction by clot or atheroma of at least 70% in

at least one coronary artery.

b. Post Mortem. A detailed autopsy report showing evidence of at least "severe atheroma" in at least one coronary artery was accepted as diagnostic of CHD in a further 48 cases.

c. Classical Myocardial Infarction. In a further 55 cases a statement of typical chest pain associated with deep Q waves on electrocardiogram, and a confirmatory rise in cardiac enzymes were the minimal acceptable criteria.

d. Others. A further 13 cases with chest pain typical of myocardial infarction or angina were accepted with:-

i. Q waves on the electrocardiograph and a corresponding defect on thallium scan (5 cases).

ii. ST or T wave changes on electrocardiograph with a defect on thallium scan (4 cases).

iii. ST segment depression on electrocardiograph of at least 2 millimeters on symptom limited maximal exercise testing, and a defect on thallium

scan with late infilling (one case).

iv. Deep anterior Q waves on electrocardiogram (3 cases).

DATA COLLECTION

5.4. Data were collected from two sources:-

a. The Central Medical Envelope which contains data on routine medicals before enlistment, before and after basic training, together with all hospital in-and out-patient summaries during service, and the final discharge medical or post mortem report.

b. Data from a cardiac rehabilitation programme conducted by the author over two years.[210]

PARAMETERS

5.5. Relative Body Weight. Measurement of height was taken from the recording at the end of basic training, when the mean age was 19.5 years. Review of army statistical tables[211] shows no increase in height thereafter. Weight was recorded from peri-incident reports, usually at readmission for definitive investigation and rehabilitation. The relative body weight was

calculated by the reputedly suitable Quetelets' formula [212]

$$\frac{\text{weight(kgs)}}{\text{height(cms)}^2} \times 100$$

and obesity defined as a relative body weight of 0.28 or more. This corresponds to a 20-25% increase over the ideal body weight according to the criteria of the Metropolitan Life Insurance Company.[213]

5.6. Family and Social History. A positive family history was recorded when myocardial infarction or angina had occurred in the patient's family, his parent's family, or in that of his grandparents. These data were collected from peri-incident reports. The type and quantity of tobacco consumed was defined numerically in a high proportion, and these data were also taken from peri-incident reports.

5.7. Blood Pressure, Plasma Lipids and Glucose Tolerance

Blood pressure recordings were taken from peri-incident reports. Plasma lipids were estimated always on a follow up admission from a fasting specimen. Glucose tolerance was measured similarly on fasting and two hour post prandial specimens in the main, although 15 were measured on a single fasting specimen, and one on urine testing.

5.8. Anthropomorphic Data. Anthropomorphic data were available in 94 cases (57%). The mean height and weight were 173.3(SD 6) centimeters and 81(SD 12) kilograms respectively (Figure 5.1.). The mean relative body weight was 0.27(SD 0.04) and 44% were obese (Table 5.2.).

5.9. Tobacco Consumption. Tobacco habits were recorded in 114 (70%). Four (4%) were non-smokers and 110 (96%) were smokers. One hundred and five smoked cigarettes (85%) 4 smoked a pipe and one cigars. In 17 cases the word "smoker" only was used, and in 5 "heavy smoker". The estimated consumption of these cases was 20 and 30 cigarettes daily respectively, as shown in Figure 5.3. Five smoked 2 ounces of shag tobacco each per week in "roll your own" cigarettes. This was taken as equivalent to 20 cigarettes per day. The average number of cigarettes smoked was 28 (SD 13). 89% of the cigarette smokers smoked 20 or more daily, 40% 30 or more, and 24% 40 or more.

5.10. Blood Lipids. The total serum cholesterol was measured in 93 (57%) and the mean level was 7.29 (SD 2.12) millimoles per litre (Figure 5.4). Taking 7.54 mmol/l as the upper limit of normal for this age group (30-40

years), there were 25 cases in which it was elevated (27%). The mean of these abnormal values was 9.85 (SD 2.17) mmol/l. Thirty-three of these cases also had high density lipoprotein cholesterol estimations carried out and the mean value was 1.16 (SD 0.33) mmol/l. The mean value of 79 triglyceride estimations was 2.05 (SD 1.24)mmol/l.

