ACCEPTANCE of the Platonic doctrine that all bodily ills proceed from the spirit led medical authors of antiquity, the Middle Ages and the Renaissance to ascribe cardiac diseases to emotional disorders almost exclusively. Postmortem studies, impressively collected by various authors, notably Zimmerman, appeared to confirm this general idea and to support such concepts as “dying of a broken heart,” where cardiac rupture was found in grief, or “hardness of heart,” in which pericarditis was found in individuals notorious for cruelty. In the early nineteenth century, Corvisart and his followers, Burns, Testa and Kreyssing, likewise believed emotion to be the most important cause of heart disease. The cause of cardiac and aortic dilatations was confidently stated to be the driving inward of the bodily humors during psychic stress. Sudden development of dropsy, cardiac hypertrophy and aortic aneurysms immediately following a single severe emotional upset was often described. Corvisart and Testa stated that heart disease increased markedly during and after the French Revolution and attributed this phenomenon to the disorder of the times. Bertin, a decade later, held that seeming increases in cardiac disease were due only to its being recognized more certainly. This controversy of over a century ago sounds familiar in the present. In spite of increase of knowledge of pathologic anatomy in the nineteenth century, etiologies of cardiovascular diseases remained obscure. The tendency to ascribe conditions of unknown etiology to emotional disturbances persisted, as exemplified by one authority of the 1870’s, who stated with assurance “It is not surprising that in the present day, when the worry of life and strain on the feelings in all ways are so vastly intensified, that there should be strong evidence to show the increase of cardiac affections”; Balfour expressed similar ideas 25 years later and today some cardiovascular diseases still are considered to be due to psychic disorder.

The problem that plagues the cardiologist arises from the fact that he sees evidence daily of the influence of emotion on the circulation but has at hand no extensive body of physiologic data that might illuminate his clinical observations. For the most part, the psychosomatic studies of the circulation merely emphasize what the competent internist appreciates, namely, that emotion may cause cardiovascular symptoms. The present discussion will analyze available physiologic studies and will attempt to relate them to clinical phenomena. Reference will be made only to the heart and peripheral blood vessels; consideration of renal and gastrointestinal circulation will be omitted.

Peripheral Vascular System

Physicians of antiquity believed that specific emotions caused specific changes in the pulse; this belief has persisted, in various forms, during the past 25 centuries. For instance, early in the present century, Wundt, in his Gefühls-theorie, classified feelings rigidly in three pairs, i.e., pleasure-pain, tension-relaxation, and excitement-calm, and his followers claimed to have

From the Laboratory of Clinical Physiology, McLean Hospital, Waverley, Mass., and the Department of Medicine, Harvard Medical School, Boston, Mass.

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observed specific changes in the pulse wave corresponding with each. This work, reviewed by Leshke in 1914, formed a seemingly convincing body of evidence which now is regarded as invalid. Recent work on specificity in peripheral vascular reactions to emotion evokes doubt because of skepticism regarding the authors' ability to characterize exactly the feelings experienced by their subjects during short experiments and because of the absence of suitable control periods.

Many physiologic studies of general effects of psychic factors on peripheral circulation are available. Cutaneous vasoconstriction in the hands and feet during emotion has been amply demonstrated; this change manifests itself by cooling of the parts and, because of stasis, excessive deoxygenation of the capillary blood. In ordinary circumstances, changes in the hands are due both to arteriolar and venular constriction, whereas those in the forearm are largely venular in origin; on the arterial side only the terminal cutaneous arteries, and not the radial, metacarpal and digital arteries, usually are involved. In extremely severe emotion, however, spasm of the radial artery may be detected. As a consequence of spasm of arterioles and venules, changes in cutaneous capillaries occur; these are visible on microscopic examination of the vessels of the fingernail folds. All the changes described are minimized by vasodilating agents such as alcohol, carbon dioxide or warmth.

