SUMMARY OF CURRENT THERAPY

The Treatment of “Impending Infarction”
(Premonitory Phase of Coronary Occlusion)

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Acute coronary occlusion with myocardial infarction is the most serious phase of coronary heart disease (“ischemic” or “occlusive” heart disease). In turn, coronary heart disease is the major cause of morbidity and death among civilized people. It is therefore of vital importance to recognize, as early as possible, any symptoms suggestive of impending infarction, so that therapy can be instituted at once in the attempt to ward off the infarction, or limit its extent.

It has been fairly definitely established that premonitory symptoms precede approximately 50 per cent of attacks of acute coronary occlusion (transmural infarction), and acute coronary insufficiency without occlusion (subendocardial infarction). Since such attacks occur in thousands of persons every day throughout the United States, obviously the incidence of the syndrome of impending infarction is great. It must be emphasized, however, that the converse series of events is not true. Although significant statistical data are not available, in the author’s experience only a small percentage of patients with the “preinfarction syndrome” go on to develop coronary occlusion. In the majority, the symptoms gradually subside after several weeks (or even months).

Various terms and definitions have been used to describe impending infarction. In this report I have restricted it to the syndrome in which anginal pain, previously relatively mild and intermittent, abruptly becomes aggravated and acquires new qualities. It is more severe, lasts longer, requires more nitroglycerin for relief and occurs more frequently. Whereas it used to come in only after appreciable strain, it now develops on the slightest exertion or apprehension and, most important, it occurs spontaneously at rest or wakes the patient from sleep. It may be accompanied by a feeling of impending death. Thus, there is a dramatic change in the gravity of the pain. Much less frequently, anginal pain with these serious characteristics of impending infarction appears suddenly in a patient without any previous history of angina. The pathologic basis of the premonitory phase of myocardial infarction has been only scantily documented. Subintimal hemorrhage and progressive sclerosis are probably the commonest fresh changes in the presence of severe coronary disease, resulting in further compromise of the arterial lumen.

In this paper I have only considered the foregoing type of “premonitory phase” of coronary occlusion or “impending infarction.” I have excluded from my definition of impending infarction cases with a single episode of chest pain or acute coronary insufficiency (subendocardial ischemia or infarction), even though coronary occlusion (transmural infarction) may eventuate. Some writers have included such an attack in this discussion of impending infarction. Others consider a bona fide spontaneous heart attack of acute coronary insufficiency (subendocardial infarction), with RS-T depressions and T wave inversions in the electrocardiogram, as a possible precursor of coronary occlusion. Again, philosophically and epidemiologically, one could consider anyone with coro-
nary heart disease, even if "silent," a candidate for coronary occlusion (myocardial infarction). I repeat, however, this report concerns itself with the one type that I have mentioned, the type I have investigated and gathered statistics.

As soon as the syndrome of impending infarction is suspected, an electrocardiogram and the usual laboratory tests (white blood count and differential, sedimentation rate, and isoenzymes) are performed to assure that coronary thrombosis has not occurred already, as shown by Q waves and ST elevation in the ECG or markedly elevated figures for the blood tests. On the other hand, if there is persistent RS-T depression and/or T wave inversion, or if the blood test figures are slightly increased (a WBC over 10,000, a sedimentation rate above 30 or an SGO-T 40 or more) it may be accepted that subendocardial necrosis is present, the result of acute coronary insufficiency. SGO-T determinations should be repeated since, if the patient's normal figure is 10, a rise to 25 or 30 may be significant. The isoenzyme partitions may prove valuable, e.g., the lactic dehydrogenase I to V should be determined.

In treating impending infarction, I believe that there is universal agreement that rest is essential, although there is question as to how complete it should be. We do not keep the patient in bed, but permit him to sit up in his pajamas, walk about the room a bit, read, and have a radio and TV set. While I am one of those who believes that, if an occlusion is in the process of formation, nothing will prevent it, it is logical to assume that performing heavy work, driving a car, or any form of tense work may be harmful in the phase of impending infarction by increasing the degree of coronary insufficiency. In fact, it is imperative to avoid emotional strain, as well, during this period. However, some patients feel better if they are allowed to use the telephone, even for business, but this is only permitted on a small scale, so to speak and where tension is not involved. Sedatives are helpful and a narcotic may be necessary. Actually, it has been my custom to prescribe one in small doses, e.g., codeine 15 mg. (1/4 gr.) to 30 mg. (1/2 gr.), or dihydromorphinone hydrochloride (Dilaudid) 1 mg. (1/64 gr.) or 2 mg. (1/30 gr.), t. i. d. for one to two weeks, when the symptoms have begun to diminish. These drugs often break up a vicious cycle of pain and apprehension. Of course, any form of narcotic may be used. If the patient becomes constipated, he takes a laxative, or even a cathartic dose, daily.