5.11. Blood Pressure. Blood pressure was recorded in 89 cases (54%). Thirteen (15%) were greater than 140/90, of which 6 (7%) were greater than 150/100, and 4 (4%) were greater than 160/110.

5.12. Glucose Tolerance. Three (4%) of 70 estimations had abnormal glucose tolerance. None were frankly diabetic.

5.13. Family History. Of 129 cases in which it was recorded, 57 (44%) had a family history of myocardial infarction or angina (Table 5.5.). The age at onset of symptoms in fathers of index cases was recorded in 28 of 29 cases: nine (32%) were under 50 years of age.

POSSIBLE SOURCES OF BIAS

5.14. Weight. Weight measurements were taken in the main from recordings taken 6 weeks after a coronary event and so might be expected to be a little high as a result

of inactivity. Whilst this is likely to bias the comparison with normals, it is probable that other studies on CHD patients would show similar bias.

5.15. Smoking Habit. A similar bias might result from a more honest confession of smoking habit following discovery of CHD than might be expected from routine questioning of the healthy.

5.16. Smoking Prevalence-"Worst Case" Situation. Tobacco habit was recorded in only 70% of cases. In order to establish that the findings from this proportion are a true reflection of the whole population, let us consider the "worst case" situation, in which all those on whom there are no data are, in fact, non-smokers. There are then 110 smokers in the population of 164, giving a "worst case" prevalence of 67% smokers. Since this figure is still in excess of the civilian male prevalence, it would seen that the inference that high prevalence of cigarette consumption is an important risk factor holds good.

5.17. Cigarette Consumption-"Worst Case" Situation. Similarly, in relation to the quantity of cigarettes consumed, 40% of the 110 smokers consumed 30 or more cigarettes per day. In the "worst case" scenario all

the unknowns are smokers who smoke less than 30 cigarettes per day. There are then 44 who smoke 30 or more cigarettes per day in a population of 160 smokers, giving a prevalence of 28%. Since, in the army as a whole, the proportion of smokers smoking 30 or more cigarettes per day is only 12% the inference that heavy smoking is an important risk factor holds good too.

COMPARISONS

5.18. Anthropomorphometry. The Pulheems Administrative Pamphlet (based on the General Household Survey 1962) in which the army lays down the ideal indices of height and weight, recommends a Quetelet's index of 0.225 for the age range 30-40 years. However, a study by Amor of over 3 000 healthy soldiers of similar age in 1975 found a mean index of 0.238.[169] This compares with an index of 0.25 in a similar study in the United States army.[170] British civilian studies on slightly older age groups show indices variously 0.243,[214] 0.244,[215] and 0.26.[216] Thus British soldiers with CHD (mean index 0.27) have greater relative body weight than healthy soldiers or healthy civilians.

5.19. Smoking Prevalence. 96% of soldiers with CHD were smokers. In a British army cohort study on the

smoking habits of healthy soldiers the figures for 1959,[217] 1964,[218] and 1969[165] were respectively 76%, 75%, and 72%. Different studies on the same population showed the figure in 1975 to be 76%[169] and in 1980, 60%.[167] Studies in the United States army variously report the prevalence of smoking in the late 1970's as 61%,[219] 58%,[170] and 38%.[220] The equivalent figure for United Kingdom male civilians in 1976 was 50% and in 1980, 45%.[221]

5.20. Cigarette Consumption. The mean number of cigarettes smoked was 28 per day. Healthy soldiers smoked 21 cigarettes per day in 1975[169] and the number smoking more than 20 per day was about twice the national average.[169] In United Kingdom male civilians the mean cigarette consumption in the age group 30-40 years was 10 cigarettes per day in 1976 and 9 in 1980.[168] In the present study 40% of smoking soldiers smoked 30 or more cigarettes per day. The figure for healthy soldiers who smoke 30 or more per day is 12% in the British army,[165] and 7% in the United States army.[170]

