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Chest Pain, Exercise Electrocardiography and Coronary Arteriography

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Abstract

Over the last 6 or 7 years Dr. Richard Ross and I in the department of medicine at Johns Hopkins Medical School have had a continuing interest in objective methods which might be used in assessing the individual who comes to the physician complaining of chest pain. Angina pectoris is many things; it is a metabolic defect with lactate excess, it is a certain pathological picture, it is a group of individuals who are disposed to have certain things happen to them, but most of all angina pectoris is pain in the chest. It is pain in the chest as far as the patient is concerned, and this is the symptom on which the physician has to base important decisions concerning diagnosis, prognosis and treatment. The problem with chest pain is that it is a very subjective complaint, and a discussion with the patient is certainly the most satisfactory way to establish its cause.

CHEST PAIN, EXERCISE ELECTROCARDIOGRAPHY AND CORONARY ARTERIOGRAPHY

(Abridged)

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Over the last 6 or 7 years Dr. Richard Ross and I in the department of medicine at Johns Hopkins Medical School have had a continuing interest in objective methods which might be used in assessing the individual who comes to the physician complaining of chest pain. Angina pectoris is many things; it is a metabolic defect with lactate excess, it is a certain pathological picture, it is a group of individuals who are disposed to have certain things happen to them, but most of all angina pectoris is pain in the chest. It is pain in the chest as far as the patient is concerned, and this is the symptom on which the physician has to base important decisions concerning diagnosis, prognosis and treatment. The problem with chest pain is that it is a very subjective complaint, and a discussion with the patient is certainly the most satisfactory way to establish its cause. If the pain be typical in quality and duration as is described in Heberden's reports and later emphasized by Osler, one can confidently conclude that it is indeed angina pectoris, that the coronary arterisclerotic process is severe, and that the prognosis – though varied – is more or less predictable. However, particularly in the current epidemic in Western societies certain serious diagnostic problems arise in individuals complaining of chest pain. There are two main reasons for this. One is that some other kinds of pain very closely mimic that of angina pectoris; oesophageal and musculoskeletal pains are two notorious examples. This is because the pain pathways that supply these structures are similar to those conducting pain from the heart. The other diagnostic problem arises in individuals who may have only very trivial causes for their chest pain, but tend to be overdiagnosed because of a rather hysterical sort of attitude on the part of both patients and doctors. The former are influenced by the lay press and awareness of the seriousness of chest pain, and the latter by

fear of "going out on a limb", so to speak, and missing a serious diagnosis. In addition there are other problems in patients with clear-cut ischaemic heart disease. Our ability to prognosticate is still inadequate and limited, and the means by which we can evaluate therapy are still not fully satisfactory. The objective methods which I am going to describe are first, functional evaluation of the individual and his complaint by exercise electrocardiography and second, anatomical assessment of the coronary circulation by selective coronary arteriography. The only question to be answered in this group of patients is, "Is ischaemic heart disease present?" Of approximately 800 whom we have seen over a 6 year period, 238 have gone through the entire gamut of investigations in an effort to answer this question. In the others we felt that more simple and perhaps less hazardous means of investigation were sufficient. The 238 individuals all had chest pain, which had been diagnosed by one or more physicians as angina pectoris, but they can be divided into two groups according to the history – those whom we concluded had ischaemic heart disease and the others in whom our history did not, clearly, suggest that it was present. The mean age in each group was 44. And, we felt that there were good and justifiable clinical reasons for full investigation of all these patients. None had persistent hypertension or X-ray cardiomegaly. I would like to review the methodology we have used, and emphasize that this was a prospective study. Our routine investigation included blood lipids, sugar, postprandial blood sugar estimations, exercise electrocardiography and selective coronary arteriography. Though one might argue about the need for arteriography, if it is done at all – the selective method provides the most information. Other studies were made on the basis of clinical indication, not routinely in all patients. We have considerable follow up information on every

individual obtained at annual intervals, either by seeing the patient again, or by letter from the referring physician. All information obtained during in-patient investigation, which usually took about 5 or 6 days, has been programmed in a computer for easy handling and correlation, the follow-up data being added as it becomes available so that we can continue to alter our "final" assessments. I believe it is very important to make a firm opinion on the history, without seeing the electrocardiogram, arteriogram or even perhaps the referring physician's letter. In this study two or more physicians specifically interested in chest pain – (in nearly all cases I have been one of them) took a history and recorded what they believed to be the cause of the pain. For the purposes of this study, "Typical" angina pectoris is defined as related to effort, relieved by rest, at least partly substernal in location and visceral in character. It can have other features as well but it must have these three characteristics, and the physician taking the history must be convinced of its ischaemic cardiac origin. If the pain is classified as atypical angina pectoris, this means that in the judgement of the clinician it is probably ischaemic myocardial pain but does not fulfil one or more of the criteria outlined. A third classification is "pain of uncertain origin", and in this case two or more clinicians, are uncertain whether the pain is cardiac in origin. There is a fourth category in which all clinicians associated with the study agree that the chest pain present does not arise from the heart. We even had 6 patients with no chest pain at all.

