PSYCHOSOCIAL PROFILE OF A SAMPLE OF EGYPTIAN PATIENTS WITH ISCHEMIC HEART DISEASE

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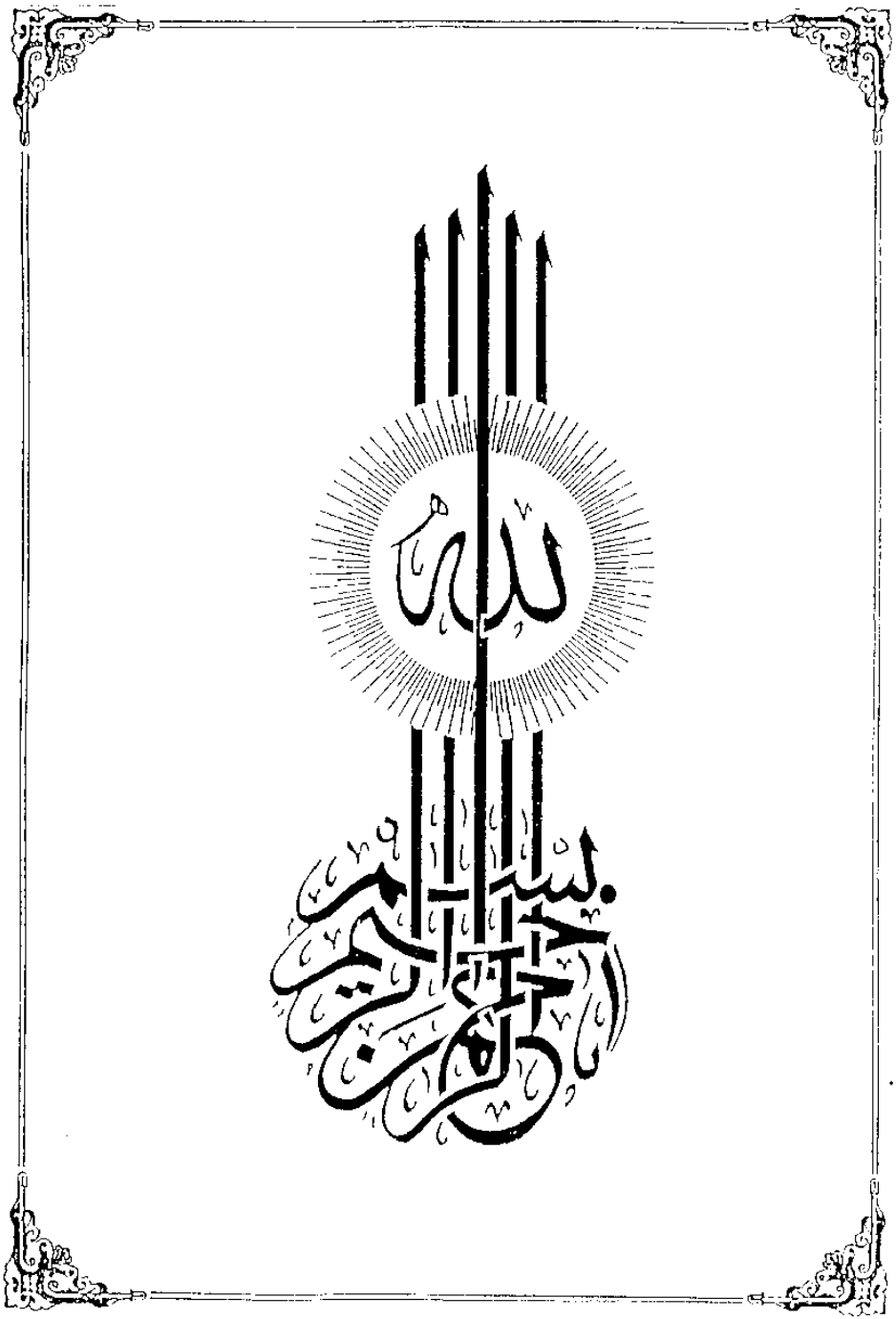
C O N T E N T S



MY LIFE IS AT THE MERCY OF ANYONE WHO CHOOSES TO ANNOY AND TEASE ME.

Sir John Hunter
18th century British surgeon
He suffered angina pectoris!

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Aida Seif El Dawla

I N T R O D U C T I O N:

No organ in the human body has attracted the attention and stimulated the fantasy of so many a man as the heart. Philosophers, writers, poets, physicians, religious men and laypeople, for all the heart was a symbol of what seemed to be the central core of emotion, temperament, thought, creativity, conscience, illness, life and death. In every language the heart is the organ of love and hatred :to love or hate from all one's heart; to love or hate from the depth of one's heart; to be kind hearted; to be blackhearted; to feel heavy hearted..etc. The heart is a thermometer for emotions: it beats faster with excitment, hope, fear, rage and joy. And the most relevant of all: it beats as long as we are alive and when it stops we die.