5.21. Total Cholesterol. In the present study the mean total serum cholesterol level was 7.29 mmol/l, and 27% had levels in excess of 7.54 mmol/l. There have been no comprehensive studies of blood lipids in the British army, but the mean level in the United States

army was 4.6 mmol/l, and 7% had a level in excess of 6.48 mmol/l.[170] This compares with a level of 5.64 mmol/l in United States civilians.[220] In normal middle aged British civilians total serum cholesterol varies between 6.0 and 6.6 mmol/l.[216,222]

5.22. HDL Cholesterol. The mean level of high density lipoprotein cholesterol in the present study (1.16 mmol/l) is not importantly differnt from the range found in normal middle aged British male civilians (1.07-1.22 mmol/l).[222]

5.23. Triglyceride. Similarly, the mean triglyceride level of 2.05 mmol/l is similar to that found among normal middle aged British male civilians (1.8 mmol/l).[216]

5.24. Other Studies on Young Sunjects with CHD. Very few studies on the risk profile of men under 40 years of age with CHD have been carried out. From Table 5.6. it can be seen that the prevalence of abnormal glucose tolerance, and of hypertension, is much lower in this study than in others (presumably as a result of army medical selection), and a positive family history has the same prevalence as in other Western studies. Hypercholesterolemia appears highly prevalent in young subjects with CHD compared with the middle aged, but the prevalence in the British army subjects is similar to that

found in young Scandinavians,[223] young Scots,[209] and young white South Africans.[224] On the other hand, high relative body weight and high cigarette consumption appear importantly more prevalent among the soldiers.

DISCUSSION

5.25. Of the parameters studied, high relative body weight, high cigarette consumption and high serum cholesterol are the main risk factors associated with CHD in young soldiers, and the first two are more prevalent than in similar civilian studies. Levels of HDL cholesterol, triglyceride, and blood pressure are similar to those among affected civilians, and the low prevalence of glucose intolerance is likely to be due to the army's invaliding policy for diabetics.

5.26. While young soldiers with CHD are markedly more obese than similar civilians, the prevalence of obesity in the army as a whole is slightly lower than among civilians. In trying to explain this phenomenon it should first be remembered that the inference of obesity from high relative body weight is a qualified one in fit and relatively muscular young men. Nevertheless, obesity may cluster among CHD cases through the medium of alcohol, heavy consumption of

which, particularly beer, would be associated with both obesity and high cigarette consumption. Obese soldiers have great difficulty in passing the Basic Fitness Test and are often downgraded medically for both reasons. The net effect is the soldier is excused exercise and consequently loses its putative cardioprotective effect.

5.27. The prevalence of elevated serum cholesterol in the army is not known, nor is it clear why levels should be so high in affected soldiers, but since the levels in the abnormals were very high indeed (mean 9.85 mmol/l) it is possible that this group consisted in part of soldiers with familial hypercholesterolemia. One might expect them to develop CHD symptoms at this time, and for their number to be large because of the large number of men of this age in the army. When those with levels greater than 9 mmol/l are excluded, the mean level in the 73 cases remaining is 6.56 mmol/l, and when 8.5 mmol/l is the cut off point, the remaining 67 cases have a level of 6.37 mmol/l. These figures are more in keeping with those for normal civilians, and for older populations with CHD. The same phenomenon is seen in the Scandinavian study[223] and to some extent in the 9 Countries study,[209] although the latter's international nature obscures the point somewhat. Thus young men with CHD show high levels of

cholesterol because CHD is uncommon at this age except in those with familial hypercholesterolemia. In older age groups where CHD is common this effect is much less dramatic. If high serum cholesterol is a risk factor common to all young people with CHD, it cannot be regarded as the explanation for the soldiers' higher mortality from CHD. It does suggest, though, that measurement of serum cholesterol on induction might be a means of reducing manpower wastage from CHD.