As regards the electrocardiagram we have devised light-weight electrodes which can be placed on the chest, so that 12 lead tracing can be obtained during exercise. Similar electrodes are now available commercially. Nearly all muscle "noise" can be eliminated by placing the arm leads below the clavicles, and this method together with normal chest and right leg leads provides a conventional electrocardiogram for interpretation. Other kinds of leads, which might be easier to apply can produce difficulties in interpretation; experience with Bi-polar leads, for instance, is too limited for complete reliability. I believe that our method increases the safety of the examination, since the electrocardiogram from multiple sites on the chest can be observed continuously during exercise.

The criterion which we have arbitrarily selected to indicate ischaemic during exercise is a square or down-sloping T.S. segment depressed one or more millimetres from the baseline with a duration of 0.08 second or longer, and persisting for 3 or more beats. As regards the exercise load, the patient currently walks upon an escalator – ergometer (we used a bicycle previously) the load being adjusted to the heart rate response. Criteria for stopping the examination are:

1. The patient complains of fatigue or breathlessness.
2. The ECG shows ischaemic changes regardless of whether pain occurs or not.
3. Pain occurs, believed by the physican to be angina pectoris.
4. The heart rate reaches 90 per cent of the predicted maximum – which is a function of age, not conditioning.

The exercise electrocardiogram obtained under these conditions is interpreted by physicians not otherwise associated with the study. The coronary arteriogram is made by the selective technique and classified arbitrarily as follows:

0. No abnormalities on the arteriogram;
1. Minimal irregularities (we cannot attribute any clinical significance to this degree of arteriographic change even though on the basis of post mortem injection studies I am convinced that the least irregularity revealed by this crude method indicates appreciable narrowing – perhaps 20 or 30 per cent of the cross-sectional area of the lumen).
2. "Localized, severe narrowing" (a debatable degree of change and therefore in quotes).
3. Multiple narrowings.
4. One or more totally obstructed vessels.

This is not necessarily a progressive scale; many individuals in class 3 (multiple narrowing) are more severely ill than some in class 4 who have only one artery totally obstructed, and good collateral circulation.

We now come to the information derived from this study, which was designed to assess whether or not ischaemic heart disease was present and assess its severity by objective methods.

On the history, 91 of the 238 individuals had typical angina pectoris by the criteria defined, of these, half had a normal electrocardiogram. 40 had atypical angina pectoris, 66 had pain of uncertain cause and 41 patients did not have angina pectoris in our opinion. Regardless of classification, most patients had a normal electrocardio-

gram. Of the 91 individuals with typical angina pectoris all except 2 or 3 participated in exercise electrocardiography, and an adequate test was defined as one in which the heart rate reached 90 per cent of the maximum, or in which angina pectoris was objectively manifest by an ischaemic change on the electrocardiogram. Of those with an adequate test 57 showed ischaemic change, 15 did not. Arteriography in these same 91 patients revealed very severe changes in 77, a "severe localized narrowing" in 7, and no, or trivial change in the remaining 7. On the basis of all the information we have to date (and we have followed up some patients for 6 years) we have attempted to decide whether ischaemic heart disease is present or absent. In the 91 individuals originally classified on the history as typical angina pectoris we concluded that ischaemic heart disease was indeed present in 86. In 4 we decided (mainly on the basis of follow up information) that we were wrong in our initial historical evaluation, and that ischaemic heart disease was absent; in 1 we remain uncertain. It is interesting to note that 84 of these 86 individuals with "typical" angina pectoris had very severe arteriographic change.

The other group of great importance in assessing the usefulness of our method consists of the 41 individuals with chest pain not thought to be angina pectoris on the history alone. 2 out of 34 who had adequate exercise electrocardiogram by our criteria did have an ischaemic change; the arteriogram in 39 of the 41 showed no or trivial abnormalities but in 2 patients the changes were very severe. Follow up data in these 41 individuals have led us to believe that one individual does indeed have ischaemic heart disease, while the initial impression that it was not present seems to be borne out in 36, and in 4 we remain uncertain. At least 3 possible explanations can be offered for the 2 patients not diagnosed on the history. First, they may indeed have had angina, related to the changes seen arteriographically. Second, their symptoms could have been a mixture of ischaemic and non ischaemic. Third, they may belong to the 5 per cent of individuals in this group (mean age 44) with severe coronary changes, but no present or previous ischaemic complaints.*

Conclusion

The arteriographic patterns seen in individuals with "typical" angina pectoris (by our definition) may vary considerably, although serious arteriographic change is nearly always found. 238

patients with troublesome chest pains thought to be due to ischaemic heart disease were evaluated by clinical and laboratory studies including exercise electrocardiography and selective coronary arteriography. The arteriographic study tended to provide the most definitive diagnostic data, but this is related in part to the fact that a relatively young age group was studied. Angina pectoris is virtually always associated with severe arteriographic change, but many persons have less marked disease arteriographically than post mortem studies would suggest. This is reasonable in light of the fact that we have studied a population in an early stage of their disease. The patterns associated with myocardial infarction are more variable than those seen in angina pectoris. Our early follow up studies suggest that coronary arteriography may increase our knowledge of the natural history of coronary artery disease and lead to more accurate diagnosis, better treatment and more reliable prognosis.

*The patients in the clinical groups, atypical and uncertain pain had variable degrees of arteriographic abnormality – from class 0–4. It is likely that it is in these groups the aerteriographic method is most helpful in clarifying clinical problems.