The immediate vasoconstriction of emotion is effected via the sympathetic nervous system and a sympathectomized extremity does not show it. The hypothalamus contains centers governing generalized vasoconstriction and vasodilatation but the occurrence in emotional states of vasomotor changes limited commonly to the acral areas raises the possibility that the impulses arise in the cortex. On the other hand, it is possible that vasomotor changes in general are most marked in the acral regions so that weak discharges from the hypothalamic centers cause changes which are detectable only in the extremities. At any rate, the importance of cortical impulses in vasomotor phenomena is now recognized; this will be discussed below, in relation to hypertension.

Although most studies of peripheral flow emphasize unpleasant emotions in causing the changes observed, there are data which show that strong pleasurable emotions may give rise to identical peripheral vascular phenomena. The thesis that subjects who show marked vasomotor changes with pleasurable emotions are expressing subconscious guilt consequent to experiencing such feelings is not supported by psychologic studies. In addition, as Lombard showed a century ago, and others have corroborated since, vasoconstriction in the extremities occurs also during concentration not obviously emotionally colored.

Peripheral vasomotor phenomena of emotion do not parallel regularly the facial expression of emotion and even less so the verbal description of it. Whether conscious or unconscious feelings are the more important is unknown; however, emotional vasoconstriction disappears during sleep, when, according to psychoanalytic thinking, unconscious emotions are at their height.

Patients with persistent and marked emotional vasoconstriction exhibit coldness of the extremities and are intolerant to cold temperatures. Confusion may arise with the symptoms of hypothyroidism or anemia so that patients may needlessly receive thyroid or antianemic medications.

The vasoconstrictor effects of emotion may aggravate manifestations of such peripheral vascular diseases as arteriosclerosis, thromboangiitis obliterans, post-traumatic causalgia and Raynaud's disease. In addition, men with a previous history of excessive vasomotor reaction to emotion are markedly susceptible to frost-bite and trench foot. Rheumatoid arthritis of the hands and feet is also demonstrably influenced by emotions and excessive vasoconstriction in this disease actually has been demonstrated.

Fragmentary observations suggest that subjects with unstable peripheral vascular systems develop conditioned reflexes involving this system more easily than do others; this may suggest a possible mechanism whereby the peripheral vascular system becomes the one which expresses emotion in some individuals. In addition, emotionally unstable individuals exhibit excessive acral vasoconstriction as a
Consequence of concentration itself, which suggests that vasomotor as well as cerebral emotional mechanisms may be abnormal.

Physiologic phenomena underlying blushing and blanching of the face and neck have not been studied; here is a promising field for observation on specificity of emotional responses, for blanching with fear and blushing with embarrassment are well known. On the other hand, various feelings may cause either pallor or flushing in different individuals or in the same individuals at different times. The belief that easy blushing is evidence of a marked paranoid personality has no support in extensive observation, and is a conclusion based on an a priori interpretation of the symptom. Vasodilatation consequent to fever, external heat, imbibing of alcohol, or emotional upset is known to be followed by urticaria in some individuals. Acetylcholine is released locally during vasodilatation and it is well known that acetylcholine is a whealing agent. Whether it acts as such, or through the liberation of histamine, is not established, however. Development of hives under these circumstances in some individuals and not in others is not understood.

**Cardiac Rate**

Marked changes in heart rate occur in relation to emotion. Poor correlation between changes in pulse rate and the facial or verbal expression of emotion is regularly found. Variation in rate from minute to minute may be marked. As a rule the rate is accelerated in emotional reactions but at times it may be abnormally slow. In some instances the sinus bradycardia is part of a syncopal reaction, while in others the bradycardia is not the precursor of any such reaction. The cause of the rapid and marked variation in rate is not known, although at times it may be due to sinus arrhythmia correlated with hyperventilation.

Changes in pulse rate during strong emotion are of the same magnitude in normal and in neurotic subjects. Neurotic subjects, however, experience more severe emotional reactions than normal subjects during mild stresses. The neurotic may, moreover, be more aware of emotional tachycardia than the non-neurotic subject. The pulse rate in exercise in neurotic subjects will be discussed below, in relation to neurocirculatory asthenia.

