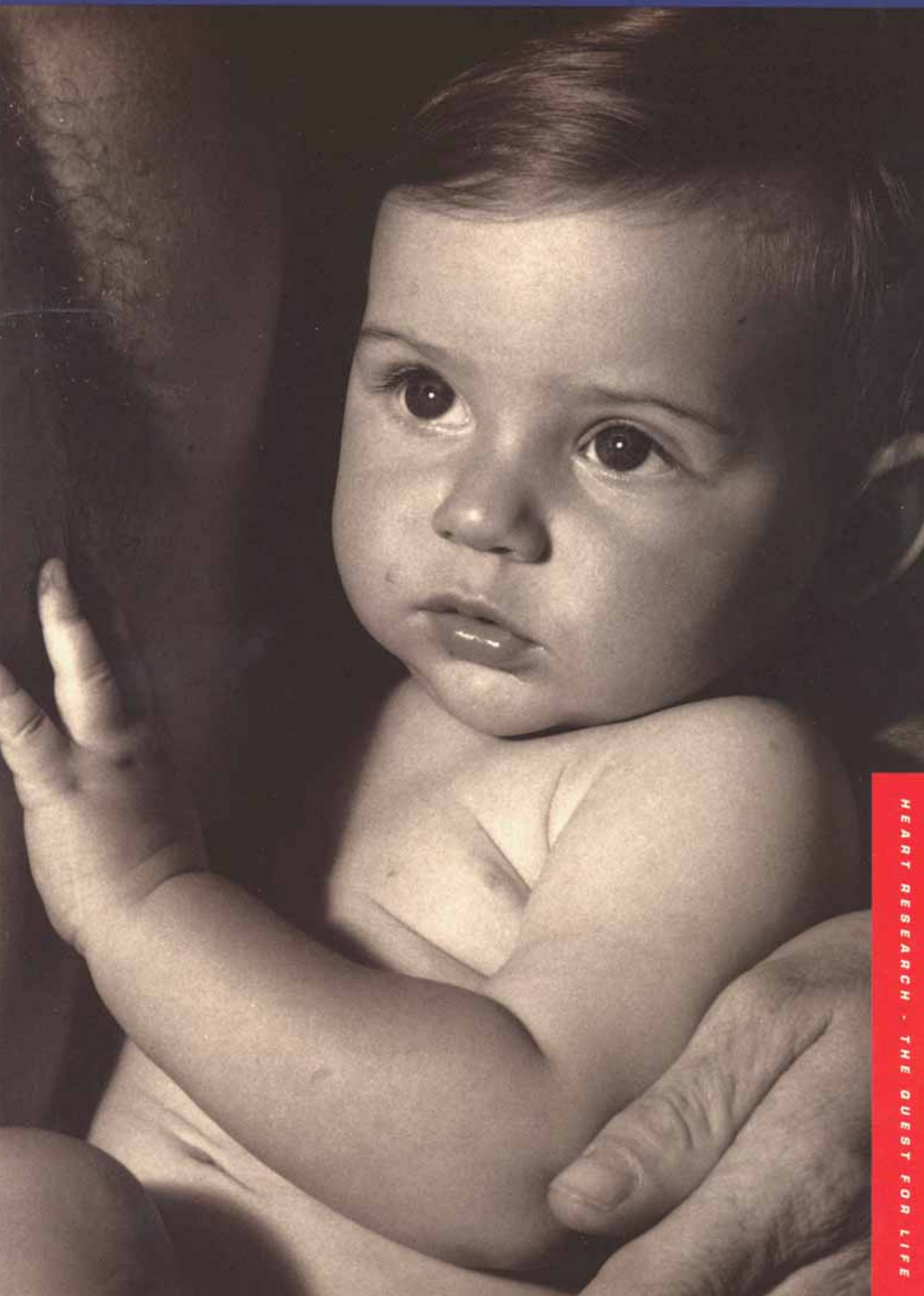
BAKER MEDICAL RESEARCH INSTITUTE ANNUAL REPORT 1991-1992



HEART RESEARCH - THE QUEST FOR LIFE

AIMS OF OUR RESEARCH

IN AUSTRALIA, 50% OF ALL DEATHS AND SERIOUS ILLNESS ARE DUE TO DISEASES OF THE HEART AND CIRCULATION.

MOST OF THEM ARE DUE TO HYPERTENSION (HIGH BLOOD PRESSURE) AND ATHEROSCLEROSIS (CLOGGING UP OF ARTERIES WITH FATTY CHOLESTEROL-LADEN PLAQUES) WHICH CAUSE STROKE, HEART ATTACK, HEART FAILURE AND KIDNEY FAILURE.

THE AIMS OF OUR RESEARCH ARE TO INCREASE UNDERSTANDING OF THE BASIC CAUSES OF HYPERTENSION AND ATHEROSCLEROSIS, TO USE THIS KNOWLEDGE TO HELP PREVENT HEART AND VASCULAR DISEASE IN THE COMMUNITY, AND TO IMPROVE MEDICAL AND SURGICAL TREATMENT.

THE BAKER MEDICAL RESEARCH INSTITUTE IS AFFILIATED WITH THE ALFRED GROUP OF HOSPITALS, AND WITH MONASH UNIVERSITY. THE INSTITUTE IS A WORLD HEALTH ORGANISATION COLLABORATING CENTRE FOR RESEARCH AND TEACHING IN CARDIOVASCULAR DISEASES.

CONTENTS

FOREWORD

In the seventies we had Mission Brown, and in the eighties Mission Statements. Both phrases touched a contemporary chord, decorative or organizational. Now, however, we feel free to use pastel colours, and to write a foreword as this without the almost inevitable 'Mission Statement' heading.

What has made the phrase dated is not what it means, but what it signifies. Missions are space talk, Cape Kennedy launches, Houston control. The challenge may continue but the buzz has gone.

What people meant by 'Mission Statement' however, continues to be relevant. Briefly, it covers four questions - for the Baker, for any institution...

- What do we do?
- Why do we do it?
- How do we do it?
- What do we hope to achieve?

What we do is medical research. Our research is mainly into the heart and circulation, but inevitably reaches out into other areas of health and disease. Much of it is bench top investigations, what makes blood vessels contract, how cells metabolize cholesterol, how to stop clots forming in blood vessels.

The questions are not new, but the answers are not in yet. It's investigator-initiated, curiosity-driven research of the classical type - hypothesis, testing, publication. When people think about such research they often ask how relevant it is, and whether many laboratories are doing the same thing. When almost half of us will die of heart disease it's fairly relevant; and the surprising thing is how little duplication there actually is, not how much.

We do research for a range of reasons. Wanting to find out about things is a very powerful motivation; doing something that may be very useful down the track is another. About a third of the research we do is directly applied to the clinical situation, first in the Alfred-Baker Medical Unit, and then elsewhere. Curiosity keeps people excited and patients keep them focussed.

How we do our research can be answered in a number of ways. We do it by using cells and tissues, experimental animals and human volunteers, chemicals and isotopes. We do it with a high degree of skill and commitment, probably more national and international than local recognition, and occasional bouts of frustration. We do it relatively on a shoestring, acknowledging the support we get, always hoping for more.

We will achieve a number of things. First, we contribute significantly to the world pool of knowledge. Second, we are suggesting and testing advances in the prevention,

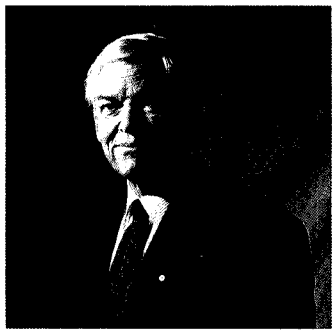
detection and treatment of disease. Third, we are training the doctors and scientists who will apply the discoveries made here and abroad.

And finally, the Baker - with its sister Institutes, and the various university and hospital departments - is testimony that there are things that Australia can do very well, and that we do medical research up with the best of them.

Our aspirations are at the benchtop, at the bedside, and in a wider sense in our society. To paraphrase Louis Pasteur: the laboratories of today are the cathedrals of tomorrow.

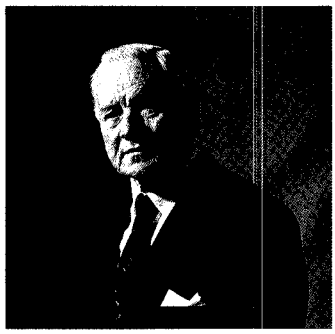
John Funder

BOARD OF MANAGEMENT



Mr J D Moir AM, President of the Baker Board of Management, is a consultant to Minter Ellison.

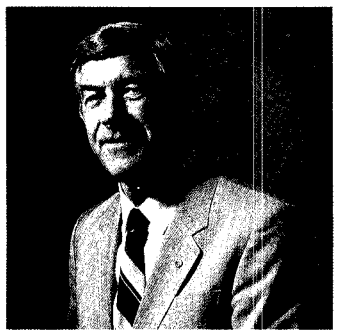
Mr D F Hogarth B Sc, Vice-President of the Baker Board of Management, was chairman of Directors of Kodak (Australasia) Pty. Ltd.



Mr J R Barcham Chair of the Baker Institute Development Committee, is a Company Director.

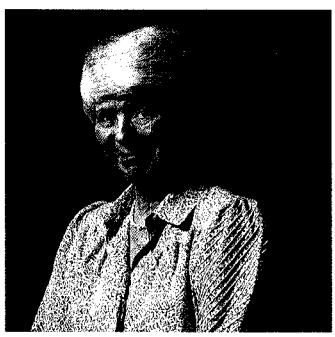
Professor J W Funder MD, PhD, FRACP, is Director of the Baker Medical Research Institute.

Mrs F S Grimwade OBE, B Sc, is Chair of Activities Committee.

