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THE BRITISH SOCIETY FOR CARDIOVASCULAR RESEARCH
Quarterly Bulletin

Edited by:
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BSCR Quarterly Bulletin
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EDITORIAL

The recent car bomb attack on a biomedical research worker which lead to the serious injury of a small child has reminded us that we, and what we stand for, remain under threat from the lunatic 'all creatures have rights' lobby. As human beings, we in the editorial office feel particularly outraged (if we may be permitted to use this overworked word) by the lack of media vitriol against the perpetrators; contrast this with the tales of tortured doggies which appear with monotonous and exasperating regularity, and the way these tales are put to effective propaganda use by the anti-humanists. Perhaps we have something to learn from them in this regard. In our view, it would be legitimate to plaster the 'tube' with posters showing a photo of a healthy child after heart surgery and the caption 'we do this', with, next to it, a large photo of the Bristol bombing push chair with its shrapnel rent in sharp relief and the caption 'they do this'. Alas, the public memory of this act is now fading, and an opportunity has been lost; the image of the wicked man in the white coat and the cat with wires leading from its head once again reigns supreme.

LETTER FROM THE SECRETARY

Rules made to be broken...

The founding fathers of the Cardiac Muscle Research Group, back in 1973, wisely decided not to burden us with a written constitution. Instead, they devised a list of rules, none of which, unfortunately, provided a mechanism for subsequent changes or additions. So were they designed to remain an immutable tribute to common sense, or were they merely a framework for enlightened anarchy? Subsequent committees seemed to have assumed the latter, and the rules have been effectively changed, either by votes at the AGM, by ballots, by votes of the committee, or simply by practice. As we grow in number and stature, the question of charitable status and hence of a formal and legally-sustainable constitution will naturally arise; probably quite soon. For the time-being, the following statement of the 'rules' as reflected in current practice may help to avoid an identity crisis!

1. The name of the Society (formerly the Cardiac Muscle Research Group) shall be the British Society for Cardiovascular Research.

2. The annual membership fee shall be fixed periodically by postal ballot, and shall fall due on 1 January.

3. Two meetings shall normally be held each year during the Autumn/Winter meeting of the Society.

5. Members of the BSCR shall nominate and elect a committee consisting of nine members. Nominations for vacant posts and postal ballots, if necessary, shall take place annually, and those elected shall take up office on 1 January of the following year. The Committee may co-opt additional members.

6. Members of the British Society for Cardiovascular Research shall be elected to the Committee for three years only, but may be co-opted for an additional period under exceptional circumstances.

7. A member of the British Society for Cardiovascular Research may stand for re-election to the Committee only when the member will have been off the Committee for one whole year.

8. At least three of the members of the Committee shall be clinicians and at least three non-clinicians.

9. The Chairman, Secretary and Treasurer shall be elected by the Committee at the AGM and shall serve for a maximum of three years. They shall take up office on 1 January of the following year.

10. The Chairman, Secretary and Treasurer of the Committee may be appointed for a period longer than three years only under exceptional circumstances. If a member of the Committee is appointed to one of these offices, then the member may remain on the Committee for three years from the time of that appointment and Rule 6 shall not apply.

Andrew C Newby
Our Society continues to have first-class scientific meetings. We are grateful to Dr Susan Coker for organising an excellent Spring meeting in Liverpool and for raising sufficient funds for a substantial surplus to be passed over to the society. We owe a great deal to our sponsors and we hope that sponsorship will continue to grow. Dr Paul England is coordinating fund raising on behalf of the Society in order to establish both a stronger financial base and a regular income from sponsorship. We hope that expansion and diversification of our activities will eventually be possible. Centralization of fund raising aims to prevent multiple approaches to industrial firms from different individuals when they organise meetings and workshops on behalf of the Society. We hope to become rich enough to potentially remove the need for local organisers to raise a large proportion of their own funds. This will give us the freedom to hold meetings on basic cardiovascular topics which are scientifically important but which may be less directly attractive in respect of industrial sponsorship. Eventually it would be good to be able to give financial help to young researchers to attend Society meetings and to spend time in the laboratories of other groups. More about these matters in later editions of the Bulletin.