The relation of emotional tachycardia to conscious appreciation of emotions has not been defined. Emotional tachycardia subsides during sleep, a fact used in differentiating anxiety from thyrotoxicosis. On the other hand, during sleep a noise, insufficient to awaken the sleeper or make him exhibit overt evidence of startle, may increase the pulse rate by a third. This phenomenon appears to vary with the depth of unconsciousness.

It is generally presumed that tachycardia of emotion is related to sympathetic hyperactivity, or production of excess epinephrine. However, norepinephrine, which does not increase pulse rate but merely raises the blood pressure, is probably liberated from the adrenal medulla together with epinephrine. An additional complication arises from the fact that acetylcholine in small amount causes tachycardia and in larger amount slows the pulse. It is known also that the brain contains areas which cause tachycardia when stimulated. It is evident that the precise cause of emotional tachycardia is not known.

Rapid heart rates in neurosis may lead to mistaken diagnoses of heart disease or thyrotoxicosis; neurotic patients who complain of tachycardia may be given antithyroid drugs or digitalis and may become disabled owing to bad advice regarding activity. It should be noted that palpitation in neurosis need not signify occurrence of a rapid heart rate; palpitation may be experienced with normal or slow heart rates if sudden rises in blood pressure occur, if the rhythm is irregular, or, on occasion, with no change in the circulatory mechanisms.

**Cardiac Rhythm**

Routine examination of neurotic subjects may not reveal the occurrence of any arrhythmia other than sinus arrhythmia, but every cardiologist has seen occasional instances in which auricular flutter, fibrillation or tachycardia, frequent auricular, nodal or ventricular premature beats, the Wolff-Parkinson-White syndrome, or minor degrees of heart block.
appear recurrently in emotionally unstable individuals without heart disease. At times the arrhythmia apparently develops in relation to some unusual emotional stress, but on other occasions similar stresses do not cause it; conversely, the arrhythmia may appear in the absence of overt emotional upset. The same fact holds in patients with established diagnoses of organic heart disease; partial heart block may increase in degree, or premature beats may become more numerous during emotional upset. The arrhythmias seen in relation to emotion are usually those produced by hyperactivity of the vagus nerve, i.e., arrhythmias involving the S-A node, the auricles and the A-V node; in the case of ventricular premature beats the effect of circulating epinephrine, or of sympathetic stimulation must be considered.

**Electrocardiogram**

Papers claiming that abnormalities of QRS complex or of T waves indicative of altered myocardial function occur in neuroses or are produced in normal subjects by emotion are unconvincing; changes shown appear to be due to variations in cardiac rate and in position of the heart. Examination made here of several hundred electrocardiograms in instances of emotional disorder corroborates the findings of others who showed no deviation from the normal.

**Cardiac Output and Work:**

**Circulation Time**

That measurement of cardiac output in tense or frightened subjects yields values that are high relative to the basal has long been known to physiologists. The earlier work of Grollman and the more recent studies of Stead and co-workers and Hickam and associates, all employing accurate methods, showed that emotion may increase cardiac output by two-thirds, and occasionally more; the cardiac output may vary markedly from minute to minute. These observations showed no relation between type of emotion and change in cardiac output. Hickam and co-workers and Wolff and Wolff, using the ballistocardiograph, found greater increases in cardiac output in many patients during emotional stresses; the latter authors claimed that some types of emotion caused decreases in the output of the heart. The ballistocardiograph is not accurate when the pulse rate changes markedly and rapidly. Discrepancies between the work of Wolff and Wolff and that of other authors may be due to the method used; however, the observations of Hickam and associates, who used the same method, do not support the concept of relation between type of emotion and circulatory change. Rises in cardiac output owing to emotion are greater than increases in oxygen consumption which occur at the same time; the cardiovascular changes resemble those which follow injection of small amounts of epinephrine. Cardiac work is proportionately increased.