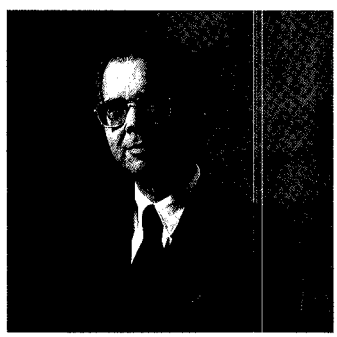


Mr W G Philip, AM, B Comm FCA, a former partner in Price Waterhouse, is currently Vice President of the Alfred Group of Hospitals and Director of several companies.

Professor R Porter B Med Sc, D Sc (Adel),MA, B Ch, DM (Oxon), FAA, FRACP, is Dean of the Faculty of Medicine, Monash University.



Mrs M Ross, SRN.



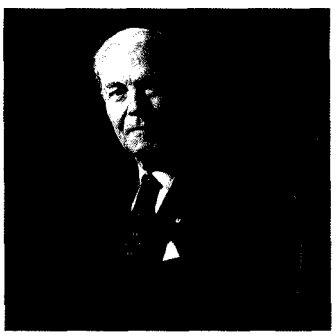
Professor G B Ryan MD, BS, PhD (Melb), FRCPA, FRACP, is Dean of the Faculty of Medicine, University of Melbourne.

Mr D Wittner is Chairman and Managing Director, Wittners Australia Ltd. (Retired 14.4.91)

Mr N O'Bryan BA, LLB, BCL, is a Partner of Minter Ellison, Solicitors - corporate and securities law and Commissioner, Law Reform Commission of Victoria. (Appointed 1.1.92).

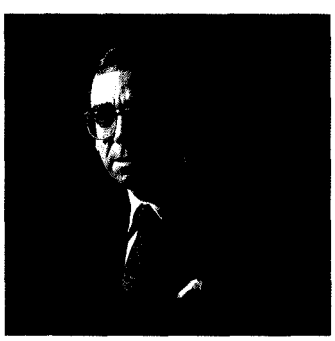
Dr G P Johnston is Deputy Managing Director and General Manager of Photographic Products Group, Kodak (Australasia) Pty. Ltd. (Appointed 15.4.91)

Sir Lawrence Muir VRD, LLB, FSIA, FAIM, is Patron of the Institute and former President of the Board of Management. He is a member of the Boards of a number of Australian and international companies.



Mr J C Habersberger AO, B Comm, is a Past President of the Institute and of Alfred Hospital. He was Joint Managing Director of Kodak (Australasia) Pty. Ltd. until his retirement in 1976.

Mr W A Kricker B Sc (Hons) (Syd), BE (Hons) (Syd), MBA (NSW), FIE (Aust), FAIM, FIDA, Secretary to the Board, is Chief Executive of the Alfred Group of Hospitals.



Mr D J Butler B Ec (Hons), FASA, CPA, Honorary Treasurer and Chairperson of Finance/Investment Subcommittee, is Group Executive, ANZ Banking Group.

Dr J Loy PhD, is First Assistant Secretary of the Health Advancement Division of the Department of Health, Housing and Community Services and Secretary of the National Health and Medical Research Council.

NOT PHOTOGRAPHED

Mr W D McPherson AO, AASA, was Director of BHP Ltd. and Tubemakers of Australia Ltd. He retired from the Board on December 31, 1991.

PRESIDENT'S REPORT

This Annual Report is a special report for several important reasons. It covers the first complete reporting period of 12 months from January to December 1991 of the Baker while under the stewardship of Professor John Funder. Secondly, it is a report designed to be read and understood by the intelligent general reader as well as the expert scientist. And thirdly it demonstrates that our Director and his staff have maintained, with the very generous help of our many supporters, the high standard of excellence in scientific research we have achieved in the past.

Any changes which have been made during the period under review are superficial changes of style rather than of substance. Eighteen months ago the Baker comprised a team of dedicated investigators, working to understand cholesterol and blood pressure and heart attacks. Today the dedication is the same, the aims are the same; the team has a new coach, and some additional new players.

And the goal is the same. The goal is to understand how the cardiovascular system works in health, and how to minimize and, if possible, prevent cardiovascular disease. What is clear - then and now - is that it is only on the basis of a thorough knowledge of what is normal that we can hope to understand how things go wrong, and only when we have such an understanding can we hope to prevent and treat disease rationally and effectively.

The Annual General Meeting, to be held on April 13 1992, marks the end of my term as President and as a member of the Board of the Institute. To have served from 1977 first as a member of the Business Advisory Committee to the Trustees, then as a Trustee before incorporation of the Institute by Act of Parliament in 1980, and subsequently as a member of the Board, Vice-President and President - has been both an honour and a pleasure. The Presidency - as my successor Don Hogarth will find - is at times a not undemanding role; and for their support I would like to thank my wife Lorraine, Don, as Vice-President, and my fellow Board members and colleagues on various Baker subcommittees.

In particular, I pay tribute to Mr. David McPherson and Mr. David Wittner who retired during 1991 and to the other retiring Board members, Mrs. Joan Grimwade and Mr. Roy Barcham. Though their contributions as Board members will be sorely missed, they will continue their work for the Institute in other ways. I am confident that those who follow in their footsteps, including Dr. Gerry Johnston and Mr. Norman O'Bryan who were appointed last year, will provide the same wise counsel and support to Don Hogarth and John Funder. I am also confident that I am leaving the ship in very good hands, with an excellent crew, all ready and able for the continuing voyage of discovery that is medical research today.

John Moir, AM

President, Baker Medical Research Institute, 1987-1992

DIRECTOR'S REPORT

At the outset, I would like to pay tribute to my predecessor as Director, Emeritus Professor Paul Korner. Paul is an outstanding cardiovascular scientist, who brought to the Baker a dedicated and talented team of younger colleagues. Through them, and with them, he created the Institute as we know it today, one of the key centres of cardiovascular research worldwide.

For three months of the 18 month period covered by this report Paul remained as Director; since then, he has commuted regularly from Sydney, and revelled in the chance to get back to science. Formally, his retirement was marked by a very successful Festschrift, organized mainly by Warwick Anderson, Jim Angus and Garry Jennings at which invited guests from overseas, interstate and locally made presentations. Informally, his retirement has been marked by a return to full-time research, freed from the shackles of administration. We look forward to a long and productive period of continuing collaboration.

At the end of October 1990 the Institute made a submission for Commonwealth funding for Capital Works, in response to an initiative announced in the Budget papers in August of the same year. A three stage development - new animal quarters, a tower block on the site of the present animal house, and refurbishment of the Western addition - was prepared in consultation with Stephenson and Turner, and in January 1991 Warwick Anderson and I took our case, and the scale model of the 'New' Baker, to Sydney for interview. It is with considerable pride and pleasure that I can announce that we have been awarded $4 million over three years (of a sum sought of $4.68 m), and that subsequently the Victorian Government has informed us of their intention to match these Commonwealth funds. The first tranche of funding is available in the 1992-3 financial year, meaning that development will be able to begin within the next six months.

At the same time as Paul Korner retired, Philip Barter accepted a Chair in Preventive Cardiology at the University of Wollongong, to be succeeded as Deputy Director by James Angus. Philip is an internationally acknowledged expert in the lipoprotein area; his appointment is testimony to his position in the field. The other departure of note was that of Julie Campbell, who accompanied her husband Gordon to a Chair of Anatomy in Queensland. Julie was an NHMRC Principal Research Fellow, the ranking woman research worker in the Institute, and an outstanding scientist; for all those attributes she has been, and will continue to be, sorely missed.

On the up side, however, are the recent arrivals. The first of the new brigade was Jerry Boublik in mid 1990, back from four years at the Salk Institute in San Diego, and now an R.D. Wright Fellow and Head of the Peptide Chemistry Laboratory. In January 1991

arrived Zig Krozowski and his Molecular Hypertension team, and Ian Smith and his Peptide Biology group. Dominic Autelitano (also an R.D. Wright Fellow), Karen Sheppard and Paul Komesaroff joined me in the Molecular Physiology Laboratory, and in October 1991 Michael Berndt arrived as a Principal Research Fellow and Head of the Vascular Biology Laboratory. Michael was a Wellcome Senior Research Fellow in Sydney until a year ago, and won the R.T. Hall prize for Cardiology in 1991. We are enormously pleased at having convinced him to move south, weather notwithstanding: Sydney's loss is Melbourne's, and more particularly the Baker's, gain.

In August 1991 the Institute Appointments and Promotions Committee met, and appointed Robyn Woods and Ian Smith Research Fellows, Zig Krozowski Senior Research Fellow, and Michael Berndt Principal Research Fellow. In addition, the committee promoted Murray Esler to Senior Principal Research Fellow, the equivalent of a Personal Chair in the NHMRC system. Murray has a truly outstanding record of contribution in that most difficult of areas, basic research in healthy human beings: testimony to his success is his being awarded both the Susman Medal of the College of Physicians, and the Wellcome Australia Medal, over the last decade.

While research clearly needs people of this calibre, the people need money to do their research. In 1991 the Baker received - and spent - $7.5 m of your money, private and public. For the first time, the contribution from the Baker Benefaction topped $1 m, reflecting an additional special grant of $100,000 to establish the incoming Director's laboratory. Glaxo Australia continued to support the Pharmacology Laboratory, for investigator-initiated, curiosity-driven research. The Victorian Government allocated $637,000 for the calendar year, and the Commonwealth Government $2.7 m total funding from the NHMRC. Additional funding came from contract research, from agencies such as the National Heart Foundation and the Victorian Health Promotion Foundation, from the Alfred Hospital Research Fund, Servier Laboratories, and from a series of very generous private and corporate donors.

For 1992 the picture does not seem as rosy. Academic salaries have had a long overdue increase; the increase is recoverable for those paid by NHMRC, but not from elsewhere. The demands on various trusts and benefactions have skyrocketed, and their income is down reflecting lower interest and dividend rates. Private and corporate support has held up relatively well - but not increased in line with outgoings. Finally, the National Heart Foundation has just announced across the board reductions of 10-15% in funds previously committed for 1992-94.