5.28. Young soldiers with CHD not only have the highest prevalence of cigarette smoking, but also the highest per capita consumption, and the highest proportion of heavy smokers of all populations, military or civil, healthy or affected, that the author has been able to study. Since their smoking habit prior to enlistment is the same as the civilian population from which they derive[217] and since their adult and lifelong smoking habits are established by the age of 20 years[218,221] it would seem that the critical time which determines their high risk profile is the first few years of army life. It is at this point that young soldiers are introduced to a population of heavy smokers and can, in many parts of the world, indulge the habit at approximately half the cost which pertains in the United Kingdom. This divergeance of smoking pattern which occurs in the early

years of army life would appear to be the critical factor in explaining why soldiers have a greater mortality from CHD than comparable civilians. This phenomenon also adds weight to the "latent period" of "incubation" of CHD as propounded by Burch [52-3] and quantified by Rose.[225]

5.29. Thus the critical difference between the risk profiles of soldiers and civilians appears to be smoking habit, and this is likely to be the most important factor in explaining why soldiers have a higher mortality from CHD than comparable civilians. Rather like the case of the Karelian lumber-jacks, any beneficial effect from strenuous exercise is swamped by an increase in cigarette consumption.

CONCLUSIONS

5.30. High prevalence of heavy cigarette consumption is the main reason why soldiers in the British army have a higher mortality from CHD than British civilians.

5.31. Young men in general, with CHD, have a higher prevalence of cigarette smoking, and a higher per capita consumption of cigarettes than older men with CHD.

5.32. Young men with CHD have high total cholesterol

levels when compared with older men similarly afflicted. This may be due to a higher preponderance of symptomatic cases of familial hypercholesterolemia in the younger age group.

5.33. The high prevalence of obesity in symptomatic young soldiers may have resulted from relative inactivity and may also be linked to heavy cigarette consumption through the social medium of heavy beer drinking.

5.34. A family history of CHD is no more prevalent in symptomatic young soldiers, and in young men in general, than it is in the general population.

5.35. Hypertension and glucose intolerance are not important risk factors in young soldiers, probably as a result of frequent medical screening.