One of the hallmarks of the Society has been its informality and I am determined that this should continue. While research workers are now free (encouraged!) to submit posters for Society meetings which may be published in the Bulletin, I believe that the freedom to exchange ideas and results without the constraints of necessarily having to publish allows people to voice ideas which they are perhaps less confident about and to discuss results which are more up-to-date. Nevertheless I think that there will have to be a bit of tightening up on some of the more boring administrative aspects of the Society so that we can serve the membership better and use our resources most efficiently. Drs Newby and Cummins are setting up a database containing details of members' addresses, research interests, etc., and you will be asked to fill in a form so that we can obtain accurate information. This will allow us to post the Bulletin and other details more easily and will make possible better exchange between members sharing similar interests. Obviously we will need to register the database and you will be kept informed of your entry. We hope to send each member a sheet of his or her stored details each year which can be returned, updated, if necessary. I am sure that these and other changes which are envisaged will be worthwhile and I am very grateful to the committee members (and in particular the Secretary and Treasurer) for their hard work on the Society's behalf.

Dr Peter Cummins organised a much-appreciated Workshop on Growth Factors (in and out of the heart) which took place in May and there are more in the pipeline. I should just like to plug workshops as one of the best ways of sharing information among those keenly involved in a developing area. They are informal, one-day meetings and members are restricted to allow efficient interaction between all participants. If you feel that your area of interest could do with a workshop please get in touch with the Secretary. The Society presently contributes £400 to the costs.

This is my first letter since assuming office and I hope that you will respond by letting me know of suggestions and criticisms which you may have of the issues raised. I am very conscious of the contribution which my predecessor David Hearse has made to raising the image of the Society and his will be a hard act to follow. I believe that the Society has a greater role than ever in these days when we all have to be able to talk each others' scientific language.

I look forward to seeing you at the meeting on E-C coupling in December.

George Hart
LETTER FROM THE TREASURER

The Society's former treasurers have been content to make the occasional request to the membership to maintain subscriptions, whilst permitting errant members to remain on the mailing list and, indeed, continue to receive full membership rights. However, it is not in the interests of the Society for this to continue. Despite a good response to the reminder in the last issue of the Bulletin, many people have yet to act. The committee has asked me to deal with this. To help you identify possible problems associated with your subscription, and thereby ensure your remaining a member of the Society, I would ask you to consider the following (by way of gentle persuasion, I will delete from the membership list the names of all those, in arrears, who fail to respond to this request by Jan 1 1991).

1. Has your membership lapsed? If you wish to remain a member of the Society, can you double check that you are paying your subscription each year. I am not asking for money in arrears, simply that money for 1991 (and thereafter) be forthcoming at the appropriate time.

2. Are you paying the correct amount? Almost 20 people are still paying the old subscription of £2. Such £2 contributions from "friends of the BSCR" will continue to be gratefully received. However, "friends" will no longer receive the Bulletin or free entry to meetings. "Friends" who wish to be upgraded to full members should revise their standing orders. Many people are paying the student subscription of £6, but it is not clear whether they remain genuine students or are currently students only in the school of life. Current subscription rates are listed at the foot of this letter.

3. Can your subscription payment be readily identified as yours? There are a few entries on the Society's bank statements that are clearly subscriptions but which cannot be linked with any individual owing to the name of the fund source. Therefore, although these people will have paid their subscription they will nevertheless be deleted from the membership in January. To avoid this happening, please consider your fund source and, if it is different from your name, please write to me or telephone, to clarify the situation (address on page two). Here are some examples of unidentifiable fund sources:

- Myocardial Research
- Mid Head Office
- IP2MC89041700230
- Cardiovascular Res
- Smith/Kane

4. Is your subscription timed for January? To ensure that your subscription be properly processed it is essential that payment arrives in January (January 1 if payment is by standing order and as close to this as possible if by post).

5. Are you paying too much? Amid this sea of neglect there are approximately 20 members who have kindly decided to pay two or three subscriptions per year (usually by standing order). Your bank will give you details of standing orders on request. One payment per year is sufficient; additional payment is welcome, but not necessary.