The latter part of 1991 has been largely occupied in preparing for our quinquennial review, to take place in the first half of 1992. As part of this process we will be seeking to have more of our current 'soft-money' scientific positions supported by NHMRC from 1993 onwards. So while 1992 may be a year of restraint and belt-tightening, 1993 may represent the light at the end of the tunnel.

One of the unique features of the Baker is the existence of the Alfred-Baker Medical Unit, the each-way conduit between the benchtop and the bedside. Proof of the closeness of the interaction is that Garry Jennings, Director of the ABMU, and Alex Bobik (Associate Director, Laboratories, ABMU) constitute the executive of the Baker with Jim Angus, Warwick Anderson (Associate Director, Baker Institute) and myself. For their work and support over the past eighteen months I, as a new Director, owe them a special vote of thanks. To the Board of the Baker, who give so generously of their time and expertise, and to all the people who work at the Baker - scientific and administrative, medical and support staff - the community as a whole owes a particular vote of thanks. They are people who work with immense dedication for the benefit of us all.

John W. Funder
Director, B.M.R.I.

LABORATORY REPORT

THE SYSTEM

When we think about the heart and blood vessels, we think of pumps and pipes. The blood vessels carry oxygen and nutrients to the tissues, and take carbon dioxide and waste products away; and the heart is the pump that keeps the whole thing moving.

So if the pump keeps pumping, and the pipes stay open, everything should be right, OK? In one sense yes is the answer: if the pump stops, or the pipes are blocked, the body cannot continue to function: this is how people die, of heart attacks or strokes.

In another sense, the answer is clearly no. The pump and pipes are not static, running at a fixed level, but change in response to exercise or cold: how? We are not born with an adult pump and set of pipes, but as we grow older they develop and continually remodel: how? The pipes carry blood, which is not only thicker than water, but has cells and salts and clotting factors, all of which affect the way it works: how?

These are three questions: there are dozens more. What they show is in some small part the complexity of the cardiovascular system in health - and the parallel complexity of what can go wrong in disease.

COMMUNICATIONS

Complex systems need communication systems if they are going to work. Think of your very first clockwork train; round and round it went, same speed, same direction, until it stopped - and that was that. Now think of a real railway system, with its array of signals and points and controls. Without communications it would never work - and even the best communications are no guarantee that it will always work absolutely perfectly forever.

The scientists and doctors who work on the cardiovascular system at the Baker are largely communication biologists. They would normally think of themselves as cardiologists, or pharmacologists, or physiologists, or biochemists; but almost to a person they work on the complex system of signals (and the signal-receiving mechanisms, or 'receptors') that make the cardiovascular system the dynamic, flexible, responsive system that it is.

THE BRAIN

In one sense, it makes sense to start with the head. Geoff Head took over the Neuropharmacology Laboratory upon Paul Korner's retirement as Director of the Baker. We think of the brain and nervous system primarily in terms of thinking, and sensation, and movement - but it's also very much involved in control of the cardiovascular system. What Geoff and his team do is to study the connections between various cells in the hindbrain, just above where the spinal cord begins, that are known to be involved in blood pressure control.

NEUROTRANSMITTERS

One of the fascinating things about the body is the way certain signals - for example, peptides (small proteins) like angiotensin and vasopressin - can raise blood pressure in different ways. Both act as neurotransmitters (signals between nerve cells) in the hindbrain, activating nerves that run down from the brain to cause blood vessels to

WHEN TONY PETROU FIRST CAME TO THE BAKER RISK REDUCTION CLINIC HIS BLOOD PRESSURE WAS CLEARLY TOO HIGH, AND HIS CHOLESTEROL TOWARDS THE UPPER LIMIT OF

NORMAL. OVER THE NEXT YEAR, HE CHANGED HIS DIET AND STARTED A REGULAR EXERCISE PROGRAM. HIS WEIGHT HASN'T CHANGED, BUT HIS BLOOD PRESSURE HAS FALLEN NICELY, AND HIS OVERALL RISK OF A CORONARY IS NOW JUST HALF WHAT IT WAS A YEAR BEFORE.

constrict; both also work as hormones, circulating in the blood, acting directly on the blood vessel wall and similarly causing it to contract. The neuropharmacology laboratory explores how these nerve cells in the hindbrain talk to one another, and the ways in which various drugs can mimic or interrupt this communication.

EXERCISE

In addition, they look at the other side of the coin - for example, how having high blood pressure affects the nerves involved in the reflex control of blood pressure. If we did a lot of stationary bicycling in an Army greatcoat, we might call it a reflex to turn down the central heating. In fact, it's obviously a conscious decision - but the feedback loop principle is the same. And in the Alfred Baker Medical Unit - of which more later - we actually use stationary bicycles to test how people with heart disease perform in response to the demands of exercise.

BACK TO THE BRAIN

Another group within the Institute concerned with the brain and blood pressure control is the Human Autonomic Function Laboratory, led by Murray Esler. Murray measures the spill over of the neurotransmitter noradrenaline and its by-products into the blood draining the brain via the jugular vein. By this technique, he has shown that patients with hypertension (high blood pressure) release abnormally high amounts of noradrenaline from the brain.

STRESS

He has also shown, by similar techniques, that certain stimuli (difficult mental arithmetic, cigarettes) but not others (coffee, strenuous exercise) cause a marked stimulation of the nerves to the heart. Since a burst of noradrenaline may trigger abnormal heart rhythms, this may indicate why postmen very rarely die of heart attacks, compared for instance with accountants.

KIDNEYS

But blood pressure is not only hearts and minds; the commonest curable form of hypertension is caused by a lowered blood supply to the kidney. The kidney is a powerful organ, and 'wins' in terms of blood pressure, by making sure that it gets enough blood even if the pressure elsewhere becomes dangerously high. Warwick Anderson in the Renal Physiology Laboratory has documented the changes in blood pressure and sodium and various hormones that follow experimental lowering of blood flow to the kidney. In other cases of hypertension, the kidney gets a normal amount of blood, but responds abnormally: Warwick has therefore also looked at how various hormones and local signals affect sodium retention by the kidney, in a cause-and-effect sense the other side of the mirror in terms of the kidney and blood pressure.

NERVE ENDINGS

So far we have talked about signals that make nerves fire off or blood vessels contract. What Sue Luff does, in the Electron Microscopy Laboratory, is to look at where the nerves end on the blood vessels, to try and puzzle out how the noradrenaline gets across the gap

TWO PEOPLE FROM DIFFERENT BACKGROUNDS WHO STARTED THEIR RESEARCH CAREERS AT THE BAKER INSTITUTE IN THE RENAL LABORATORY IN 1982 ARE ROBYN WOODS, ON THE LEFT AND DEBORAH RAMSAY. DEB IS A

HIGHLY EXPERIENCED AND SKILLED ANIMAL TECHNICIAN, WHO IS NOW THE OFFICER IN CHARGE OF THE BIOLOGICAL RESEARCH UNIT WHERE SHE SUPERVISES THE GROUP OF PEOPLE THAT LOOK AFTER OUR EXPERIMENTAL ANIMALS. ROBYN IS AN NHMRC RESEARCH FELLOW, A WHOLE ANIMAL PHYSIOLOGIST WHOSE CARDIOVASCULAR-RENAL RESEARCH INVOLVES DAILY CONTACT WITH THE ANIMAL CARE PEOPLE.

from the nerve to excite the muscle. Different arteries respond (by constricting or dilating) to different demands (for example, the rat tail artery to heat, and the arteries in the gastrointestinal tract to food), so that Sue looks at the neurovascular (nerves and blood vessels) architecture in various tissues, to work out the similarities and differences across different organs. Michael Lew, in the Pharmacology Laboratory, is asking a different but related question: how molecules arriving at the smooth muscle cell as hormones may have different access, and different effects, from the same molecules released from nerve endings.

GROWTH

But blood vessels do things other than just contract; for example, they grow. When Rod Dilley, from the Morphology Laboratory, looks at small arteries under the light microscope, he finds that those from genetically hypertensive rats are thicker, more muscle-bound, than those from normal rats - even before their blood pressure goes up. It's easy to imagine how the blood vessel wall might hypertrophy (get thicker) in response to high blood pressure, just like your biceps and triceps get bigger in response to exercise; but in this instance the hypertrophy precedes rather than follows the elevation in blood pressure.

GENES

How can this be? In studies on similarly genetically hypertensive rats, Alex Bobik from the Biochemical Pharmacology Laboratory has shown that there are very marked differences in the growth responses to TGF1ß (Transforming Growth Factor 1ß) between heart and vascular smooth muscle cells from normal rats and those with high blood pressure. TGF1ß is not a hormone (released into the bloodstream) or a neurotransmitter (released from nerves); it's a local signal, carrying information from one cell to its neighbours, and responsible (with a whole host of other factors) for coordination in growth and development of various tissues.

RECEPTORS

Whatever the response - making a nerve fire off, causing a blood vessel to constrict, inducing neighbouring cells to grow - the message has to be received; for each signal there has to be a receptor. Work on receptors within the Institute essentially covers the waterfront. There are laboratories which study receptors for neurotransmitters, for peptide hormones, for steroid (cholesterol derived) hormones, for locally acting control factors, for lipid (fat) particles in the blood, and even for whole cells.

PHARMACOLOGY

The Pharmacology Laboratory, under the direction of James Angus, is involved in a number of areas. Among these are a series of studies on receptors for neurotransmitters like noradrenaline, acetycholine and serotonin. As a pharmacology laboratory, the group are particularly interested in drugs that can bind to these various receptors, and either activate or block them. Recently, for example, the laboratory has done a lot of work on the

CHOLESTEROL IS REMOVED FROM THE BODY BY 'RECEPTORS', WHICH PICK UP THE CIRCULATING LIPID DROPLETS AND TRANSFER THEM INSIDE THE CELLS OF THE BODY, LIKE THE HOISTS THAT

LIFT SAILING BOATS OUT OF THE WATER. THE ANALOGY IS APT FOR ROBERT DEBENHAM, A YACHTSMAN AND A LONG-TIME INSTITUTE PATIENT. HE HAS HETEROZYGOUS FAMILIAL HYPERCHOLESTEROLAEMIA, BECAUSE HE MAKES ONLY HALF THE NUMBER OF RECEPTORS FOR CHOLESTEROL WHICH THUS RISES TO HIGH LEVELS IN HIS BLOOD.

serotonin blocker Sumatriptan, which has revolutionised the treatment of migraine. Migraine is not 'just a headache', but a constellation of signs and symptoms following inappropriate dilatation (the opposite of constriction) of blood vessels to the brain - so it's an entirely logical and appropriate area for cardiovascular pharmacologists to find themselves in.