The patient is permitted to use nitroglycerin reasonably. If there is no relief after one tablet, it may be repeated in a few minutes, but no more for at least one-half an hour. A small dose, 0.3 mg. (gr. 1/200), is used, although 0.6 mg. (gr. 1/100) may be required or if headache occurs, 0.15 mg. (gr. 1/400) or 0.075 mg. (gr. 1/800), may be effective. If the pain is not relieved by the nitroglycerin, then the physician should be notified and other means of therapy employed.

It has been my tendency to withhold other antianginal drugs during the first two weeks of impending infarction, since they do not help and, in fact, I have the clinical impression that they may aggravate the pain.

The major point of contention in the treatment of impending infarction is the question of the use of anticoagulant drugs. It should be stated at the outset that these are administered routinely and promptly by the majority of physicians in this country. Heparin may be given for the entire period of four to six weeks or may be replaced by a coumarin derivative after two or three days. In our experience, however, there has been no difference in outcome whether these drugs are used or not; in either case, the incidence of transmural and subendocardial infarction, the incidence of death, occurring later was approximately the same. The value of anticoagulant drugs, in impending infarction in our opinion, has not been proved; therefore, whether they
should be used or not should be left to the decision of the physician in each case. I repeat, my conclusions in regard to anticoagulant therapy concerns only the type of "impending infarction" or "premonitory phase" of coronary occlusion that I have described. I have not accumulated statistics in the other types.

EMPHYSEMA AND THE CHEST FILM

On the basis of radiologic and pathologic study, the following conclusions were reached: a reduction in the caliber and number of peripheral pulmonary arteries, often with an increased transradiancy of the background due to a reduction in the vascular bed were the most reliable radiologic signs of emphysema; centrilobular emphysema was not detectable radiologically when mild but moderate or severe grades, which were usually accompanied by diffuse (panacinar) emphysema, could sometimes be detected radiologically; there was little correlation between the percentage area of the lung slice involved by emphysema and the radiologic evidence of emphysema. Only the severest grades were detectable. Sometimes almost the whole of a lung slice showed diffuse emphysema, with loss of alveolar detail and yet the radiograph was normal or inconclusive; the radiologic demonstration of severe emphysema in one area did not necessarily indicate that the rest of the lung was emphysematous, but it was frequently low, flattened, diaphragmatic domes, wide, horizontal rib spaces and a large retrosternal space were found to correlate with large lungs measured radiologically and pathologically. Although severe emphysema was found in some large-volume lungs, not all large-volume lungs were emphysematous and equally severe emphysema was found in lungs of normal volume.


BRONCHOGENIC CARCINOMA

Two cooperative clinical studies on cancer of the lung are being done by Veterans Administration hospitals. The results with cancer chemotherapy used as an adjuvant to surgery have as yet shown no increase in the cure rate of this disease, but on the contrary, have revealed an increase in the postoperative mortality. The several compounds used to treat unresectable cancer of the lung have not given prolongation of life; instead, the use of cortisone has resulted in a significant decrease in the survival time of these patients.


SEGMENTAL INFILTRATIONS DUE TO BRONCHIAL OBSTRUCTION IN PRIMARY PULMONARY TUBERCULOSIS

The bronchial wall becomes involved in the tuberculous inflammation of the hilar lymph nodes. The bronchial lumen becomes narrowed. Next, the lumen becomes blocked by polyedps masses of granulation tissue, developing around a fistula when perforation of a caseated lymph node has taken place. The caseous debris of the lymph node expelled through the fistula become accumulated in the pulmonary segment (or lobe). The caseous material contains both tubercle bacilli and non-bacterial elements. The former cause focal of tuberculous inflammation, whereas, the latter give rise to a nontuberculous pneumonia. It is imperative that patients with these conditions be treated with tuberculostatic drugs. Bronchoscopy is essential for making the diagnosis. Its regular repetition is, furthermore, all-important for the treatment; endoscopic treatment not only enables us to prevent the bronchial occlusion to a considerable extent, but at the same time prevents the accumulation of caseous matter containing tubercle bacilli. Since it has been found that some 70 per cent of the cases only reach a stationary phase after six months, pulmonary resection should be considered only when the infiltration has been present for half a year.

An indication for surgical treatment is present: when the infiltration, in spite of drug treatment, is distinctly increasing; when no sufficient regression occurs or when periods of regression alternate with periods of increase; when the sputum remains positive for a long time; when a pronounced shrinkage of the affected area persists, with deformation of bronchi; when non-tuberculous inflammations or recurrences develop in the area affected.