6. What does it cost to become a member? To subscribe to the BSCR, the following rates apply:

- Member: £10
- Student member: £6
- Joint membership with ISHR: £25
- Student joint membership with ISHR: £21

7. How should you make your payment? We prefer payment by standing order. This should be arranged as follows:

- Account: British Society for Cardiovascular Research.
- Account number: 06124151
- Bank: National Westminster Bank plc,
- 13 Stonehills,
- Welwyn Garden City,
- Herts, AL8 6ND.

To make life even easier for you, (and for new members) we have special standing order forms. These are available from the Society's secretary (Dr Andrew Newby, see page two for address). If you don't like the idea of standing orders, payment by cheque is acceptable. Subscriptions cover a one year period beginning on January 1 of each year.

M R Boyett
ELECTIONS TO THE BSCR COMMITTEE 1990: THE CANDIDATES

There have been three nominations as detailed below for the two places on the Committee to be vacated by David Hearse and Stuart Cobbe. A ballot paper is enclosed with this Bulletin and this should be returned to the Secretary by 31 September 1990. The result of the ballot will be announced in the October Bulletin and those elected will take up office from 1 January 1991.

GIANNI ANGELINI

Gianni Angelini received a degree in Mechanical Engineering from the University of Siena in 1972 and went on to study medicine receiving his MD in 1979. He received specialised training in cardiac surgery in the University Hospital of Wales, Cardiff and the Thoraxcentre, Rotterdam between 1981 and 1989 and was awarded his FRCS in 1986. The work for his MCh thesis carried out in the University of Wales College of Medicine won him the Young Research Workers Prize of the British Cardiac Society in 1986. Mr Angelini is now Senior Lecturer in Cardiac surgery at the Northern General Hospital in Sheffield. He joined the BSCR in 1988. He is proposed by A C Newby and seconded by A H Henderson.

TREVOR POWELL

Dr Trevor Powell was educated at King’s College and the Institute of Basic Medical Sciences, London, obtaining his Batchelor’s degree in Physics and his Ph.D. in Biophysics. He was then awarded an MRC Junior Research Fellowship, before being appointed to a lectureship at the Middlesex Hospital Medical School. In 1968 he went to Baylor College of Medicine in Houston, Texas, to study Physiology and returned to London after obtaining his Ph.D. to develop a research program on cardiac function using single ventricular myocytes as an experimental model. In 1986 he was appointed to the Winstone Readership in Cellular Cardiology (funded by the British Heart Foundation) at the University Laboratory of Physiology, Oxford, and he is also an Official Fellow and Tutor in Physiology at New College. His present research interests are concerned with mechanisms underlying chemical- and ion-gated transport processes in the regulation of cardiac rhythm and inotropic state. He is proposed by M R Boyett and seconded by G Hart.

DEREK YELLON

Derek Yellon obtained a BSc from the University of Cape Town and a PhD from the University of Bath in 1978. He held a Postdoctoral Fellowship, Honorary Lecturership and Honorary Senior Lecturerships at the Rayne Institute at St Thomas's Hospital from 1978 to the present. He was Medical Director of Lorex Pharmaceuticals from 1987 to 1988 and is now Director and Head of the new Cardiovascular Studies Unit at University College Hospital. He is also Honorary Senior Clinical Lecturer in the Department of Physiology at University College London. Derek has organised many national and international meetings, workshops and round table meetings on behalf of the former Cardiac Muscle Research Group and the International Society for Heart Research. He has been a member of the BSCR since 1975, and served as a Committee member from 1981 to 1986; as Secretary from 1983 to 1986. He is proposed by A-M L Seymour and seconded by K A A Fox.

Ballot papers (enclosed) should be returned by 31 September 1990 (postmark) to Dr A C Newby, Department of Cardiology, University of Wales College of Medicine, Heath Park, Cardiff, CF4 4XN
CARDIOVASCULAR RESEARCH - PRESENT IMPERFECT, FUTURE UNCERTAIN?

A recent survey among US researchers revealed an almost universal perception that research funding had shrunk and that grants had become more difficult to obtain, despite figures showing an increase in the research budget in real terms! Discounting statistical aberrations, either we have a built-in pessimism, or, more plausibly, our ambitions and aspirations simply outstrip the growth in resources. Looked at optimistically, our funding problems arise from ever greater competition between more, bigger and better applications. If this view is even partly correct, then cardiovascular research will have to rise to meet this challenge.