STEROIDS

In the Molecular Physiology Laboratory, the focus is on receptors for steroid hormones. Steroids are not just the pill, or Ben Johnson; those of particular concern to the Institute come from the adrenal gland, and are concerned with salt retention (aldosterone: mineralocorticoid) and stress (cortisol: glucocorticoid). Dominic Autelitano and Karen Sheppard work on the pituitary gland, to see what other signals from inside the cell affect glucocorticoid receptors, in addition to cortisol itself.

SALT AND WATER

Paul Komesaroff, in the same laboratory, has cloned and sequenced the mineralocorticoid receptor from the first patient in the world with pseudohypoaldosteronism, PHA for short. People with PHA, as the name implies, seem to be low on aldosterone, and thus lose salt and water. The real problem, however, is not in the signal, but a defective receptor, so that they cannot maintain their blood volume even with very high levels of aldosterone.

LIPIDS

The neurotransmitters and steroid hormones are relatively small molecules, and their receptors have been more or less identified. High density lipoprotein (HDL), on the other hand, is a very complicated thing; a core of lipid (fat), including cholesterol and triglycerides, surrounded by a coating of protein. Think of a Jaffa, where the chocolate represents the lipid (which it is, close enough), and the orange casing the protein (which it isn't: it's actually largely sugar).

LIPOPROTEINS

HDL is a 'good' lipid in terms of risk of heart attacks, and low density lipoprotein (LDL) is 'bad', at least in terms of large population studies. LDL is low density because it is bigger - the same sort of thickness of protein coat around much more lipid; imagine a Jaffa as big as a golf ball. Ten years ago, the receptors for LDL were isolated, purified and cloned in Dallas - an achievement recognized by a subsequent Nobel prize.

LIPOPROTEIN RECEPTORS

For some years, there was doubt whether HDL had a similar, particular receptor. From the work done by Noel Fidge in the Lipoprotein and Atherosclerosis Laboratories it is clear that there is an HDL receptor. Noel and Alana Mitchell have purified a part of the receptor, two distinct peptides, as stage one in a long term study on what the HDL receptor is, and how it operates in the protective effect of high HDL levels in blood.

Even if LDL and HDL particles are hundreds of times bigger than neurotransmitters or steroids, they are small beer when compared with cells. Michael Berndt, of the Vascular

LIZ DEWAR IS A CARDIAC TECHNOLOGIST IN THE ALFRED BAKER MEDICAL UNIT, AND TAKES PATIENTS (AND NORMAL VOLUNTEERS: IT'S IMPORTANT TO HAVE NORMAL, BASELINE DATA)

THROUGH THEIR ECHOCARDIOGRAMS AND 24-HOUR BLOOD PRESSURE MONITORING AND TREADMILL TESTS. LIZ HERSELF HAS AN ALMOST INEXHAUSTIBLE TOLERANCE FOR EXERCISE - RUNNING, AEROBICS AND SWIMMING....TO THE EXTENT THAT IN 1991 SHE WON THE NATIONAL HEART FOUNDATION'S ONE HOUR DISTANCE SWIM FOR WOMEN.

Biology Laboratory, studies how one cell recognises and attaches to another. A bit basic, you might say, and a fair distance from pumps and pipes: it might come as a surprise, then, that Michael won the R.T. Hall prize for Cardiology in 1991.

BLOOD CLOTS

What Michael studies is the receptors through which platelets bind to the cells lining blood vessels. Platelets are the circulating Kamikaze cells involved in blood clotting - physiologically, when we cut ourselves, and pathologically, when a clot forms over an area of atheroma in one of the coronary arteries supplying the heart muscle.

So it is cardiovascular; but Michael also studies how white blood cells bind to the cells lining blood vessels, which has to do with inflammation and possibly how cancer cells spread via the blood. Like Jim Angus and migraine, there's nothing that's just cardiovascular; in 1992 biology really is becoming a seamless garment, and medicine has the chance of being truly holistic.

ENZYMES

So far we have talked about signals and receptors, molecules and particles and cells. In terms of these sorts of communication systems there are two other areas we need to address. One is enzymes, and the way in which they can modify signals between the time they are released and their site of action. The other is what happens after a signal has bound to a receptor, after the key has gone into the lock.

DIAGNOSIS

Enzymes make things go much faster - in digestion, in washing powders, whatever. They can help break down large molecules into their constituent parts, or make complex molecules out of simple building blocks. After a heart attack the dead cells release their content of intracellular enzymes into the blood, where they can be measured, one of the ways of distinguishing a heart attack (myocardial infarct, coronary occlusion) from angina.

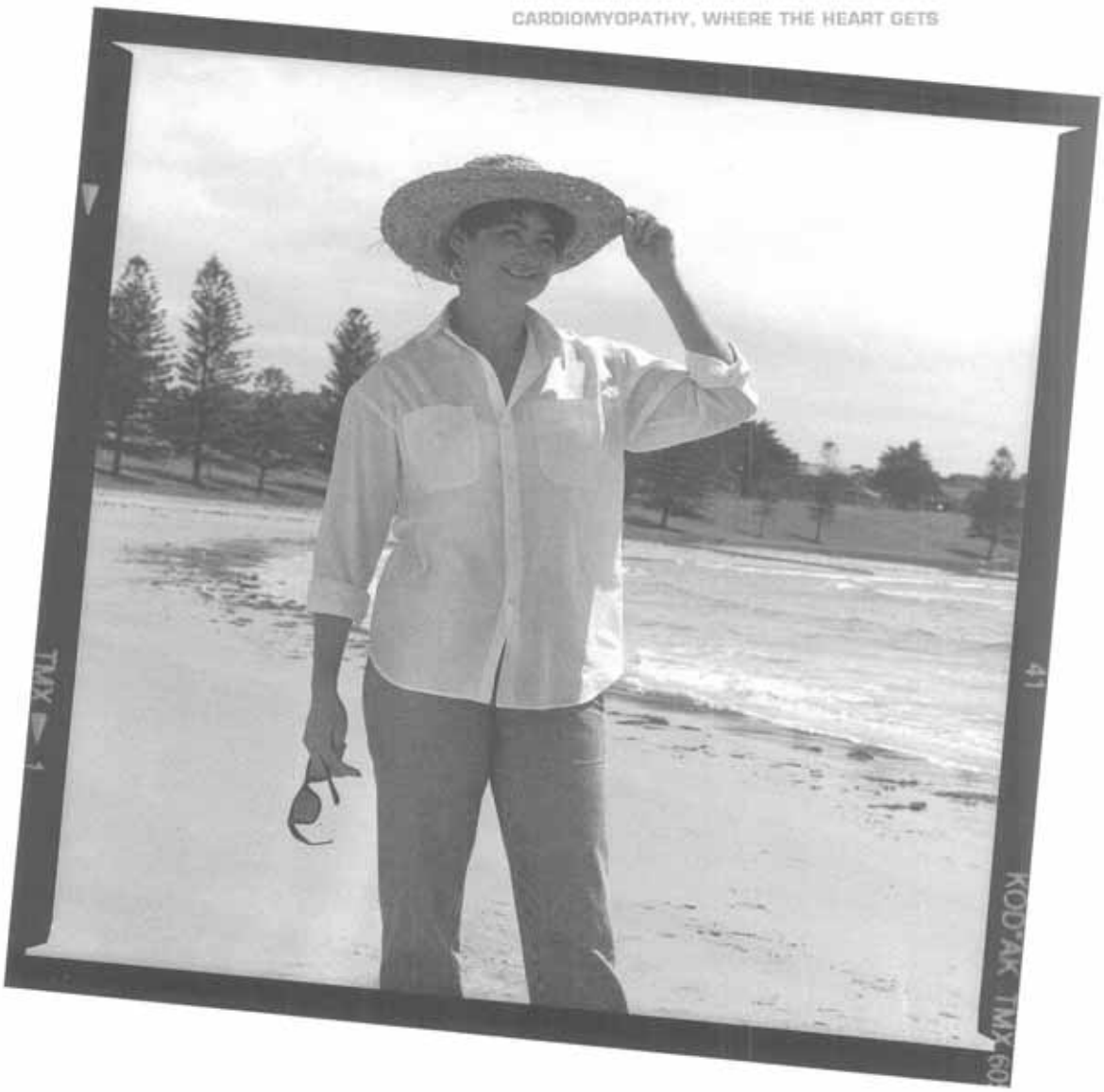
TREATMENT

Enzymes are also involved in treatment; for example, currently probably the most effective treatment of high blood pressure involves taking ACE (for angiotensin converting enzyme) inhibitors. Angiotensin is made by a series of enzymatic cleavages from a circulating protein secreted into the blood from the liver. Alex Bobik and Jane Black have shown that it not only makes smooth muscle cells contract, but long-term it makes them grow. The final step in getting the active signal is to clip off the last two amino acids of the inactive immediate precursor, which gives a peptide that constricts blood vessels and raises blood pressure. This is the step that ACE inhibitors block - so enzymes are important in cardiovascular medicine, as well as signals and receptors.