Circulation time is usually accelerated in emotional tension although marked emotionally-induced venoconstriction in the forearm may cause some slowing in the observed time and so give no true indication of the increase in cardiac output.

Change in cardiac output during anxiety in patients with heart disease but without failure are similar to those in normal subjects; in congestive failure, however, little or no increase in cardiac output (and work) occurs during anxiety although the pulse rate rises. The increase in metabolism induced by emotion in these circumstances results in further deoxygenation of capillary blood and exacerbation of the anoxia already present.

Stevenson and his co-workers studied the effects of exercise on cardiac output in anxiety and found the rise to reach higher absolute values and also in some instances to be greater in amount than when the subjects exercised while relaxed. Makinson found the increase to be normal but more prolonged in neurosis. However, the use of a method of low accuracy makes acceptance of these data only tentative. Nevertheless a metabolic effect of emotion, i.e., failure to oxidize carbohydrate completely, as described below, suggests that the cost of work is higher during stress and therefore the circulatory response in exercise should be greater than in normal circumstances.

Increases in resting cardiac output by about
half owing to anxiety is not significant in causing emotional dyspnea during exertion except when the effort attempted is maximal; the maximal possible cardiac output is not approached during moderate physical activity and the patient does not need the cardiac reserve lost as a result of the increase in resting cardiac output occasioned by emotion. In patients with serious organic heart disease but no failure, the increases in cardiac output during strong emotion, may, if long continued, act in a manner like that of thyrotoxicosis, anemia, fever and other conditions which strain the heart and precipitate decompensation.

**Cardiac Discomfort: Sudden Death**

A variety of sensations seemingly localized about the heart occur during emotional reactions. The clutching discomfort occasioned by fear is well known; it differs from angina pectoris, according to those who have experienced both. The heavy sensation associated with sadness is also commonly experienced. The mechanism of these sensations is completely unknown.

Another type of discomfort, commonly seen in neurosis and more particularly in neurocirculatory asthenia, is the typical persistent, sticking pain close to the left breast. Its occurrence does not depend on changes in cardiac rate or rhythm, although in occasional patients it occurs only with premature beats or with palpitation. The mechanism of its production and the pathways of its transmission are not known. Commonly it is associated with persistent tenderness over the left chest although each of the two symptoms may occur without the other. Anxiety pain is occasionally confused with angina pectoris, leading to induction of a needless state of invalidism in the patient; this occurrence is a reflection on the physician who makes the error, for anxiety pain and anginal pain have only the most superficial resemblance. The confusion between anxiety pain and angina pectoris vitiates all of Dunbar’s work on the latter disorder.

Attacks of true angina pectoris may be precipitated by “disturbance of the mind,” in the words of Herberden. In the presence of coronary arterial insufficiency, increases in cardiac work owing to emotion may cause anginal pain or may cause exertion more readily to induce it. John Hunter, whose clinical history and postmortem findings contributed to the modern theory of angina pectoris, often stated, “My life is at the mercy of any scoundrel who chooses to put me in a passion.” Hunter's death occurred during a dispute with the Governors of St. George’s Hospital regarding the exclusion of students whom he considered to be victims of a prejudice against the Scotch. The pain of angina pectoris occurs when the work of the heart is increased to a degree which cannot be paralleled by increased flow through diseased coronary arteries; the importance ascribed by Wolff and Wolff to their findings of a lowered cardiac output in one patient during one attack of angina induced by emotion may be criticized in view of the fragmentary data, obtained by a method of low accuracy.

Another possible mechanism for the precipitation of anginal pain during emotional stress has not been studied adequately. That cooling of the skin of the hands favors the occurrence of anginal attacks in patients with the syndrome has been established; the fact that strong emotion usually causes cooling of the skin over the acral areas may therefore be significant.