As far back in history as 1600 B.C. the greatest Egyptian Medical Document, the Eber Papyrus, translated by Ebbell in 1937 and known also as the Book of the Heart , reveals one of the earliest documents in history relating the heart to the psyche. In Ebbell's translation of the papyrus, heart and mind seemed to have meant the same thing. Depression, dementia, retardation, thought disorders, negativism and subacute delirious states were described in 14 perscriptions ascribed to various descriptions of the heart: vascular, purulency, faecal matter, poison...etc.(Okasha, 1978).

Since then, and despite the great advancement in medicine, and understanding the functions of the body, and realizing that it is the brain rather than the heart that carries within it the as yet unreplaceable pacemaker of life, the fear of a stopping heart remains till now to be the ever empending threat of death.

REVIEW OF LITERATURE

ISCHEMIC HEART DISEASE

INCIDENCE:

While rare at the turn of the century, a rapid increase in the incidence of ischemic heart disease (IHD) has occurred in most industrial countries since World War II. Now, it is responsible for one third of all deaths in Western societies (Chesney et al., 1981).

Clayton et al. (1977) report arteriosclerotic heart disease to be one of the major causes of death and disability, especially in males of 45 to 65 years. In 1950 it represented about 20% of the total mortality rate in the U.K., while by 1973 this had risen to 30 - 40 %. Clayton et al.(1977) do not consider this a relative change due to the decline of other causes of death, but also an absolute change in the majority of nations.

Sokolov et al. (1983) point to the special attention that has been given to the cardiovascular system in studies dealing with stressor effects on physiological systems, because - they note - apart from the fact that cardiovascular pathology has been the most common cause of death in modern conditions, this system is the finest effector instrument closely linked with the regulatory mechanisms of the cortex and the subcortical structures.

What is striking, however, is that this trend varies from one country to the other for reasons not yet fully apparent (Davies,1979).

With this increase, coronary heart disease (CHD) received the attention of the medical and scientific communities and has stimulated research regarding its risk factors and clinical expression. (Chesney et al., 1981).

PATHOPHYSIOLOGY OF IHD:

IHD is a disease spectrum of diverse aetiology, with the common factor being an imbalance between myocardial oxygen supply and demand. This imbalance is usually related to either an absolute reduction in coronary blood flow or an inability to increase coronary blood flow relative to the needs of the heart, and is most often due to atherosclerotic obstruction of large coronary arteries (Harrison,1983).

CORONARY CIRCULATION:

Under basal conditions, the heart extracts a high and relatively fixed percentage of oxygen from the coronary

arterial blood. Since increased oxygen extraction is thus not possible, augmented myocardial oxygen demands must be met by increases in coronary blood flow. Blood flow in the coronary bed, as in other vascular beds, is related directly to coronary perfusion pressure and inversely to vascular resistance. Since arterial pressure does not increase sufficiently or at all during stress, such as exercise, variation in the resistance of the vessels of the coronary bed is the mechanism by which coronary flow and thus oxygen supply are regulated. The immense dilatory reserve of the normal coronary circulation is proved by the observation that patients without coronary disease may tolerate prolonged periods of hypotension or severe anaemia without sustaining ischaemic myocardial damage (Harrison,1983).

PATHOLOGY OF IHD:

In CHD - also called obliterative atherosclerosis of the coronary arteries - smooth muscle cell proliferation combined with a disorder of lipid metabolism is thought to be responsible for the localized subintimal accumulation of fatty and fibrous tissue that progressively obstruct the epicardial portions of the coronary arteries and their main branches. A deficiency or imbalance in certain tissue enzymes such as prostaglandins has been proposed as the mechanism that allows adherence of platlets and accelerates the atherosclerotic process, a subject under intensive investigation.

Although it has been considered for years that total serum cholesterol and low density lipoproteins are the most important components of lipids with respect to atherosclerosis, some recent data suggest that high density lipoproteins may be equally important. There seems to be an inverse relationship between the concentration of subclasses of high density lipoproteins and the incidence of clinical coronary events, so a high concentration is protective (Sokolov,1985).

EFFECTS OF ISCHAEMIA:

The state of inadequate myocardial oxygenation induced by the atherosclerotic process results in abnormalities of the biochemical, electrical and mechanical function of the heart. One biochemical response to ischaemia which is used to evaluate the adequacy of myocardial perfusion relates to the aerobic metabolism of the normal heart; glucose and glycogen are oxidized completely to carbondioxide and water and intermediary metabolites of carbohydrate breakdown, such as lactate, neither accumulate in the myocardium nor appear in coronary sinus blood. Under conditions of impaired oxygenation, anaerobic patterns of metabolism are utilized and lactate is formed and is eventually released into

coronary sinus blood.