ATHEROSCLEROSIS

Like receptors, enzymes are the focus of attention for a number of groups at the Institute. In the Molecular Biology Laboratory, Alana Mitchell and her colleagues have cloned the enzyme hepatic lipase from rabbit liver. Rabbits fed the right (or rather, the wrong) diet are very prone to atherosclerosis, like humans; rats are not. Rabbit hepatic

SHARON McCOURT LIVES AT TORQUAY, BUT HER HEART IS IN BICYCLING ALL OVER THE BELLARINE PENISULA. SHARON HAD A HEART TRANSPLANT 16 MONTHS AGO, FOR A HEREDITARY CONDITION CALLED HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY, WHERE THE HEART GETS



MORE AND MORE STRAINED TRYING TO PUMP THE BLOOD. HER STORY SHOWS WHAT CAN BE DONE - LOTS OF EXERCISE, A NORMAL LIFESTYLE - AND YOU DON'T HAVE TO LIVE ON THE HOSPITAL'S DOORSTEP.

lipase activity is only a tenth that in the rat, suggesting that what the enzyme does may be very good news in terms of atherosclerosis. Now that the enzyme is cloned, Alana can study what turns it on and off - and thus how it may protect us, and not just rats, from the sort of food we eat.

HYPERTENSION

Atherosclerosis is one thing, and high blood pressure another. Taking their lead from a very lethal (and mercifully very rare) form of hypertension, Zig Krozowski and his team in the Molecular Hypertension Laboratory are cloning and studying the family of enzymes - 11beta hydroxysteroid dehydrogenases, or 11HSD for short - which keep glucocorticoids out of receptors in certain tissues. In patients lacking the enzyme, glucocorticoids inappropriately activate mineralocorticoid (salt-retaining) receptors, so that the patients commonly die of malignant hypertension as teenagers. What we don't know is the role that the enzyme plays in the normal control of blood pressure, and 'ordinary' hypertension; and the sorts of studies Zig is doing (and some of those done by the Director) may provide answers to these questions.

HORMONES

In the Peptide Biology Laboratory, Ian Smith and his coworkers are asking even more basic questions. The heart has very high levels of an enzyme which amidates peptides, but no known amidated peptides. Amidation makes peptides very resistant to further enzymatic degradation, and many peptide hormones are amidated so that they are not immediately chewed up by enzymes in the blood. The question is obvious: is the heart making an amidated peptide hormone for release into the blood?

HEART HORMONES

You may not think of the heart as an 'endocrine' organ, like the thyroid gland or the pancreas, since it was only ten years ago that the first 'heart hormone' was described (atrial natriuretic peptide, or ANP). Robyn Woods, in the Circulatory Control Laboratory, has shown ANP to have previously unexpected actions, including constriction of the blood vessels supplying the intestines. Robyn has done her studies *in vivo*, whereas the previous received wisdom was from in vitro ('in glass', or test-tube) studies. What she has found underlines the importance of doing things both ways - of which, more later.

LOCAL COMMUNICATION

Another new signal is nitric oxide. Not nitrous oxide (laughing gas), or nitric acid, but a single atom of nitrogen and a single atom of oxygen, written NO; yes, NO. Over ten years ago Tom Cocks and Jim Angus provided the first convincing evidence for a factor which relaxed vascular smooth muscle, and which came from the endothelial cells lining blood vessels, separating the blood itself from the vascular smooth muscle. Subsequently this has been shown to be NO, and Tom and his group are hard at work blocking the enzyme that makes NO from the amino acid arginine (enzymes, again), and testing the effects on how different blood vessels react.

JIM ANGUS, ON THE RIGHT, IS THE DEPUTY DIRECTOR OF THE BAKER, CHAIRMAN OF THE NHMRC GRANTS COMMITTEE, AND AN OUTSTANDING PHARMACOLOGIST. MURRAY ESLER IS DEPUTY DIRECTOR (HYPERTENSION) OF THE ALFRED BAKER MEDICAL UNIT, A SENIOR

PRINCIPAL RESEARCH FELLOW OF NHMRC, AND WINNER OF THE SUSMAN AND WELLCOME AWARDS FOR MEDICAL RESEARCH. BOTH ARE, IN THEIR 'SPARE' TIME, KEEN FISHERMEN: YOU CAN DO RESEARCH TWELVE HOURS A DAY, FIVE DAYS A WEEK, WITH A LOT OF READING AND WRITING OVER WEEKENDS - BUT SOMETIMES, WHEN THE SNAPPER ARE RUNNING, AN UNDERSTANDING FAMILY WILL FORGIVE YOUR GETTING HOME EVEN LATER THAN USUAL.

Hormones like ANP, or neurotransmitters, or local signals like NO occupy a receptor in their target cells, like a key fitting into a lock. Normally, when we put a key into a lock, nothing happens; we have to turn it, and then things start to move inside the lock, catches come loose, doors open. Elizabeth Woodcock, in the Cellular Biochemistry Laboratory, studies signal transduction, the ways in which the receptors on the surface of the cell communicate with the rest of the cell to pass on the message from signal to the cell.

DOWN THE LINE

The same signal may have different messages for different cells - grow, contract, secrete, whatever; are the same intracellular 'second messengers' involved? The same signal may have different messages for the same cell; how are these second messages distinguished? So Liz compares heart cells and blood vessel cells and cells from the adrenal gland, and measures what seems to be an ever-growing complexity of intracellular signals under various conditions - because if we don't know what makes it work normally, we've got no baseline against which to assess the abnormal states we find in disease.

CHANNELS AND IONS

One of the things that happens as a result of 'second messenger' action is that cells change - they grow, they contract, they secrete. These are often major changes, needing orders to be heard throughout the cell, and close coordination of a number of cellular processes. To get something like this going, cells rely on differences in concentrations of various common ions - sodium, chloride, calcium - between inside and outside the cell. When things are quiet, the concentration gradients are established; when the signal comes and binds to its receptor, one of the things second messengers commonly do is to open up channels in the cell membrane - and sodium floods in, or potassium drains out, and so on, depending on the particular channel activated.

CONOTOXIN

Grant McPherson and his team in the Pharmacology Laboratory study these 'ion channels'; they plug them up in various ways, and work out whether they can affect responses in one tissue (e.g. blood vessels, to lower blood pressure) without getting dangerous side effects by blocking the same channels in other tissues. Although Jerry Boublik's main activity in the Peptide Chemistry Laboratory is on neuropeptide Y, a physiological peptide with marked effects on blood pressure, he is also making a series of analogues of conotoxin, made by the fish-eating sea snail to paralyze its prey. Conotoxin blocks calcium channels; Christine Wright and Jim Angus have shown that conotoxin lowers blood pressure in rabbits, and we are all looking forward to the experiments with the custom-altered analogues, to see whether they have increased potency with even lower side effects.

BENCHTOP TO BEDSIDE

Side effects sounds very clinical, and indeed it is. The process of discovery is important in itself, of knitting up the different and often divergent strands, of finding out

MAX THATCHER HAS HAD A LONG INVOLVEMENT WITH THE BAKER. INITIALLY HE WAS SEEN IN THE CLINIC FOR HIS HIGH BLOOD PRESSURE, WHICH IS NOW WELL UNDER CONTROL. SUBSEQUENTLY, MAX HAS BEEN IN AND OUT OF THE PLACE - NOT AS A PATIENT, BUT AS A

VOLUNTEER - FIRST FOR A STUDY ON THE BENEFICIAL EFFECTS OF SALT RESTRICTION AND EXERCISE IN TERMS OF BLOOD PRESSURE, AND MORE RECENTLY IN MORE COMPLICATED STUDIES ON HOW WE REGULATE BLOOD PRESSURE. GOOD RESEARCH NEEDS TO BE DONE ON CELLS AND RATS AND HUMANS: AND ALL OF US OWE A DEBT OF GRATITUDE TO MAX AND HIS FELLOW VOLUNTEERS, FOR WHAT THEY ARE DOING FOR THE WIDER COMMUNITY.

how things actually are. For most of these studies we study cells and tissues from rats and rabbits, although some baseline physiological studies (like many of those Murray Esler does) use normal human subjects (volunteers, of course). What makes the Baker special, however, is the way in which these baseline or basic studies can be applied to the clinical situation, thanks to the Alfred-Baker Medical Unit. Conversely, the ABMU can help focus the bench-top investigators on real clinical puzzles, or seek their assistance in solving clinical problems.

POPULATION STUDIES

For example, as a complement to the basic work on HDL receptors and hepatic lipase done by Noel Fidge and Alana Mitchell, Garry Jennings and Tony Dart study human populations. They check their diet, measure their cardiac function and analyze their blood, to try and establish additional 'indicators' for heart disease; currently, we know probably just over half of the 'risk factors' (smoking, cholesterol, etc). The Baker is recognized by the WHO as Australia's first WHO Collaborating Centre for Research and Training in Cardiovascular Diseases, acknowledgement of the role the ABMU plays in training young medical registrars as well as its research contributions. It is also supported by the Victorian Health Promotion Foundation, and the Australian Better Health Program, for some of its community studies.

SURGERY

A final example of research at the interface between the laboratory and the clinic is the sort of investigation Frank Rosenfeldt does in the Cardiac Surgery Research Laboratory. He has worked out, using animal hearts, a much better way of preserving hearts for transplants. Result: more flexibility, better recovery of function and lower costs for heart transplants. Frank reported that the veins used for bypass surgery often spasm: Jim Angus came up with an anti-spasm cocktail, which Frank tested successfully in the laboratory, and which is now in routine use in the human coronary artery graft surgery program.

THE BAKER

Put it all together, and what have we got? To someone outside medicine or medical research, the Baker is probably a bewildering kaleidoscope of people, long words, excitement and equipment, activity and hope. The present brief account may have generated rather than dispelled such a kaleidoscopic picture; if this is the case, apologies are due to readers and research workers alike - and we have covered a tiny fraction of what the various laboratories do. Other research workers can appreciate the breadth and extent of the ongoing studies by a glance at the Institute publication list on pages 29 to 33. For our general readers, it is our hope that this very brief account of what we do may serve as an introduction to why we do it, and what we hope to achieve - for ourselves as scientists and doctors, and for the wider community that supports the research we do.