Discussion of mechanisms for producing anginal attacks applies also to myocardial infarction. Sudden death during emotional strain has been recognized for at least 25 centuries as a common occurrence in the middle-aged or elderly. Although ventricular fibrillation may account for some, in many such instances myocardial infarction is the cause for this accident; it must not be concluded, however, that strong emotion is the sole or the most important cause of myocardial infarction. That strong pleasurable emotions may be as dangerous in causing sudden death as grief, rage or despair is important in relation to advising patients regarding their daily activities. Benjamin Rush described how “...the door-keeper of Congress, an aged man, died suddenly, immediately after hearing
of the capture of Lord Cornwallis’s army. His death was universally ascribed to a violent emotion of political joy. This species of joy appears to be one of the strongest emotions that can agitate the human mind."

**Syncope**

A fundamental phenomenon in syncope is strong vagal discharge causing marked sinus bradycardia; occasionally minor degrees of heart block and ventricular escape may occur. Total peripheral resistance decreases markedly and blood pressure falls; there is also evidence of generalized loss of venous tone and, to judge from the cutaneous vasoconstriction which develops, pooling of blood in the viscera. The cutaneous vasoconstriction, manifested by disappearance of visible capillaries in the fingernail fold and by smallness of the superficial veins of the arm, may result from a sympathetic discharge elicited by the falling blood pressure. Cardiac output measured with the patient recumbent is normal, but in the upright position it is markedly decreased. In animals stimulation of areas in the frontal lobes causes hypotension and bradycardia; the relation of these findings to emotional syncope may be important.

Tendencies to recurrent fainting in neurotic patients may be ameliorated by the drugs effective against increased vagal tone, i.e., atropine, ephedrine and the like.

**Hypertension**

Much has been written on the importance of emotion in hypertension and opinions vary. Most cardiologists believe that psychic influences are among those which most strongly influence the course of the disease; on the other hand, some internists and most psychiatrists feel that emotional factors actually cause essential hypertension. The first of these opinions is well established but the second is not securely founded in observation. The present discussion will not attempt to resolve this issue but only to point out certain factors that must be borne in mind in considering it. Hypertension may cause only mild symptoms and accordingly introverted patients are most aware of these minor complaints; high incidence of neurosis in patients with complaints of hypertension therefore results. Also, since emotional upsets do aggravate hypertension, the incidence of neurosis is high in patients with early or moderate hypertension. In addition, stresses of various types cause more marked and more persistent increases in blood pressure in neurotics than in normal subjects. Definition of physiologic mechanisms responsible for exacerbation of hypertension is difficult in the present state of knowledge, a fact that has not deterred many authors from doing so. There are observations which show that anxiety in normal or neurotic subjects is accompanied by decreased peripheral resistance; the diastolic blood pressure is elevated only occasionally and remains unchanged or falls for the most part. The systolic pressure rises parallel with increases in cardiac output. These changes resemble effects of injection of small amounts of epinephrine. However, there is good evidence that shows that in essential hypertension the peripheral resistance is increased, both systolic and diastolic pressures rising, while the cardiac output remains normal. It is evident that the cardiovascular physiology of essential hypertension differs from that of anxiety. Nevertheless it is true that anxiety further elevates systolic pressure in hypertensive subjects and increases cardiac work greatly. The fact that norepinephrine is also liberated during stimulation of the adrenal medulla is also important. It acts entirely on peripheral blood vessels, increasing peripheral resistance and not influencing cardiac output directly, thereby producing changes resembling those of essential hypertension. To assume that one adrenal medullary hormone alone, epinephrine, is liberated during stress in normotensive subjects while the other, norepinephrine, is liberated alone in hypertensive subjects is not reasonable; however, work should be done to ascertain whether stress of short duration results largely in epinephrine effects, while stress of long duration causes predominately norepinephrine effects, as work of Bulbring and Burns suggests may be possible.

The cold-pressor test in normotensive patients with anxiety causes rises in blood
pressure which at most are only slightly greater than normal\(^{25}\) and are much smaller than those found in hypertensive patients or some of their normotensive siblings. This finding again suggests that anxiety is not fundamental to essential hypertension.