MORTALITY RATE:

The average mortality rate following entry into the symptomatic phase of IHD is - according to Harrison - 4% per year, irrespective of whether the initial event is the development of angina pectoris or myocardial infarction, from which the patient has recovered.

ANGINA PECTORIS (AP):

AP is a clinical syndrome, resulting from transient myocardial ischaemia due to arteriosclerotic heart disease, but in rare instances it may occur in the absence of significant disease of the coronary arteries as a result of coronary spasm, severe aortic stenosis or insufficiency, syphilitic aortitis, increased metabolic demands as in hyperthyroidism or after thyroid therapy, marked anaemia or paroxysmal tachycardias with rapid ventricular rates. The underlying mechanism is a discrepancy between the myocardial demands for oxygen and the amount delivered through the coronary arteries, either by increased demand or decreased coronary flow (Sokolov,1985).

Approximately 4/5 of all patients with angina pectoris are men, and an even larger fraction of these younger than 50 years of age are men.The typical patient is in his fifties or early sixties and seeks medical advise because of chest discomfort. The most important diagnostic feature of angina pectoris is its relation to exertion or emotion or a heavy meal and its relief by rest. The discomfort comes on during physical or emotional stress, anger, fright, hurrying or sexual activity (Harrison,1983).

MYOCARDIAL INFARCTION (MI):

This is a clinical syndrome due to ischaemic necrosis secondary to occlusion of a coronary artery by thrombus or subintimal haemorrhage at the site of atheromatous narrowing. Less often, complete occlusion by intimal plaques or by haemorrhage into a plaque is responsible. Infarction may occur in the absence of complete occlusion , if coronary blood flow is temporarily reduced, as in postoperative or traumatic shock, gastrointestinal bleeding or hypotension due to any cause, or dehydration. Coronary vasoconstriction or spasm may be intense and prolonged and has been proved to result in myocardial infarction in a small number of cases (Harrison,1983).

Like in angina, men are more often affected than women

by an overall ratio of 4:1; before age 40 the ratio is 8:1, and beyond age 70 it is 1:1. In men the peak incidence of clinical manifestations is at age 50 - 60, in women at age 60 - 70 (Sokolov,1985).

RISK FACTORS:

Risk factors (established by prospective studies) that predispose to the development of IHD include age, genetic predisposition, hypercholesterolaemia, arterial hypertension, diabetes mellitus, hypertriglyceridaemia and cigarette smoking. Other factors include obesity and possibly physical fitness and personality type (Sokolov,1985).

Among all those risk factors smoking and hypercholesterolaemia have been repeatedly advocated as strong contributors to the causation of IHD.

1. Hypercholesterolaemia:

Those who believe that a high intake of saturated fat is a major determinant of CHD point to the composition of the atheromatous lesions and to the strong correlation in international comparisons between fat intake and CHD mortality. A series of epidemiological studies from the 1950 - 60s show that people with elevated serum cholesterol have an increased risk of CHD, with several experiments indicating that a reduction in the daily intake of dietary lipids will reduce serum cholesterol (Kringlen,1986).

On the basis of historical statistical data, Michaels (1966) did not find any evidence for the assumption that the British population had become any fatter in recent years, nor did he observe any marked alteration in fat consumption over the years.

Walker (1977) has also noted that there has been little change in the US diet between 1909 and 1965, despite the increased incidence of IHD.

Kringlen (1986) finally notes that there are much higher rates of CHD in the densely populated and industrialized regions of the US and the UK, compared to the farm belts that are often characterized by high habitual consumption of dairy products and other saturated fats.

Oliver (1981) although agreeing to a moderately good correlation between diet and CHD amongst populations in different parts of the world, denies the presence of any sound correlations between habitual fat intake and either the blood cholesterol or the incidence of CHD itself and CHD within the same cultural community or for individuals.

There are also reports that directly negate the diet hypothesis.Guberan (1979) observed a considerable decrease,namely 13% in males and 40% in females, in the total mortality from CHD and hypertension in Switzerland from 1951 to 1976, although the per caput consumption of animal fat rose by 20 %.

2. Smoking:

In most studies from the United States and Western Europe, there is a statistical association between cigarette smoking and CHD. Kringlen (1986) agrees that cigarette smoking has declined among men in the United States during the last decade, however, cigarette smoking among women has not decreased and still women have experienced the most marked decline in CHD mortality in the USA over the last 15 years. In fact, the author points to the particular increase in heavy smokers among black women, who have had the most dramatic decline in CHD mortality rate.