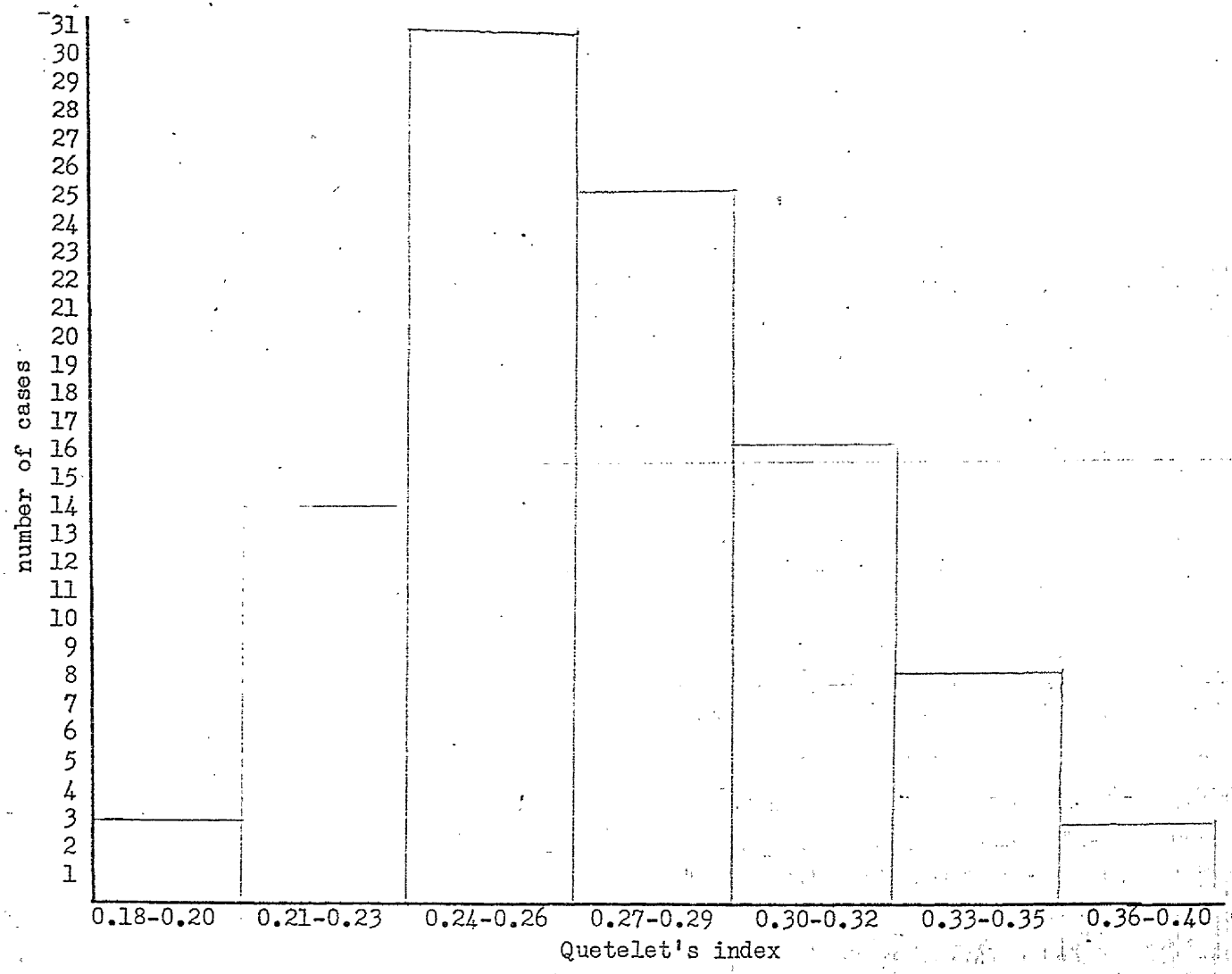
5.36. The first few years of army life is the time at which soldiers' smoking habits increase to very high levels.

5.37. Access to cheap cigarettes appears to be an important contributant to high cigarette consumption, and so to high prevalence of CHD.

5.38. In the British army, the expected protective effect of physical fitness is overwhelmed by the deleterious effect of high cigarette consumption.