Another humoral mechanism has been invoked to explain how anxiety may cause lasting hypertension. Decrease in renal blood flow, consequent to renal vasoconstriction, may occur in emotional stress; it has been suggested that renal ischemia so induced may give rise to elaboration of renin, and so cause elevation of both systolic and diastolic pressures without increasing cardiac output. Against this hypothesis is the fact that renin is not found in the blood in essential hypertension. In addition, not all patients with essential hypertension exhibit the changes in renal vascular dynamics required by this concept.

Excellent work in animals and more recently in man\(^{26}\) has extended knowledge bearing on nervous pathways which carry vasoconstrictor impulses from the brain. Existence of vasoconstrictor centers in the hypothalamus has long been known; recently it has been shown that stimulation of the frontal cortex, especially that of its posterior orbital surface, elevates blood pressure. The location of this area in the general region in which is situated cortical representation of visceral changes and of somatic movements associated with expression of emotion is highly significant.

Some patients with essential hypertension exhibit significant changes in blood pressure in relation to variations in the intake of salt. The fact that a tendency toward salt retention may possibly exist during emotional stress, as discussed below, makes it necessary to consider this mechanism as possibly involved in relations between emotion and hypertension in some patients.

In spite of uncertainties regarding the manner in which emotion exacerbates hypertension, clinical observations nevertheless puts the relationship between the two on a sound basis; observation thus far, however, lends no support to the concept that emotion causes essential hypertension. Statements that certain types of personality or emotional conflict are the cause of the disorder are ill-founded and should be received with skepticism.

**NEUROCIRCULATORY ASTHENIA**

The importance of neurocirculatory asthenia has been rediscovered during every war in the course of the last century; there is no evidence that it is increasing in frequency. Its relation to neurosis is indicated not only by the nature of its symptoms, but also by the fact that the physiologic changes induced by exercise in patients with various types of neurosis are similar to those considered characteristic of neurocirculatory asthenia. A physiologic difference between these two groups of patients lies in the fact that on the average changes are greater in neurocirculatory asthenia; however, there is much overlapping of the two groups.\(^{27}\)

In addition to pain and precordial tenderness discussed above, symptoms pertinent to the present discussion consist in diminution in tolerance to exercise owing to abnormally severe palpitation, dyspnea, or fatigue.

In emotionally disturbed subjects, exercise causes a greater rise in pulse rate than in the same subject when not upset, or in normal subjects; the pulse rate falls slowly after the exercise. The mechanism underlying this phenomenon is not known; the concept held in some quarters that hyperventilation is the cause of the tachycardia is not securely founded. The severity of the palpitation, due both to the markedly abnormal pulse rate and also to the patients' increased awareness of symptoms, results in disinclination toward exercise. If tachycardia is very marked during exercise in a neurotic subject it is possible that some impairment of circulatory function might develop owing to lessened filling of the heart due to shortening of diastole. Otherwise tachycardia is important only because of discomfort it may cause.

To consider neurocirculatory asthenia solely as a state in which the flesh is willing but the spirit weak is an error, for when patients with neurocirculatory asthenia, or indeed with
neurosis, exercise, evidence of a metabolic disorder becomes evident: 27, 28; minor degrees of this disorder may be apparent at rest also. The resting blood lactate level may be slightly elevated in some, but during exercise it rises regularly to a higher level and remains elevated longer than in normal subjects doing the same work. Oxygen consumption during strenuous work is not increased as much as in normal subjects and consequently there is a large and prolonged oxygen debt. Associated with excessive and prolonged rises in blood lactate in exercise there are corresponding increases in respiration, so that the carbon dioxide content of expired air falls and carbon dioxide may be washed out of the blood; during work the respiratory rate and minute volume increase more than in normal subjects and remain elevated for an abnormally long period after the end of exertion.