What then is the present relative position of cardiovascular research in the U.K., and how does it compare with similar countries in Europe and elsewhere? There has, to my knowledge, been no detailed survey to answer these questions, but the conclusions from any such survey would have to take into account the actual and perceived importance of cardiovascular diseases. For example, the MRC spent 3.5% of its budget directly on cardiovascular research in 1988/9 (although as much as 10% was spent on research with some relevance to the cardiovascular system). In the same year, cardiovascular causes accounted for 41% of deaths among men of working age. The German D.F.G. gives direct spending on cardiovascular research a higher priority (7%), as does the American N.I.H. (14%). These figures certainly support the perception of government underfunding of cardiovascular research made worse by the general climate of retrenchment. Given the increasing emphasis in the private-sector on short-term commercial goals, the situation might indeed be desperate were it not for buoyant charitable funding of basic research by the British Heart Foundation and others. Even there, however, the resources are perhaps only 20% of those of the major cancer research charities, despite an almost identical justification on the grounds of premature mortality.

Why, therefore, is cardiovascular research relatively underfunded and what can we do about it? In practice, priorities for funding are strongly influenced by the size and strength of the existing research base and by the energy and skill with which the case is made. Are we then to conclude that cardiovascular research has been relatively weak, and that it has failed to promote itself adequately? Despite obvious exceptions, I feel that we have to take both these proposals seriously. Notwithstanding the past efforts of the CMRG and the BSCR to promote the quality and quantity of cardiovascular research, we may have been slow, unlike our clinical colleagues, to embrace new technologies and take on new directions. Being in the vanguard of new developments is important also because it attracts the brightest and most energetic young researchers into our field. Recent BSCR initiatives in giving a platform for cell and molecular biology, even where the clinical applications may be long-term, are welcome therefore. The BSCR may also have a developing role in bringing together groups of investigators, so as to obtain the most benefit from increasingly expensive research infrastructure.

Whether the BSCR should take the lead in promoting our speciality in government and other public spheres may be a more controversial question. Let us have informed debate on this topic through the bulletin and elsewhere.

Andrew C Newby
BSCR MEETING REPORT
Blood Borne Factors in Myocardial Ischaemia
Liverpool, 28-29 March 1990

This two-day meeting, held at the University of Liverpool was opened by Peter Sleight (Oxford) with a review of the use of thrombolytics in the medical management of myocardial infarction. He compared the results of different trials with streptokinase and tissue plasminogen activator (tPA). Cardiac rupture may be a problem with streptokinase but this can be prevented by β-blockers whereas with tPA there appear to be more strokes than with streptokinase. In view of the vastly greater cost of tPA and its lack of significant advantages over streptokinase it was felt that the future use of tPA may be restricted to patients who had received streptokinase previously and who would therefore be at risk of an adverse reaction if it were given again. Some discussion also centred on their time of administration of thrombolytics after the onset of symptoms. Although some trials have suggested that the earlier the treatment the better, Prof Sleight emphasised that you can treat late and successfully.

This conclusion was endorsed by Dr Miles Maxwell (Beckenham) who described experimental work with recombinant double chain tPA (duteplase). In anaesthetized beagles a copper coil was placed in the left anterior descending coronary artery to induce thrombosis. Either 1.5 or 4 hours after complete occlusion of the artery, tPA was administered. Both bolus injections or infusions of tPA were equally effective in lysing clots and successful lysis also occurred when tPA was given at the later time as well as when given earlier.

Cherry Wainwright (Strathclyde) then discussed the role of platelets in experimental myocardial ischaemia. She quoted evidence that both thromboxane A₂ reduced both ischaemia-induced arrhythmias and reperfusion-induced ventricular fibrillation. In similar experiments PAF antagonists also reduced the number of ischaemia-induced ventricular premature beats (log-transformed? Ed.) and the incidence of reperfusion-induced ventricular fibrillation. In isolated Langendorff perfused rat hearts administration of a platelet suspension 20 sec prior to 10 min of low flow ischaemia exacerbated arrhythmias.