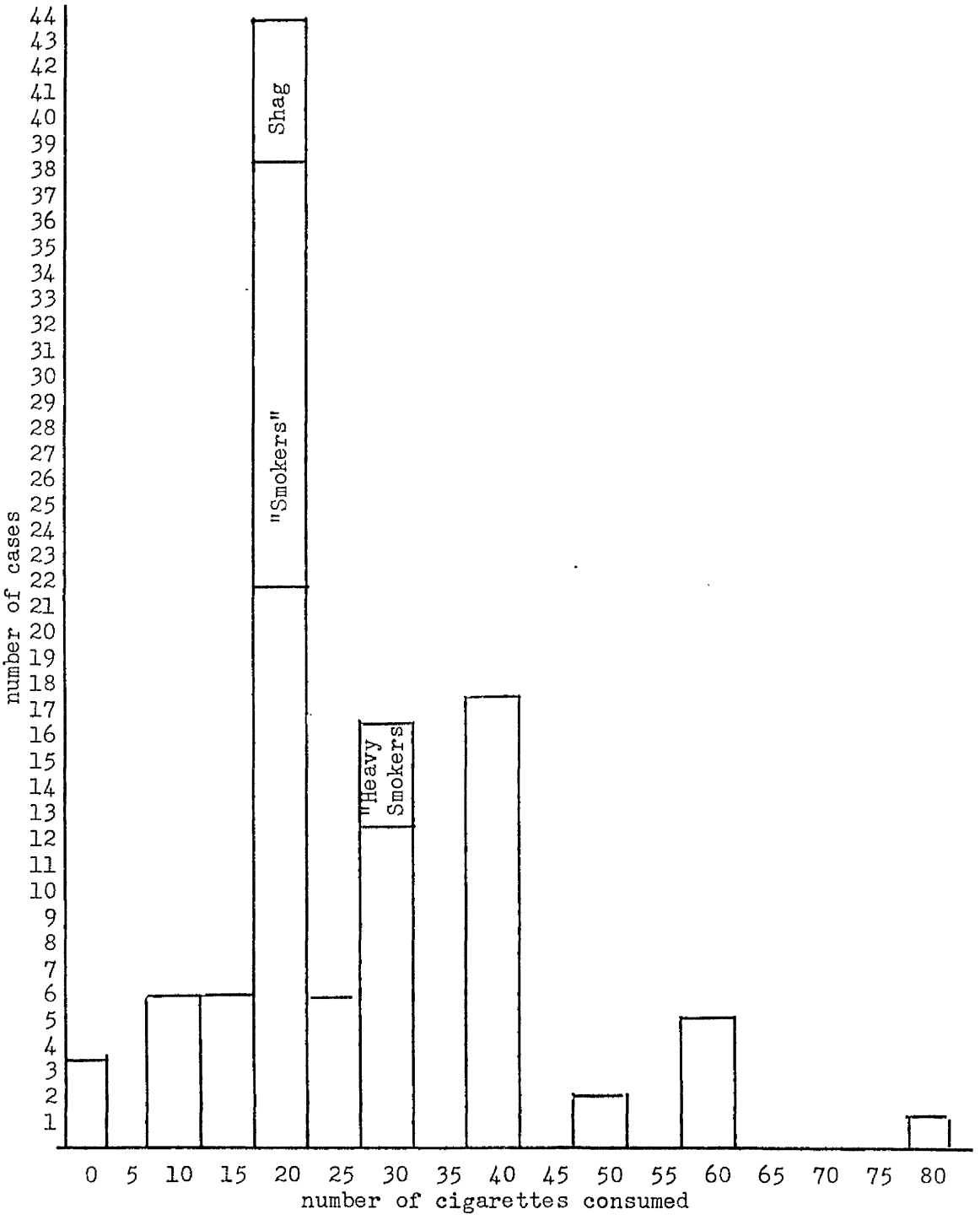
NO	mean height±SD cms	mean weight±SD kgs	relative body weight			
			mean±SD	< 0.24	0.24-27	≥ 0.28
94	173.3±6	81.4±12	0.27±0.04	17(18%)	36(38%)	41(44%)