Mechanisms responsible for this metabolic phenomenon are not established but the changes observed are consistent with the effects of adrenocortical hormones known to be liberated during stress. Vital capacity and venous pressure at rest and after exercise are normal, and so no possibility of significant cardiac or pulmonary insufficiency need be considered. The exertional dyspnea of emotion is based upon impairment of lactic acid metabolism and the fact that neurotic patients are discomfited more than normal subjects by visceral symptoms. These mechanisms, important in neurosis or emotional upset in normal subjects, are all the more so in congestive failure.

Emotional dyspnea, since it does not involve stasis in the lungs or the venous system, is not associated with orthopnea, a fact useful in differential diagnosis. Patients with neurocirculatory asthenia may do large amounts of hard labor under some circumstances and need not show the excessive prostration exhibited by patients with congestive failure who attempt exertion beyond their capacities; as a rule, however, emotional dyspnea causes enough disability to force the patient into some limitation of activity. Nevertheless there is no harm in the patient's exceeding what he believes to be his capacity for exercise. Misdiagnosing the condition as myocardial insufficiency may do harm to a nervous patient if marked revision of way of life is advised.

**Some Metabolic Effects of Emotion**

Metabolic disturbances found in emotional disorders are numerous and complex; many are not pertinent to the present discussion and only three will be considered.

An extensive literature, based on work in animals, has shown that discomfort or emotional stress stimulates the hypothalamic center that regulates the function of the hypophysis and results in liberation of antidiuretic hormone. This effect is shortlived in normal animals but excretion or inactivation of this hormone is slowed in the presence of liver damage, such as exists in congestive failure. That some edematous cardiac patients retain more water than salt is recognized, and this finding suggests the importance of antidiuretic hormonal effects as a mechanism of edema formation in heart disease. Patients with congestive failure regularly exhibit retardation of water diuresis as do patients with mental disturbances also. 29 Patients with mental disorders also show abnormally small responses to the injection of diuretic drugs. 29 The above discussion regarding one possible relation between emotion and exacerbation of edema in heart disease is largely theoretic and is designed only to call attention to the problem.

Patients with emotional disorders show many evidences of increased activity of pituitary-adrenocortical mechanisms. It has been shown also that distressing thoughts are followed by physiologic evidence in normal subjects, of increased formation of adrenocortical hormones. That these hormones favor salt retention is well known and accordingly this phenomenon must be taken into account in consideration of those patients in whom diuresis with the commonly successful measures is unsuccessful.

One action of the adrenocortical hormones liberated during stress is to impair carbohydrate metabolism so as to retard removal of
lactate from the blood. This mechanism is basic in neurocirculatory asthenia, as discussed above, but also must be of importance, when present in congestive failure, in contributing to dyspnea, since the lactate metabolism is already markedly impaired in myocardial insufficiency.

COMMENT

Physiologic effects of emotion on the circulation in health and disease are many and varied. Their importance lies in the facts that (1) they may exacerbate cardiovascular diseases; (2) their manifestations may resemble those of coronary sclerosis or myocardial insufficiency and so lead to erroneous diagnosis, and (3) their occurrence may call attention to the presence of emotional disorders not previously recognized.

No comment has been made relative to the effect of disease itself in causing emotional upset which in turn acts unfavorably upon the initial condition; this is true when the condition is organic heart disease or cardiovascular neurosis. An example is afforded by Sir Walter Scott who stated, "what a detestable feeling this fluttering of the heart is... I know it is nothing organic, and that it is entirely nervous, but the sickening effects of it are dispiriting to a degree." This problem involves one of the most important aspects of the relation between patient and physician.