The possible role of serotonin (5-HT) in myocardial ischaemia was then discussed by me (Susan Coker, Liverpool). 5-HT is released from platelets when they aggregate and causes further platelet aggregation and vasoconstriction thus qualifying as a possible causative factor in myocardial ischaemia. Experiments in anaesthetized rats with a number of 5-HT antagonists showed that these drugs had no effect on ischaemia-induced arrhythmias but some reduced reperfusion-induced ventricular fibrillation. In separate studies it was found that the drugs which were effective against reperfusion-induced arrhythmias were those which also prevented the effect of 5-HT on platelets.

Cathy Nieman (Horsham) then talked about platelet fibrinogen receptors and the development of substances which may interfere with the action of fibrinogen at its receptors. The functional expression of the fibrinogen receptor, the GPIIb/IIIa complex, only occurs when platelets are activated and then fibrinogen must bind to this complex to allow platelet-platelet adhesion, i.e., aggregation. Irrespective of the agonist used to stimulate platelets, e.g., thromboxane A₂ or PAF, the final step prior to platelet aggregation is the binding of fibrinogen to its receptors. Thus any substance which blocks this will prevent platelet aggregation initiated by any known activator and could play an important role in the prevention of thrombosis.

The meeting restarted the following morning with an overview of leucocytes and leucocyte-derived mediators in myocardial ischaemia by Kevin Mullane (San Diego). Leucocytes and mediators derived from them seem to be more important in infarct development rather than in the early stages of acute myocardial
ischaemia when arrhythmias occur. Although the platelet accumulation ratio increases in parallel with neutrophil accumulation it appears that mediators derived from leucocytes, such as proteolytic enzymes, oxygen-derived free radicals and lipid mediators, are important in determining the ultimate size of an infarct. Neutrophils, like erythrocytes, must deform to pass through capillaries, but they are less capable of doing so than erythrocytes. Thus neutrophils are trapped during ischaemia and are not washed out during reperfusion. Complement activation is an early event and leukotriene production precedes neutrophil accumulation. It is also thought that changes in endothelial function may precede leucocyte adhesion.

Frances Williams (London) then discussed the contribution of neutrophils to changes in microvascular permeability. An anaesthetized rabbit model in which neutrophils labeled with $^{31}$In was used. Occlusion of the left coronary artery for 30 min followed by 3h reperfusion caused accumulation of neutrophils in the ischaemic region. Pretreatment of neutrophils with a monoclonal antibody to the CD18 glycoprotein (Mo Ab 60.3) reduced neutrophil accumulation. In animals depleted of neutrophils by mustine hydrochloride plasma protein leakage was still evident. The PAF antagonist WEB 2086 also failed to alter the ischaemia-induced increase in microvascular permeability. These results suggest that the altered microvascular permeability seen after ischaemia and reperfusion in rabbits is independent of neutrophils or PAF.

David Nicholson (Newhouse) discussed the influence of blood rheology in ischaemia. Since very little work has been done on rheological aspects in myocardial ischaemia most of the studies described by Dr Nicholson were performed on skeletal muscle, although it was felt that the results could also be applicable in myocardial ischaemia. Haematocrit is a major determinant of blood viscosity and when this was reduced to 25% by haemodilution, blood flow and tissue $PO_2$ were increased. Pentoxifylline and denbufylline improve blood flow by increasing filterability of both erythrocytes and leucocytes. This action is also shared by $\mu$M concentrations of the calcium channel blocker bepridil.

Moving away from the cellular aspects, Rudolph Riemersma (Edinburgh) presented work on the role of fatty acids in myocardial ischaemia. In a rat model infusion of sodium oleate depressed myocardial function and decreased ventricular fibrillation during ischaemia. Adding linoleic acid to the diet also has protective actions and the effect of a change in diet occurs very quickly. If, however, you reduce total fat too much, then you lose the antiarrhythmic effect of increasing the ratio of linoleic acid in the diet. He was also of the opinion that cholesterol was overestimated as a risk factor.