Adherents of the smoking hypothesis for CHD have also emphasized that persons who have stopped smoking have a substantially lower mortality from CHD than persons who have continued to smoke (Gordon et al., 1974). However, Friedman et al. (1979) advocate that ex-smokers are not a representative sample of smokers. They followed up more than 25000 subjects, males and females and were able to show that ex-smokers are not a representative sample of smokers with regard to CHD-related traits. Smokers who later stopped smoking had a lower CHD risk profile than smokers who continued smoking. They argued that ex-smokers are self-selected to begin with and are at lower CHD risk at baseline.

Seltzer (1980) went so far as to state:' For the present it is reasonable to believe that stopping smoking does not reduce the risk of CHD and there is no established proof that cigarette smoking is causally related to coronary heart disease.' Even if this sounds too harsh a statement, yet it serves the questioning of the overused, oversimplified smoking hypothesis.

SOCIODEMOGRAPHIC DATA:

Gomez (1987) refers to a coronary attack as the 'Western Way of Death'. More patients are surviving myocardial infarction, which to his mind, presents physicians and psychiatrists with the challenge of reversing a life pattern that leads to disaster, often at the peak time in a man's progress, when his employer and his family value him most.

Several authors have emphasized that cultural, social, psychological and behavioural factors were undoubtedly the main determinants of all standard CHD factors, which can be influenced e.g. cigarette smoking, dietary habits, physical inactivity..etc.

Culture

An investigation invoking cultural factors sought to explain the gradient incidence of coronary artery disease in Japanese males - lowest in Japan, highest in Japanese domiciled in California, and intermediate in those living in Hawaii (Marmot and Syme, 1976). Differences of diet, serum cholesterol level, blood pressure, and cigarette smoking apparently accounted for only part of the variation. Accordingly these authors classified a large sample of Japanese men living in California in terms of the degree to which they retained their traditional Japanese culture. Those most acculturated to the West had three to five times higher prevalence of coronary artery disease than the least acculturated. This seemed to support the suggestion that in a stable society members enjoy the benefit of fellowship in a close-knit group and may be relatively protected against the forms of social strain that lead to coronary artery disease. The same kind of comparison has been suggested as between Greece, Italy and Yugoslavia, with their cohesive family and village groups, and the countries of Northwestern Europe , where society is more unstable and individualistic (Davies,1979).

This evidence strongly indicates that a combination of habitual patterns of social interaction , specific traumatic experiences , and a relative lack of social relationships of a supportive kind can trigger or contribute to physiological processes leading to heart disease.

Smoking

As previously mentioned, smoking per se has been frequently advocated as a risk factor on its own right in IHD. In addition to its being a biological risk factor, some authors regard it as one of the risky behaviours brought about by our modern culture and thus considered as a cultural behaviour.

Grossarth et al. (1983) started a prospective psychometric investigation in 1965-66 with 1 353 relatively old inhabitants of a village in Yugoslavia, investigating smoking as a risk factor for lung cancer and cardiac infarct as mediated by psychosocial variables. The main results were:

1. The relevance of smoking was reduced, but not eliminated

by introducing psychosocial control variables, suggesting that the latter have direct influences both on smoking and on the diseases.

2. The relevance of smoking interacted very strongly with psychsocial background conditions, it was nearly reduced to zero, when the latter were favourable and was correspondingly high when they were unfavourable.

Jenkins et al. (1983), on the other hand identified cigarette smoking as one of the main predictors of angina in a psychological and behavioural study of 2204 men awaiting coronary artery by-pass graft surgery.

Level of Education

Several studies failed to detect a correlation between level of education and CHD (Hinkle et al., 1968; Rosenman et al., 1975). Angina pectoris was more prevalent in educated people , whereas AMI was more frequent in people with low education (Shekelle et al., 1969). Incongruity between occupation and education was suggested to be among the factors on which the effect of occupation on the risk of CHD depends.

Rather interesting were the findings reported in this context among participants in the Framingham Heart Study (FHS) (Haynes et al., 1978) and the Western Collaborative Group Study (WCGS) (Rosenman et al., 1975). In the FHS men were found to have a significantly higher incidence of IHD if they were married to wives with 13 or more years of education, compared to men with wives who had less education.In the WCGS population, on the other hand, the association of the husband's incidence of IHD with the wife's educational level was mediated by the husband's behaviour pattern. Thus it was found that type A men married to wives with 13 or more years of education were at much higher risk for IHD than their type B counterparts.

Another study has reported on findings from a sample of 1698 spouse pairs aged 45 to 79 years, who were followed up prospectively for a period of 9 years (Suarez and Barrett-Connor, 1984). The risk of IHD in husbands was again found to be greater for men whose wives had higher levels of education, and was greatest among men with the least education, who were married to women with the highest education.

(COMMENT: It would be funny to conclude from that, that a preventive means against IHD would be for men to avoid marrying highly educated women, or that women in order to maintain their husband's health should abstain from educating themselves!!)