The problems raised by emotional disorders are probably not more numerous now than they were in the past, although they are probably different in character. Psychiatric and other medical writings of the past, from those of Burton down to those of the present, recurrently exclaim at the rapid spread of emotional disorders consequent to loosening of family ties, disintegration of morals, loss of influence of the Church and deterioration of economic security. It is startling to find that Erb, for instance, used these same explanations at the turn of the century, a period which today is nostalgically considered to have been one of stability and contentment. It is difficult to accept the pronouncements of today's Cassandras regarding trends in the incidence of emotional disorders. The importance of neurosis may seem particularly large today as a consequence of the fact that measures are developed in rapid succession for the exact diagnosis and specific treatment of many diseases; no such measures exist in the field of emotional disturbances and so, by comparison, they cause the physician more perplexity and dissatisfaction than any other group of disorders.

Except for the fact that the cardiovascular changes in startle reactions are similar in nature if not degree in various individuals, no consistency is encountered in the occurrence or character of cardiovascular phenomena which may appear in relation to environmental factors which influence the psyche. The emotional significance of the environmental factor to the individual patient determines the occurrence and in a measure the severity of the response to it. On the other hand, except in a small minority of instances where the nature of the cardiovascular response to emotional stress is evidently determined by neurotic identifications or by conditioning in the past, there is no indication as to the mechanisms which result in the appearance of each of the various types of cardiovascular change.

In spite of the lack of a body of definitive data explaining the mechanisms underlying the occurrence of emotional reactions, the reasons for their variations in degree and nature, and the manner in which they cause bodily changes, there is no lack of positive statements bearing on all of these matters in the current literature. Much of the current writing on mechanisms whereby emotional situations cause cardiovascular symptoms consists in validation by anecdote, and is no more conclusive in establishing etiology than the writings of half a century ago that regularly ascribed heart attacks to the eating of spoiled or exotic foods. Attempts to prove that specific diseases are associated with or the consequence of specific types of personality arouse skepticism because of their lack of control studies, the limited amount of data presented, or evidence in some cases that the author is ignorant of the criteria used in recognition of the disease in question. Concepts which relate specific illnesses to specific types
of emotional conflicts likewise are not convincing in that they appear to be based on superficial resemblances of one or a few of the many characteristics of the clinical syndrome to one or a few of the many characteristics of the conflict; it is not established, moreover, that conflicts are single, simple and invariable, or that they can become known or understood completely. Symbolic explanations currently encountered in the psychosomatic literature also invite skepticism. Acceptance of symbolic formulations regarding cardiac symptoms requires acceptance of one of two hypotheses: (1) that the heart is a thinking organ, a concept established in the minds of the ancients but not seriously considered in recent times since Virey stated more than a century ago that the heart originates thoughts and sends them to the brain via the vagi, or, (2) that specific changes in cardiovascular function in emotion always are willfully selected (consciously or unconsciously) by the patient, a concept that has no basis in substantial evidence. Similarly, attempts to explain symptoms as the result of vagotonic or sympathicotonic trends in patients on the basis of reactions of the circulation to epinephrine, atropine, pilocarpine and to various maneuvers involving autonomic reflexes, are not grounded in acceptable theory and are vitiated by inadequate observation and circular reasoning.

John Hunter wrote "There is not a natural action in the body, whether voluntary or involuntary, that may not be influenced by the peculiar state of the mind at the time." It may be taken for granted that the course of any illness can be influenced by emotional factors, and accordingly the physician must seek evidence of their presence in every case. The lack of adequate data for systematizing information regarding reactions of patients to emotional factors makes it essential for physicians to understand in general what these factors might be and to learn as much as possible about the life and personality of each patient in particular. Evaluation of the significance of emotional factors and their treatment will test his every art, for there is no skill that can be learned quickly and precisely in these matters. Although the barbiturates and other drugs are helpful at intervals, they are less so, and at times may be harmful, in the long run. Circulatory disorders consequent to severe neurosis cannot, as a rule, be alleviated to any great degree or for any length of time without psychiatric treatment. On the other hand, the milder emotional disturbances aroused in normal subjects by ordinary vicissitudes of everyday life, and the somewhat more serious ones precipitated by serious cardiac illness, can be handled satisfactorily, if recognized, by the artful physician.

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