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Panel Discussion

Chairman – Dr D. G. Julian
Panel – Richard Gorlin, Ottar Müller, G.C. Friesinger, C.F. Borchgrevink, Sir John McMichael, F.R.S.

Abstract
The Management of the Patient with Angina Pectoris

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PANEL DISCUSSION

Saturday 22nd April

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The Management of the Patient with Angina Pectoris.

Chairman: I thought that we might concentrate this discussion on the more controversial aspects of treating angina but I realise that almost the only uncontroversial aspect is the use of short term nitrites. It is a tribute to Lauder Brunton that this seems to be the only form of treatment on which we would probably all agree. Much more controversial is the use of long-action nitrites. Should one employ these clinically?

Dr. Friesinger: I myself prescribe a long-acting nitrite but I must be confident that the angina is at a stable phase. My usual practice is to have the patient keep a log; he is asked to describe the effects he is experiencing with the drug. He is told to take the drug for three weeks and then discontinue it for three weeks, continuing in this fashion for some length of time. I see him every few months, review the log and decide whether it is worth the expense. I believe that the adequancy of the dose is an extremely important point and in my opinion some manufacturers of long acting nitrites recommend the wrong dose; it is too small. As I review the log I look for the side effects resulting from the pharmacological action, such as headache and postural hypertension to establish the adequacy of the dose.

Dr. Fulton: It is my impression that most clinicians utilise short-term nitrites and apparently with good effect. They are difficult to evaluate because of the placebo effect of treatment and the varying degree in which a patient experiences angina over the sort of period that one may conduct a trial; published work confirms this. As far as long-term nitrites are concerned, I have not found that they have more to offer than the repeated use of short-term nitrites.

Question: Has anyone used aerosol inhalers for a more accurate delivery of dose and if so, with what sort of results?

Dr. Muller: I have tried one type of aerosol inhaler containing nitroglycerine in a few patients and found very similar effects to those achieved with amyl nitrite.

Dr. Oliver: In relation to sustained release of nitrogylcerine as distinct from other long-term nitrites could the panel tell me whether they are convinced that any nitrites do not cause peripheral vasodilation?

Dr. Friesinger: I must see peripheral vasodilation before I am satisfied, but what form the drug takes seems unimportant to me. One must titrate the dose to the patient to the point where peripheral vasodilation is occurring, and I think that in the prophylaxis of attacks this is a necessary guide line.

Question: Has nicotinic acid or nicotinamide been tried?

Sir John McMichael: Nicotinic acid treatment has been extensively tried and is said to lower blood cholesterol but I do not think the results have been dramatic in angina.

Chairman: Propranolol has created the most interest recently and I would like to ask if anyone has found this to be effective in the treatment of angina?

Dr. Fulton: The published reports by others would suggest that it is. A proportion of my patients have benefited from treatment with the drug and so have others in whom angina has been associated with tachycardia. In some instances some individuals are more satisfactorily treated with propranolol than with nitrites

although of course there is no objection to using both concurrently. Those who do use both have found that they have been able to reduce the nitrite requirements.

Chairman: Do you think there are any dangers in giving propranolol?

Dr. Fulton: I have not noted many problems and would have thought the dangers are quite small if the dosage is gradually built up for the individual patient who was not in some unusual state, who simply had angina pain without severe cardiac failure. There has been very little evidence that anyone has run into much trouble this way. The curious thing is that the dosage which has surely been used for the relief of angina has been far in excess of the dose which is accepted as being sufficient to produce full beta-adrenergic blockade.

Dr. Friesinger: It is a useful drug but I do not think that one can generalise. It seems to me that in patients in whom the tachycardiac response or the elevation of blood pressure seems undue for the amount of effort that they are performing, this drug should be very useful.

Dr. Muller: I have only had experience with propranolol with regard to its possible ability to precipitate left ventricular failure. I tried propranolol on sub-normals and on exercise found increased left heart diastolic pressures. In patients with coronary heart disease there was no increase in these pressures, so I gather that there must have been a balance between the positive and the negative effects of these patients; they did not seem to go more into left heart failure without it.