5.1. Height, weight and relative body weight.

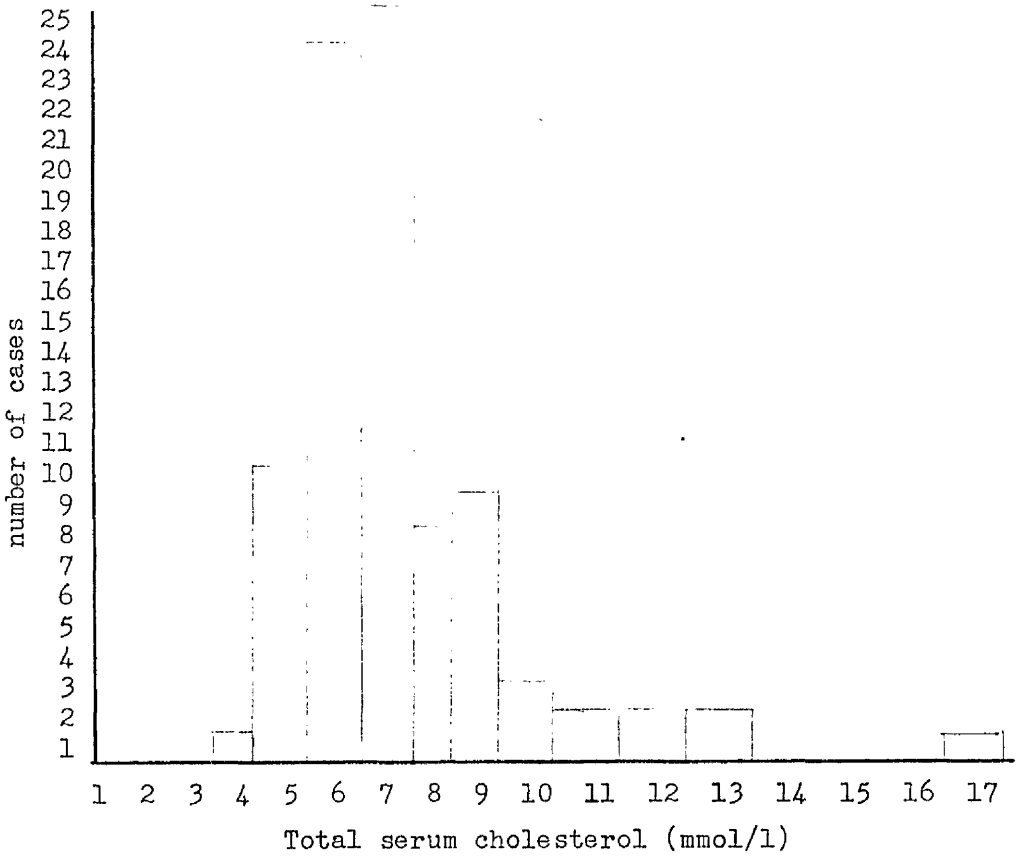


5.2. Relative body weight of young soldiers with CHD

5.3. Cigarettes consumption in young soldiers with Coronary Heart Disease



5.4. Total serum cholesterol in young soldiers with Coronary Heart Disease



conversion from SI units to traditional units:
Cholesterol 1mmol/l = 38.6 mgs/dl

5.5. Family history of Coronary Heart Disease

No family history	72(56%)
Father	29(22%)
Mother	12(9%)
Both parents	2(2%)
Siblings	4(3%)
Others	10(8%)

	Present study	(226) Glover	(223) Nitter-Haug	(228) Bergstrand	(209) Dolder	(227) Chinniah	(224) Kennelly
number of cases	164	89	66	24	240	94	100
total cholesterol (mmol/l)	7.29	normal	8.2	7.5	--	--	6.75
% hypercholesterolemic[1]	21.5	20	35	60	25	9	17
% smokers	96	89	86	92	80	76	85
% heavy smokers[2]	49	--	--	50	39	27	33
% overweight[3]	44	--	30	8	19	25	27
% with family history	44	48	44	20	25	4	31
% hypertensive	4	21	24	9	15	20	11
% glucose intolerance	4	--	--		10	20	7

5.6. Risk profiles of subjects under 40 years of age with Coronary Heart Disease.

1. Present study $\geq$ 9mmol/l. Glover, not specified. Nitter-Haug $\geq$ 8.8mmol/l. Bergstrand $\geq$ 7.0mmol/l. Dolder $\geq$ 7.25mmol/l. Chinniah $\geq$ 7.8mmol/l. Kennelly $\geq$ 7.8mmol/l.
2. Consuming 25 or more cigarettes per day.
3. 20% overweight or Quetelet's index $\geq$ 0.28.

CHAPTER SIX

CONCLUSIONS

SOLDIERS' MORTALITY FROM CHD

6.1. Soldiers in the British army under the age of 40 years have a significantly higher mortality from CHD than their civilian counterparts irrespective of the civilians social class.

6.2. British officers of all ages have a significantly lower mortality from CHD than civilians of any social class.

6.3. There is an inverse relation between CHD mortality and rank in the army similar to that seen in the civilian social classes.

6.4. There is a trend of diminishing difference in CHD mortality with age between officers and men similar to that between the civilian social classes.

6.5. Death before reaching hospital is commoner in the young.

EXERCISE AND SUDDEN DEATH

6.6. The annual incidence of sudden unexpected death during strenuous physical exercise in the British army is very low at 3.5 per 100 000, and these deaths account for only 2% of the total deaths.