A series of short (10 min) free oral communications was followed, after lunch, by a session focused on the endothelium. Roger Wadsworth (Glasgow) described experimental studies on endothelium-derived relaxing and contracting factors. Sheep coronary arteries were examined under normal conditions, during hypoxia and in conditions designed to mimic ischaemia. During ischaemia an endothelium-dependent contraction equivalent to 10-12% of the maximum contraction induced by potassium was observed. This contraction was eliminated by the combined cyclo-oxygenase/lipoxygenase inhibitor BW755C, but was not altered in the presence of the leukotriene antagonist ICI198615. These results suggest that the endothelium-derived contracting factor released in this situation is a cyclo-oxygenase product. Under hypoxic conditions, prostacyclin release was reduced.

Christoph Thiemermann then gave a presentation on endothelin, an extremely potent peptide vasoconstrictor produced by endothelial cells. Adrenaline, angiotensin-II, vasopressin, thrombin, tPA and other substances can stimulate the release of endothelin-1. Endothelin itself releases several factors including atrial natriuretic factor, EDRF, prostaglandins, aldosterone and possibly PAF. The vasoconstrictor effect of endothelin is long lasting but can be prevented by calcium channel blockers. Since it is released by and stimulates the release of other factors it is likely that endothelin contributes to the regulation of vascular tone.

The final contribution was made by Alan Higgins (New Jersey) who talked about neutrophil
endothelial cell interactions. When neutrophils are stimulated with tumour necrosis factor, interleukin-1 (IL-1) or lipopolysaccharide (from endotoxin) there is an increase in neutrophil adhesion to endothelial cells whereas leukotriene B4 has no effect. Increased shear stress abolished the response to IL-1 which may explain why neutrophil accumulation only occurs in venules where shear stress is less. Advances in molecular biology have led to a better understanding of the role of certain molecules such as ELAM-1 and ICAM-1 in the regulation of endothelial cell adhesiveness. ICAM is normally present on cells, but ELAM is only expressed in response to stimulants. After 45 min of myocardial ischaemia in rabbits the expression of ELAM was detected.

After a quick summing-up by Andrew Henderson (Cardiff) the meeting closed. I would like to thank Professors Henderson, David Hearse and Alasdair Breckenridge for chairing the sessions.

Susan J Coker

BOOK REVIEW

Doppler Echocardiography, A Clinical Guide
P Wilde et al, Churchill Livingstone,
168 pages, 270 illustrations,
£29.95

This book should be considered as a primer for newcomers - students, nurses, technicians, doctors in the field of doppler echocardiography. Readers of this book are expected to be reasonably experienced in the imaging aspects of cardiac ultrasound and to understand the basics of cardiac pathology. The book includes a concise and simple discussion of both the physics of ultrasound and the examination techniques. Two chapters deal with the application of doppler echocardiography to diagnosis of both acquired and congenital heart diseases.

Alberto Volpi and Emma Riva

BOOK REVIEW

Clinical Applications of Two-dimensional Echocardiography and Cardiac Doppler
Arthur D Hagan & Anthony N DiMaria
Churchill Livingstone
600 pages, 400 illustrations
£50.00

This is a comprehensive and updated textbook of clinical echocardiography. This second edition includes the discussion of the role of doppler echocardiography in the many clinical settings of adult and congenital heart disease. Particular attention has been paid to the correlation, in normal and abnormal cardiac anatomy, between the various tomograms and the corresponding anatomic sections.

However, one problem of this book relates to the heavy emphasis placed on the latest advancements in the field of colour doppler imaging. Therefore, few colour images are given in the various chapters. Information provided by colour flow imaging are now believed to be essential for better accuracy in the recording of high velocity blood flow across stenotic and regurgitant valves (jets).

Moreover, given the wide application of transoesophageal echocardiography both for intraoperative monitoring and diagnosis of aneurysms and dissections of the thoracic aorta, too few pages have been devoted to this topic.