Sir John McMichael: I understand that the drug was something we had in reserve for use when anginal pain appears to be quite uncontrollable by ordinary means.

Dr. Lassers: Yesterday Drs. Gorlin, Friesinger and Muller talked about the elevation of left ventricle end-diastolic pressure and wedge pressures on exercise in patients with angina. There is at least some evidence that the apex cardiogram correlates with these parameters. Has Dr. Friesinger had any experience with apex cardiography and how it correlates with other parameters in the diagnosis of ischaemic heart disease, and secondly, I should like to ask the panel what work has been done with the apex cardiography and the effects both of propranolol and nitrites?

Dr. Friesinger: You are of course quite right in saying that the apex cardiogram, that is the amplitude of the (a) wave can be a reasonable guide to the elevation or the degree of elevation of left ventricular end-diastolic pressure. In a number of patients in whom we have carried out these exercise tests we have obtained apex cardiograms before and after exercise and in a number of them at a time when they were undergoing catheterisation with a catheter in the left ventricle. We obtain an apex cardiogram and correlate directly the reading that we get from the ventricle with the apex cardiographic signal obtained simultaneously from the chest. In this way, one can get a linear correlation between the left ventricular end-diastolic pressure and the signal from the apex cardiogram, provided that the elevation of the left ventricular end-diastolic pressure(?) is mainly due to an (a) wave kick. If all the prameters of the left ventricular end-diastolic pressure are elevated – the early as well as late – and there is not a large atrial component contributing to the final diastolic volume – hence the left ventricular end-diastolic pressure, then the correlation tends to be not so good. It is indeed a useful sort of thing to look at when the correlation is good and the patient has a normal apex cardiogram on exercise; if it becomes abnormal you obviously have good evidence to indicate that he has gone into a kind of ventricular failure.

Chairman: Would there be a good case for digitalising our angina patients?

Dr. Muller: I have no experience whatsoever of the digitalisation of patients with angina. Some have reported good effects and others to the contrary. In a recent study of the effects of intensive digitalisation of patients with coronary heart disease, the author found he could produce an exercise test which precipitated anginal pain and on repeating this test 50 per cent of his patients did not complain of pain, and this seemed to be correlated to improved left ventricular function as was seen by cardiac catheterisation.

Chairman: Can we now deal with anticoagulants? Perhaps Dr. Borchgrevink would tell us whether his controlled group were in fact receiving small doses of anticoagulants.

Dr. Borchgrevink: I would first like to make one general comment and it is that anticoagulants therapy is prophylactic when given to survivors after myocardial infarction to prevent further infarctions and it seems odd to me that 90 per cent

of the publications dealing with anticoagulant therapy are primarily concerned with preventing second and third infarctions when the first infarction is fatal in about forty per cent of cases. If people use long-term anticoagulants after myocardial infarction it seems illogical not to use it prophylactically in patients with angina whom they know are more prone to develop myocardial infarction. In our study we gave intensive treatment to half the patients and the remainder what we thought to be moderate in terms of P & P test used at that time. The first group were treated at fifty per cent to sixty per cent intensity and the second at twenty per cent intensity, the former being more successful of the two. I drew the conclusion that if we are going to use anticoagulant therapy our treatment should be intensive.

Dr. Fulton: I have already put forward the suggestion that in the majority of instances clinical coronary heart disease is due to thrombosis, although the evidence is very hard to come by; one does not know just what is the position at the onset of clinical illness which then goes on for quite a long time before there is any pathological examination. The clinical evidence of sudden onset of anginal symptoms, (by sudden, I mean that one day the patient has no angina and the following day he could recall an experience of angina) which has apparently entered his life. In the course of time this often passes as having been gradual in onset because it was not very severe, but I think that very often this was thrombosis right at the onset of the symptoms. Accordingly, one would feel that if anticoagulant therapy is going to benefit this situation the earlier it is used the better, and of course, prophylactically.

Sir John McMichael: I have examined the published papers on the use of anticoagulants and noted flaws in some of the trials which were carried out. There were faults in the selection of patients and often it was seen that control cases were found to be much worse than expected. It was said that patients bled from overdosage of anticoagulants whilst at the same time they produced new thromboses. After trying this form of treatment and having looked at all the depressing evidence we decided to give it up.