JO CHALLENOR IS ON THE THRESHOLD OF HER CAREER IN MEDICAL RESEARCH. IN 1990, SHE WAS JUDGED TOP STUDENT IN PHARMACOLOGY

AT MONASH, AND IN 1991 TOP HONOURS YEAR STUDENT IN THE SCIENCE AND MEDICAL FACULTIES. JO SPENT 1991 IN GRANT MCPHERSON'S LABORATORY WORKING ON THE CHANNELS IN SMOOTH MUSCLE CELL MEMBRANES AND THE WAY THESE CHANNELS AFFECT BLOOD PRESSURE, AND THIS YEAR SHE STARTS HER PhD. IF WE ARE EVER GOING TO BE A CLEVER COUNTRY, WE NEED PEOPLE LIKE JO.

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BAKER INSTITUTE PUBLICATIONS (1991)

1. Ahima R, Krozowski Z and Harlan R. Type I corticosteroid receptor-like immunoreactivity in the rat CNS: distribution and regulation by corticosteroids. J Comp Neurology 313:1-17, 1991.
2. Allan C, Tetaz T and Fidge N. Epitope mapping of apolipoprotein A-I using endoproteinase cleavage and monoclonal antibodies in an enzyme-linked immunosorbent assay. J Lipid Res 31:595-601, 1991.
3. Allen RS, Boublik JH, Houger L, Rivier JE and Brown MR. Neuropeptide Y radioimmunoassay: characterization and application. Clin Exp Pharmacol Physiol, in press.
4. Anderson WP, Woods RL and Gao Y. Renovascular hypertension: information from experiments using conscious dogs. Clin Exp Pharmacol Physiol 18:29-32, 1991.
5. Angus JA. Symbiotic relationship between a research institute and a pharmaceutical company: the Baker Institute/Glaxo Australia story. Clin Exp Pharmacol Physiol 19:67-71, 1992.
6. Angus JA, Broughton A, Cocks TM and McPherson GA. The acetylcholine paradox - a constrictor of human small coronary arteries even in the presence of endothelium. Clin Exp Pharmacol Physiol 18:33-36, 1991.
7. Angus JA, Broughton A and McPherson GA. Membrane potential and contractility responses to acetylcholine and other vasoconstrictor stimuli in human small coronary arteries. In: Resistance Arteries, Structure and Function, (eds) MJ Mulvany, C Aalkjaer, AM Heagerty, NCB Nyborg and S Trandgaard, Amsterdam, Elsevier, pp. 255-260, 1991.
8. Angus JA, Ward JE, Smolich JJ and McPherson GA. Reactivity of canine isolated epicardial coronary collateral arteries: relation to vessel structure. Circ Res 69:1340-1352, 1991.
9. Ashman LK, Aylett GW, Mehrabani PA, Bendall LJ, Niutta S, Cambareri AC, Cole SR and Berndt MC. The murine monoclonal antibody, 14A2.H1, identifies a novel platelet surface antigen. Brit J Haematol, in press.
10. Bajt ML, Ginsberg MH, Frelinger AL, Berndt MC and Loftus JC. A point mutation in beta3 results in alphaIIbbeta3 defective in ligand binding. J Biol Chem, in press.
11. Bobik A, Grooms A, Little PJ, Cragoe EJ Jr. and Grinpukel S. Ethylisopropylamiloride-sensitive pH control mechanisms modulate vascular smooth muscle cell growth. Am J Physiol 260:C581-C588, 1991.
12. Bobik A, Little PJ, Grooms A and Grinpukel S. Amiloride sensitive pH control mechanisms modulate vascular smooth muscle growth. Relationship to protein synthesis during late G1 phase. Am J Physiol 260:C581-C588, 1991.
13. Bobik A, Little PJ and Neylon CB. Ionic mechanisms regulating sodium entry into vascular smooth muscle. Clin Exp Pharmacol Physiol 18:117-120, 1991.
14. Bohn M, Howard E, Vielkind U and Krozowski Z. Glial cells express bothe mineralocorticoid and glucocortoid receptors. J Steroid Biochem Mol Biol, in press.
15. Boublik JH, Spicer MA, Scott NA, Rivier JE and Brown MR. High dose Neuropeptide Y elicits biphasic effects on the conscious rat cardiovasculature: Structure-activity studies. In: Proceedings of the 2nd International Symposium on Endocrinology Under 35; Horizons in Endocrinology Vol II, (eds) M Maggi and V Geenen, Raven Press, pp. 229-234, 1991.
16. Campbell GR and Campbell JH. Microscopic and ultramicroscopic methods of identifying and quantitating areas of diffuse fibrous arterial thickenings and atheromatous plaques. In: Progress, Problems and Promises for an Effective Quantitative Evaluation of Atherosclerosis in Animals and Man, (eds) RW Wissler, MG Bond, M Mercuri and P Tanganelli, New York, Plenum Press, in press.
17. Campbell JH and Campbell GR. The macrophage as an initiator of atherosclerosis. Clin Exp Pharmacol Physiol 18:81-84, 1991.
18. Campbell JH, Rennick RE and Campbell GR. The importance of quantitating the levels and activity of endoglycosidases (heparanases) as an indicator of the initiating role of macrophages in smooth muscle cell proliferation. In: Progress, Problems and Promises for an Effective Quantitative Evaluation of Atherosclerosis in Animals and Man, (eds) RW Wissler, MG Bond, M Mercuri and P Tanganelli, New York, Plenum Press, in press.
19. Campbell JH, Rennick RE, Kalevitch SG and Campbell GR. Heparan sulphate-degrading enzymes induce modulation of smooth muscle phenotype. Exp Cell Res, in press.
20. Campbell JH, Tachas G, Black MJ, Cockerill G and Campbell GR. Molecular biology of vascular hypertrophy. Basic Res Cardiol 86/51:3-11, 1991.
21. Chong BH, Du X, Berndt MC, Horn S and Chesterman CN. Characterization of the binding domains on glycoproteins Ib-IX and IIb-IIIa complexes for the quinine/quinidine-dependent antibodies. Blood 77:2190-2199, 1991.
22. Christy IJ, Woods RL, Courneya CA, Denton KM and Anderson WP. Evidence for a renomedullary vasodepressor system in rabbits and dogs. Hypertension 18:325-333, 1991.
23. Clay MA, Newnham HH and Barter PJ. Hepatic lipase promotes a loss of apolipoprotein A-I from triglyceride-enriched human high density lipoproteins during incubation in vitro. Arterioscler Thromb 11:415-422, 1991.
24. Cochrane AD and Rosenfeldt FL. Temperatures attained by donor hearts stored in ice during transport are not too cold. J Thoracic Cardiovas Surg, in press.
25. Cocks TM and Angus JA. Evidence that contractions of isolated arteries by L-NMMA and NOLA are not due to inhibition of basal EDRF release. J Cardiovasc Pharmacol, in press.
26. Cocks TM, Malta E, King SJ, Woods RL and Angus JA. Oxyhaemoglobin increases the production of endothelin-1 (ET-1) by endothelial cells in culture. Eur J Pharmacol 196:177-182, 1991.
27. Courneya CA and Korner PI. Neurohumoral mechanisms and the role of arterial baroreceptors in the reno-vascular response to haemorrhage in rabbits. J Physiol 437:393-408, 1991.
28. Cramer EM, Lu H, Caen JP, Soria C, Berndt MC and Tenza D. Differential redistribution of platelet glycoproteins Ib and IIb-IIIa after plasmin stimulation. Blood 77:694-699, 1991.
29. Dart AM, Lacombe F, Yeoh JK, Cameron JK, Jennings GL and Esmore DS. Aortic distensibility in patients with isolated hypercholesterolaemia, coronary artery disease or cardiac transplant. Lancet 338:270-273, 1991.
30. Dart AM and Riemersma RA. Noradrenaline release from the rat heart during anoxia: Effects of changes in extracellular sodium concentration and inhibition of uptake mechanisms. Clin Exp Pharmacol Physiol 18:43-46, 1991.