Alberto Volpi and Emma Riva

QUOTE OF THE WEEK

"Some 86,000 rats were disposed of during 1988 in poisoning, electrocution, force-feeding and brain-damage experiments - mostly without anaesthetic" (Time Out, July 25 1990, page 20).
A PERSONAL VIEW
This week: Myocardial Ischaemia-II

In the most recent issue of the BSCR Bulletin there appeared an article concerning how best to go about dealing with the seemingly insurmountable difficulties we encounter when considering the possibilities relating to various approaches to protecting the ischaemic myocardium. My honourable colleague from the opposite side of the fence put forward a set of proposals which clearly and abundantly fail, completely and utterly, to tackle the real issue which is, as all of us who have made a genuine and proper attempt to come to grips with the subject, as indeed we have, is a matter of an unfair and unequal distribution of blood to the areas which need it most, while at the same time, those privileged areas which have inherited an abundance, and indeed, superfluity of blood beyond their basic needs and requirements continue to maintain an unequal opportunity of excessive overindulgence. I will say one thing; it is unfair, uncaring and unrealistic to expect the vast majority of the ordinary working myocardium, which will have been systematically underperfused by years of neglect, to put up with an increase in flow, and furthermore, a pooling of blood in the minority of areas with a rich vascular supply. This pooling will unfairly tax the resources of the ischaemic region, and is therefore not, as the lady maintains, haemodynamic responsibility, but is nothing less than a pool tax. We have been sifting the alternatives to this travesty, and intend to announce our research policy document at our next full laboratory meeting. We can tell you now, however, that our policy will be a radical return to the past, with only 25% of those regions currently less well perfused than before being even less well perfused which rates better than the best projection of the alternative. Furthermore, we intend to support the most disadvantaged hearts with a safety net, and remove the minimum demand for output. No output means no work, and no work means no energy expenditure, so with a redistribution of blood supply from the working myocardium, haemodynamic equality will be restored with minimum cost to the better perfused regions. It's simple, it's radical, and it's a fair alternative.

MP Bedwelty

BRITISH HEART FOUNDATION June 1990 Fellowships

Dr G Sutherland
W. General Hospital, Edinburgh

Dr Jill Bishop
Natl. Heart and Lung Inst., London

Dr Fiona Lyall
Western Infirmary, Glasgow

Dr John Warren
Royal Postgrad. Med. Sch., London

Dr Jonathan Townend
Q. Elizabeth Hospital, Birmingham

Mr Timothy Hooper
Wythenshawe Hospital, Manchester

Miss Eileen McCall
University of Leeds

Mr Ashley Izzard
Leicester Royal Infirmary

Dr Albert McNeill
Ballymena

Dr Donald MacLeod
Royal Infirmary, Edinburgh

Senior Research Fellowship (3 years) £193,936
Intermediate Research Fellowship (3 years) £90,678
Intermediate Research Fellowship (3 years) £67,167
Intermediate Research Fellowship (3 years) £163,808
Junior Research Fellowship (2 years) £49,580
British/American Research Fellowship (1 year) £25,000
British/American Research Fellowship (1 year) £25,000
British/American Research Fellowship (1 year) £25,000
British/Dutch Research Fellowship (1 year) £25,000
British/Dutch Research Fellowship (1 year) £25,000

10 AWARDS TOTALLING.................................................. £690,169
BSCR CALENDAR OF EVENTS

1990

1 October Last date for receipt of abstracts for the Autumn meeting.
       Last date for submitting copy for the October Bulletin.

12 October Autumn Workshop: "Coronary Flow Regulation"

15 October Last date for submitting inclusions with the October Bulletin.

31 October Last date for receipt of postal votes for committee members and
       constitutional amendments.

6-7 December Autumn Meeting: "Physiology and Pathophysiology of E-C Coupling"

7 December Annual General Meeting.

1991

1 January Last date for receipt of subscriptions for 1991.
       Last date for receipt of abstracts for the Spring meeting.
       Last date for submitting copy for the January Bulletin.

15 January Last date for submitting inclusions with the January Bulletin.

10-11 April Spring Meeting: "Endothelium and Atherogenesis"

Advertisement

Düsseldorf University, West Germany

NMR-Spectroscopist

A postdoctoral fellowship position is available for a motivated individual with NMR
experience to join a group studying the energetics of isolated hearts and endothelial
cells in culture. The newly established NMR laboratory is well-equipped (wide bore
Bruker AMX 400) and there is a broad background in cardiac metabolism and cell
physiology.

The salary range will be from £23,000 to £25,000 per annum.

Please send application (CV, list of publications and names of two referees) to:
Dr Jürgen Schrader, Institute für Herz und Kreislaußforschung, Universität Düsseldorf,
Moorenstraße 5, 4000 Düsseldorf, W-Germany.