Dr. Borchgrevink: I think this complex of bleeding with thromboses occurring at the same time is not as confusing as it seems. Thrombosis as you know, consists of two parts: the white head consisting of platelets and the red tail which is more or less clotted blood. The anticoagulant therapy we use, and that includes heparin, does not influence the formation of the white head; it can only delay or inhibit the formation of the red tail. That means that even with ideal anticoagulant therapy making the blood unclottable a white head may still form and occlude the lumen.

Chairman: May we now discuss surgery? Do the participants believe that internal mammary implantation in the myocardium is an effective way of supplying blood to the myocardium?

Dr. Fulton: I have not seen the original angiocardiographs of these studies, but the published statements in some studies would suggest that forward flow through the internal mammary has been achieved into areas distal to coronary occlusion. The unfortunate thing about some of the earlier cases has been that in the course of time the artery which was grafted has again become occluded. From the pathological point of view it would seem to me that there must be opportunity for introducing new blood supply into the coronary circulation in the presense of coronary artery disease. The coronary arteries of man and of mammals have been designed to supply the heart muscles from without, inwards. In the presence of disease the deep vessels may be greatly dilated and intercommunicating to the point at which entry of blood into any one part of that system could in fact be distributed to all parts of the left ventricular wall, and this occurs in man. At the moment it would seem that the difficulties of supplying new blood to the system are technical rather than absolute. In principle I can see that there should be a good future for such procedure.

Dr. Friesinger: We have carried out very few surgical procedures in the Sir Johns Hopkins Hospital; about twenty five patients have been operated on for ischaemic heart disease in the last five years. We consider that this is an investigative surgical procedure whose worth is not known, but on reviewing the data up to the present time it would seem that surgical morbidity and mortality are low; the theory on which surgery is based seems relatively sound. A small group of patients who are incapacitated because of angina of long duration and which can be demonstrated on a severe anatomic basis can be subjected to these procedures, but one must follow up results over a number of years before one can give a true opinion as to its value.

Chairman: Would Dr. Rees give his opinion on

the effectiveness of surgery in terms of providing a new blood supply?

Dr. Rees: The main problem is which patients should we select to undergo surgery and what would be the effect on the prognosis if to some extent we improve the collateral flow in the area of the myocardium. I believe, without question, that it is possible to improve the blood flow to an area of ischaemic myocardium by implanting a systemic blood vessel into that area but whether or not one then improves prognosis by doing so is still not proven. If an improved prognosis can be obtained by subjecting the patient to surgery then perhaps we could extend the procedure to those patients who have not suffered a first infarction and hopefully prevent infarction occurring. This would mean those patients who have severe disease which has been demonstrated by arteriography and whose myocardium has not yet become necrosed or scarred.

Chairman: Would you say something about the mortality risk and technical difficulties?

Dr. Rees: There are two things involved, the first is the type of patient you are dealing with and the other concerns technical aspects of surgery. Firstly, the mortality is going to be directly proportional to the care with which you select your patients. If you limit surgery to those patients who have angina decubitus, that are in degrees of heart failure, and who have previous infarctions, then your operative mortality may be as high as two or three per cent, as suggested in the Bigelow and Effler reports. Secondly, technical aspects of bringing one vessel from one place and from another; the only thing that has been proven statistically is that you can implant the internal mammary artery into the anterior wall of the left ventricle with a patency of eighty per cent. Whether or not bringing a graft from the posterior aorta or bringing up a splenic artery or implanting both internal mammary arteries is useful remains to be proven. They may be useful anatomically but there are other ways in which one can pick up an artery from the chest wall, such as by using both internal mammary arteries, bringing one up through the diaphragm or posteriorily using the intercostals.

Chairman: Do you regard a previous infarction as a contraindication to surgery?

Dr. Rees: No. In approximately one hundred patients on whom surgery had been carried out, Vineberg reported 60–65 per cent had had previous infarctions so that the patient who has ischaemic muscle and is suffering pain, not necessarily from scar tissue, would still be a candidate even though he had had a previous infarction.