31. Denton KM and Anderson WP. Morphometric analysis of the actions of angiotensin II on renal aterioles and glomeruli. Hypertension, in press.
32. Denton KM and Anderson WP. Glomerular ultrafiltration in rabbits with superficial glomeruli. Pflugers Arch-Eur J Physiol 419:235-242, 1991.
33. Dilley RJ. Genetic control of vascular wall mass. In: Resistance Arteries, Structure and Function, (eds) MJ Mulvany, C Aalkjaer, AM Heagerty, NCB Nyborg and S Trandgaard, Amsterdam, Elsevier, pp. 296-298, 1991.
34. Dilley RJ, McGeachie JK and Tennant M. The role of cell proliferation and migration in vein graft intimal hyperplasia. Cell Tissue Res, in press.
35. Dorward PK and Rudd CD. Influence of brain renin-angiotensin system on renal sympathetic and cardiac baroreflexes in conscious rabbits. Am J Physiol 260:H770-H778, 1991.
36. Dorward PK, Saigusa T and Eisenhofer G. Differential effects of central and peripheral desipramine on sympathoedrenal function and heart rate in conscious rabbits. J Cardiovas Pharmacol 17:519-531, 1991.
37. Dry KL, Phillips JH and Dart AM. Catecholamine release from bovine adrenal chromaffin cells during anoxia or metabolic inhibition. Circ Res 69:466-474, 1991.
38. Du X-J, Dart AM, Riemersma RA and Oliver MF. Sex differences in presynaptic adrenergic inhibition of norepinephine release during normoxia and ischaemia in rat heart. Circ Res 68:827-835, 1991.
39. Eisenhofer G, Cox HS and Esler MD. Noradrenaline reuptake and plasma dihydroxyphenylglycol during sustained changes in sympathetic activity in rabbits. J Auton Nerv System 32:217-232, 1991.
40. Eisenhofer G, Esler MD, Goldstein DS and Kopin IJ. Neuronal uptake, metabolism, and release of tritium-labeled norepinephrine during assessment of its plasma kinetics. Am J Physiol 261:E505-E515, 1991.
41. Eisenhofer G, Esler MD, Meredith IT, Ferrier C, Lambert GW and Jennings GL. Neuronal re-uptake of noradrenaline by sympathetic nerves in humans. Clin Sci 80:257-263, 1991.
42. Eisenhofer G, Meredith IT, Ferrier C, Cox HS, Lambert GW, Jennings GL and Esler MD. Increased plasma dihydroxyphenylalanine during sympathetic activation in humans is related to increased norepinephrine turnover. J Lab Clin Med 117:266-273, 1991.
43. Eisenhofer G, Saigusa T, Esler MD, Cox HS, Angus JA and Dorward PK. Central sympatho-inhibition and peripheral neuronal uptake blockade after desipramine in rabbits. Am J Physiol 260:R824-R832, 1991.
44. Eisenhofer G, Smolich J and Esler MD. Neuronal reuptake of norepinephrine and production of dihydroxyphenylglycol by cardiac sympathetic nerves in the anesthetized dog. Circulation 84:1354-1363, 1991.
45. Eisenhofer G, Smolich JJ and Esler MD. Release and disposition of epinephrine compared to norepinephrine in the canine heart. Naunyn-Schmiedeberg's Arch Pharmacol, in press.
46. Eisenhofer G, Smolich JL and Esler MD. Different desipramine-sensitive pulmonary removals of plasma epinephrine and norepinephrine in dogs. Am J Physiol, in press.
47. Esler M, Eisenhofer G, Chin J, Jennings G, Meredith I, Cox H, Lambert G, Thompson J and Dart AM. Is adrenaline released by sympathetic nerves in man? Clin Auto Res 1:103-108, 1991.
48. Esler MD, Dudley F, Jennings GL, Debinski H, Lambert GW, Jones P, Crotty B, Colman J and Willett I. Increased sympathetic nervous system activity, and effects of its pharmacological inhibition, in alcoholic cirrhosis. Annals Int Med, in press.
49. Esler MD, Eisenhofer G, Dart AM, Chin J, Cox HS, Lambert GW and Jennings GL. Adrenaline release by the human heart. Clin Exp Pharmacol Physiol 18:67-70, 1991.
50. Esler MD, Ferrier C, Lambert GW, Eisenhofer G, Cox HS and Jennings GL. Biochemical evidence of sympathetic hyperactivity in human hypertension. Hypertension 17:III29-III35, 1991.
51. Esler MD, Wallin G, Dorward PK, Eisenhofer G, Westerman R, Meredith IT, Lambert GW, Cox HS and Jennings G. Effects of desipramine on sympathetic nerve firing and norepinephrine spillover to plasma in humans. Am J Physiol 260:R817-R823, 1991.
52. Ferrier C, Esler MD, Eisenhoffer E, Wallin G, Horne M, Cox HS, Lambert GW and Jennings GL. Increased norepinephrine spillover into the cerebrovascular circulation in essential hypertension: Evidence of high central nervous system norepinephrine turnover? Hypertension, in press.
53. Fullerton MJ, Krozowski ZS and Funder JW. Adrenalectomy and dexamethasone administration: effect on ANP synthesis and circulating forms. Mol Cell Endocrinol 82:33-40, 1991.
54. Funder JW. Contribution to "Biology - The Common Threads". (Ed) M Swanage, Canberra, Australian Academy of Science, 1991.
55. Funder JW. A la recherche du temps perdu. In: Aldosterone: Fundamental Aspects, (eds) JP Bonvalet, M Lombes, ME Rafestin-Oblin and N Farman, France, John Libbey Eurotext Ltd, pp. 77-83, 1991.
56. Funder JW. Corticosteroid receptors in the brain. In: Brain Endocrinology, 2nd edition, (ed) M Motta, New York, Raven Press, pp. 133-151, 1991.
57. Funder JW. Enzymes, receptors and steroid specificity. In: Neurosteroids and Brain Function, Fidia Research Foundation Symposium Series, (eds) E Costa and SM Paul, New York, Thieme Medical Publishers, Inc., pp. 11-15, 1991.
58. Funder JW. Metabolic intervention in the specificity of steroid hormone action. In: The New Biology of Steroid Hormones, Serono Symposia Publications, (eds) RB Hochberg and F Naftolin, New York, Raven Press, pp. 71-76, 1991.
59. Funder JW. Steroids, receptors, response elements: the limits of signal specificity. In: Recent Progress in Hormone Research, (ed) W Bardin, New York, Academic Press, pp. 191-210, 1991.
60. Funder JW. What the nose knows. Eureka Street 1(7):24-26, 1991.
61. Garland CJ and McPherson GA. Nitric oxide does not mediate the hyperpolarization and relaxation to acetylcholine in the rat small mesenteric artery. Br J Pharmacol, in press.

62. Giraud AS, Clarke IJ, Rundle SE, Parker LM, Funder JW, Simpson RJ and Smith AI. Distribution, isolation and sequence analysis of the C-terminal heptapeptide of Pro-enkephalin A (YGGFMRF), from the ovine median eminence. J Neuroendocrin 3(2):215-220, 1991.
63. Head GA. Cardiovascular functions of central noradrenergic neurons in rabbits. Clin Exp Pharmacol Physiol 18:51-54, 1991.
64. Head GA and Burke SB. Importance of noradrenergic and serotonergic pathways in the cardiovascular actions of rilmenidine and clonidine. J Cardiovasc Pharmacol 18:819-826, 1991.
65. Head GA and Minami N. Importance of cardiac but not vascular hypertrophy in the cardiac baroreflex deficit in spontaneously hypertensive and stroke prone rats. Am J Med, in press.
66. Head GA, Minami N, Adams MA and Bobik A. Development of cardiac hypertrophy and its relation to the cardiac baroreflex deficit in hypertension. J Hypertens 9 (Suppl 6):580-581, 1991.
67. Hidaka H and Fidge N. Affinity purification of the hepatic high density lipoprotein receptor identifies two acidic glycoproteins and enables further characterization of their binding properties. Biochem J, in press.
68. Horrigan S, Campbell JH and Campbell GR. Oxidation of beta-very low density lipoprotein by endothelial cells enhances its metabolism by smooth muscle cells in culture. Arterioscler Thromb 11:279-289, 1991.
69. Hua XY, Boublik JH, Rivier JE, Brown MR and Yaksh TL. The Antinociceptive effects of spinally administered Neuropeptide Y in the rat: Systematic studies on structure-activity relationships. J Pharmacol Exp Thera 258:243-248, 1991.
70. Jeffreys D and Funder JW. The forced swimming test: effects of glucose administration on the response to food deprivation. Eur J Pharmacol 205(3):267-269, 1991.
71. Jennings GL, Dart AM, Meredith IT, Korner PI, Laufer E and Dewar E. Effects of exercise and other non-pharmacological measures on blood pressure and cardiac hypertrophy. J Cardiovasc Pharmacol 17:S70-S74, 1991.
72. Jennings GL, Deakin G, Korner PI, Meredith IT, Kingwell BA and Nelson L. What is the dose-response relationship between exercise training and blood pressure?. Annals Int Med 23:313-318, 1991.
73. Jennings GL, Esler MD, Dart AM, Dyke A, Lambert GW, Nelson L, Cox HS, Dewar E, Laufer E and Eisenhofer G. Effects of nebivolol on haemodynamics, cardiac dimensions and function, cardiovascular reflexes and biochemical measures of sympathetic activity in normal human subjects. Drug Investigation 3:51-59, 1991.
74. Jennings GL, Korner PI, Sudhir K, Esler MD, Angus JA and Laufer E. Evidence for a role for the cardiovascular amplifiers in human primary hypertension. Clin Exp Pharmacol Physiol 18(1):37-42, 1991.
75. Kawabata H, Knight KR, Coe SA, Angus JA and O'Brien BMcC. Experience with calcium antagonists nitrendipine, diltiazem and verapamil and beta2-agonist salbutamol in salvaging ischemic skin flaps. Microsurgery, in press.
76. Keightley MC, Funder JW and Fuller PJ. Molecular cloning and sequencing of a guinea-pig pro-opiomelanocortin cDNA. Mol Cell Endocrinol 82:89-98, 1991.
77. Kingwell BA, Dart AM, Jennings GL and Korner PI. The effect of exercise training on the blood pressure-heart rate baroreflex in man. Clin Sci, in press.
78. Kingwell BA, Dart AM, Jennings GL and Korner PI. Exercise and baroreflex sensitivity. Lancet 338:183-184, 1991.
79. Kingwell BA, McPherson GA and Korner PI. Assessment of gain of tachycardia and bradycardia responses of cardiac baroreflex. Am J Physiol 260:H1254-H1263, 1991.
80. Knight KR, Kawabata H, Coe SA, Angus JA, O'Brien BM and Sakai K. Prostacyclin and prostanoid modifiers aid ischemic skin flap survival. J Surg Res 50:119-123, 1991.
81. Korner PI, Bobik A, Jennings GL, Angus JA and Anderson WP. Significance of cardiovascular hypertrophy in the development and maintenance of hypertension. J Cardiovasc Pharmacol 17 (Suppl 2):S25-S32, 1991.
82. Krozowski Z, Obeyesekere V, Smith R and Mercer W. Tissue specific expression of an 11a-hydroxysteroid dehydrogenase with a truncated N-terminal domain: A potential mechanism for differential intracellular localization within mineralocorticoid target cells. J Biol Chem, in press.
83. Lambert GW, Eisenhofer G, Cox HS, Horne M, Kalff V, Kelly M, Jennings GL and Esler MD. Direct determination of homovanillic acid release from the human brain, an indicator of central dopaminergic activity. Life Sciences 49:1061-1072, 1991.
84. Lawson A, Ahima R, Krozowski ZS and Harlan R. Postnatal development of corticosteroid receptor immunoreactivity in the rat hippocampus. Developmental Brain Research 62:69-79, 1991.
85. Lew RA, Clarke IJ and Smith AI. Distribution and characterization of peptidylglycine alpha-amidating monooxygenase (PAM) activity in the ovine brain and hypothalamo-pituitary axis. Endocrinology, in press.
86. Little PJ. Na+/H+ exchange in an excitable tissue. In: Amiloride and its Analogues: Unique Cation Transport Inhibitors, (ed) Simchowitzet al, New York, CRC Press, in press.
87. Liu JP, Clarke IJ, Funder JW and Engler D. Evidence that the central noradrenergic and adrenergic pathways activate the hypothalamic-pituitary-adrenal axis in the sheep. Endocrinology 129:200-209, 1991.
88. Luff SE. Neuromuscular junctions and neuromuscular transmission in the vasculature. In: Resistance Arteries, Structure and Function, (eds) MJ Mulvany, C Aalkjaer, AM Heagerty, NCB Nyborg and S Trandgaard, Amsterdam, Elsevier, pp. 144-146, 1991.
89. Luff SE. Ultrastructure of arterioles. In: The Resistance Vasculature, (eds) JA Bevan, B Halpern and MJ Mulvany, Humana Press, in press.
90. Luff SE, Hengstberger SG, McLachlan EM and Anderson WP. Two types of sympathetic axons innervating the juxtaglomerular arterioles of the rabbit and rat kidney differ structurally from those supplying other arteries. J Neurol Cyto 20:781-795, 1991.
91. Mason WT, Hoyland J, NeyLon CB, Kato M, Akerman S, Bunting R, Tregear RT and Zorec R. Dynamic, real time imaging of fluorescent probes of biological activity in living cells. J Microsc, in press.