6.7. Sudden unexpected deaths during strenuous exercise are due to a small number of causes, thus 63% were due to CHD, 18% to SAH, 9% to congenital cardiac abnormalities, and 5% each to infection and unknown causes.

6.8. Those who died from CHD had severe and widespread disease and were already at very high risk. The risk for those with lesser degrees of severity appears to be very small indeed.

6.9. Strenuous exercise does not appear to precipitate death from CHD since those who died had very severe disease and the age at death was the same whether death occurred in relation to exertion or not.

6.10. Death from SAH occurred during heavy work rather than strenuous exercise. The high proportion associated with exertion (30%) suggests a causal relation in spite of there being no difference in the age at death of

exercisers and non-exercisers.

6.11. Subjects with congenital abnormalitites of the coronary arteries are at high risk of sudden death during strenuous exercise since half of all such deaths were related to exertion, and these cases were significantly younger.

6.12. No deaths occurred from aortic stenosis, or from hypertrophic cardiomyopathy despite their anecdotal reputation.

6.13. No sport could be identified as particularly dangerous. No deaths occurred in relation to the game of squash although it is a popular game in the army and has something of a reputation as a dangerous sport.

6.14. Unexplained deaths were no more common during exercise than at rest. Only 3 such deaths in 10 years were associated with exercise.

6.15. There is no evidence that concommittant minor infection increases the risk of sudden death during exercise.

RELATIVE MORTALITY OF EXERCISE ASSOCIATED CORONARY EVENTS

6.16. There is no increase in mortality when a coronary event occurs in relation to strenuous exercise.

CORONARY RISK PROFILE

6.17. High prevalence of heavy cigarette consumption is the main reason why soldiers in the British army have a higher mortality from CHD than British civilians.

6.18. Young men with CHD in general have a higher prevalence of cigarette smoking and a higher per capita consumption of cigarettes than older symptomatic men

6.19. Young men with CHD have high total serum cholesterol levels when compared with older men with CHD. This may be due to a preponderance of symptomatic cases of familial hypercholesterolemia in the younger age group.

6.20. The high prevalence of obesity in symptomatic young soldiers may have resulted from relative inactivity and may also be linked to heavy cigarette consumption through the social medium of heavy beer drinking.

6.21. A family history of CHD is no more prevalent in

symptomatic young soldiers, and in symptomatic young men in general, than it is in the general population.

6.22. Hypertension and glucose intolerance are not important risk factors in young soldiers, probably as a result of frequent medical screening.

6.23. The first few years of army life is the time at which soldiers' smoking habits increase to very high levels.

6.24. Access to cheap cigarettes appears to be an important contributant to high cigarette consumption, and so to high prevalence of CHD.

6.25. In the British army, the expected protective effect of physical fitness is overwhelmed by the deleterious effect of high cigarette consumption.

VALEDICTORY REMARKS

6.26. This thesis was concerned to show that mortality from CHD is greater among young British soldiers than among similar civilians, and to investigate why this should be so, with particular reference to the role of strenuous exercise. It has shown that strenuous exercise does not precipitate death from CHD, or from any other

cause, with the exception of subjects with congenital coronary artery abnormalities who are at definite risk, and subjects with the propensity to subarachnoid haemorrhage who may be at risk. Further, the risk of death from a coronary event occurring randomly during strenuous exercise is no greater than that from one occurring at rest. In any case the incidence of death during strenuous exercise is too small to influence the mortality comparison to any important extent. Rather the answer lies in the coronary risk profile of those affected. These young men smoke four times as many cigarettes as their civilian counterparts, more than any group available for study, and they derive from a population whose per capita consumption is three times that of the civilian population. An important reason for this is likely to be the army's access, in many stations, to cigarettes at roughly half the normal cost in the United Kingdom. Since adult and lifelong smoking habits are fixed before the age of 20 years, the first few years of service are critical in this respect. A problem has been identified and its cause elucidated. Treatment is political and dependent on many variables, an important one of which is medical will.

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