92. Matthew P, Agrotis A, Taylor AC and McKechnie SW. An association between ADH protein levels and polymorphic nucleotide variation in th Adh gene of Drosophila melanogaster. Mol Biol Evol, in press.
93. Mazurov AV, Vinogradov DV, Vlaski TN, Repin VS, Booth WJ and Berndt MC. Characterization of an antiglycoprotein Ib monoclonal antibody that specifically inhibits platelet-thrombin interaction. Thromb Res 62:673-684, 1991.
94. McLachlan EM and Luff SE. Sympathetic innervation of renal and extra-renal arterial vessels. Kidney Int, in press.
95. McPherson GA and Angus JA. Evidence that acetylcholine-mediated hypertolarization of the rat small mesenteric artery does not involve the K^+ channel opened by cromakalim. Br J Pharmacol 103:1184-1190, 1991.
96. McPherson GA and Angus JA. Spasmolytic effect of cromakalim in the dog coronary artery. Naunyn-Schmiedeberg's Arch Pharmacol 343:519-524, 1991.
97. McPherson GA and Stork AP. Rat cerebral arteries are resistant to the vasorelaxant effect of cromakalim and other K^+ channel openers. Br J Pharamcol, in press.
98. Mercer M and Krozowski Z. Localization of an 11beta hydroxysteroid dehydrogenase activity to the distal nephron: Evidence for the existence of two species of dehydrogenase in the rat kidney. Endocrinology, in press.
99. Meredith IT, Broughton A, Jennings GL and Esler MD. Evidence for a selective increase in resting cardiac sympathetic activity in some patients suffering sustained out of hospital ventricular arrhythmias. New Eng J Med 325:618-624, 1991.
100. Meredith IT, Eisenhofer G, Lambert GW, Jennings GL and Esler MD. Plasma norepinephrine responses to head-up tilt was misleading in autonomic failure. Hypertension, in press.
101. Meredith IT, Esler MD, Cox HS, Lambert GW, Jennings GL and Eisenhofer G. Biochemical evidence of sympathetic denervation of the heart in pure autonomic failure. Clin Autonom Res 1:187-194, 1991.
102. Meredith IT, Friberg P, Jennings GL, Dewar EM, Fazio VA, Lambert GW and Esler MD. Exercise training lowers resting renal but not cardiac sympathetic activity. Hypertension 18:575-582, 1991.
103. Meyer B, Kecorius E, Barter P, Fidge N and Tetaz T. Application of reversed-phase high-performance liquid chromatography to the separation of apolipoproteins A-IV, A-I and E from rat high-density lipoprotein. J Chromatogr 540:386-391, 1991.
104. Morrison J, Fidge N and Tozuka M. Determination of the structural domains of AI apolipoproteins recognised by the high density lipoprotein receptor complex. J Biol Chem 266: 18780-18788, 1991.
105. Munsch C, Newman M, Chang V, Davis BB and Rosenfeldt FL. Absence of particle-induced coronary vasoconstriction during cardioplegic infusion: is it desirable to use a micro-filter in the infusion line?. J Thoracic Cardiovas Surg 101:473-480, 1991.
106. Munsch CM, Rosenfeldt FL, O'Halloran K, Langley LH, Conyers RJ and Williams JF. The effect of orotic acid on the response of the recently infarcted rat heart to hypothermic cardioplegia. Eur J Cardiothoracic Surg 5:82-93, 1991.
107. Myers D, Fidge N, Stanton H and Larkins R. The effects of low density lipoprotein and high density lipoprotein on phosphoinositide hydrolysis in bovine aortic endothelial cells. Atherosclerosis, in press.
108. Neylon CB and Irvine RF. Thrombin attenuates the stimulatory effect of histamine on Ca2+ entry in confluent human umbilical vein endothelial cell cultures. J Biol Chem 266:4251-4256, 1991.
109. Neylon CB, Mason WT and Irvine RF. Histamine-induced calcium oscillations in human vascular smooth muscle: temporal sequence and spatial organization in single cells. Clin Exp Pharmacol Physiol 18:299-302, 1991.
110. Neylon CB, Nickashin A, Little PJ, Tkachuk VA and Bobik A. Thrombin-induced calcium mobilization in vascular smooth muscle utilises a slowly ribosylating pertussis toxin-sensitive G protein. J Biol Chem :accepted for publication, 1991.
111. Nishimura H, Saltis J, Habberfield AD and Simpson IA. Phosphorylation state of glucose transporter isoforms in subfractions of the rat adipose cell: effects of insulin, isoproterenol and phorbol ester. Proc Natl Acad Sci USA, in press.
112. Rabinov M, Newman M, Smolich J and Rosenfeldt FL. Adverse effects of low pressure reperfusion after hypothermic cardioplegia in the hypertrophic heart. J Thorac Cardiovasc Surg 102:695-706, 1991.
113. Reid CM, Jennings GL, Leonard RF and Conyers RAJ. Measuring blood cholesterol outside the pathology laboratory: issues of accuracy and reliability. Aust J Public Health 15:142-146, 1991.
114. Rennick RE, Campbell JH and Campbell GR. Binding of beta-VLDL by cultured smooth muscle cells and their accumulation of cholesterol esters are enhanced by macrophages. Atherosclerosis, in press.
115. Rogers PF, Head GA, Lungershausen YK and Howe PRC. Effects of depleting central and peripheral adrenaline stores on blood pressure in stroke-prone spontaneously hypertensive rats. J Auton Nerv Syst 34:9-16, 1991.
116. Rosenfeldt FL, Chi L, Black AJR, Waugh JR, Pedersen JS and Levatter J. Excimer laser angioplasty in the atherosclerotic rabbit: comparison with balloon angioplasty. Am Heart J, in press.
117. Rosenfeldt FL, Muller D, Harper R, Shardey GC and Broughton A. Early and late results of surgery for Wolff-Parkinson-White Syndrome. ANZ J Med 21:325-329, 1991.
118. Rosenfeldt FL, Munsch C, Langley L, Conyers R and Williams JF. Enhanced myocardial metabolism improves recovery after cardiac surgery and transplantation. Clin Exp Pharmacol Physiol 18:121-122, 1991.
119. Saltis J, Agrotis A and Bobik A. Transforming growth factor-beta1 enhances the effects of epidermal growth factor on vascular smooth muscle from the spontaneously hypertensive rat. J Hypertens, in press.
120. Saltis J, Habberfield AD, Egan JJ, Londos C, Simpson IA and Cushman SW. Role of protein kinase C in the regulation of glucose transport in the rat adipose cell. Translocation of glucose transporters without stimulation of glucose transport activity. J Biol Chem 266:261-267, 1991.
121. Saltis J, Little PJ and Bobik A. Multiple growth abnormalities in vascular smooth muscle from spontaneously hypertensive rats. Clin Exp Pharmacol Physiol 18:319-322, 1991.

122. Schwartz J, Pham T, Rao A and Funder JW. Effect of vasopressin on susceptibility of ovine pituitary cells to a cytotoxic analog of CRF. Am J Physiol, in press.
123. Schwartz SM and Dilley RJ. Vascular Injury. In: Textbook of Vascular Medicine, (eds) J Loscalzo, M Creager and V Dzau, Little, Brown and Company, in press.
124. Shen A, Chin J, Fullerton M, Jennings G and Dart A. Increases in plasma beta-endorphin levels during exercise do not contribute to increases in heart rate following autonomic blockade in man. Brit J Clin Pharmacol, in press.
125. Sheppard K, Roberts JL and Blum M. Adrenocorticotropin-releasing factor down-regulates glucocortoid receptor expression in mouse corticotrope tumor cells via an adenylate cyclase dependent mechanism. Endocrinology 129:663-670, 1991.
126. Shirinsky VP, Birukov KG, Koteliansky VE, Glukova MA, Spanidis E, Rogers J, Campbell JH and Campbell GR. Density-related expression of caldesmon and vinculin in cultured rabbit aortic smooth muscle cells. Exp Cell Res 194:186-189, 1991.
127. Shukovski L, Findlay JK and Smith AI. Identification of a noncovalent oxytocin/neurophysin I complex in the bovine ovary. J Endocrinol 128:305-314, 1991.
128. Simpson IA, Cushman SW, Egan JJ, Habberfield AD, Londos C, Nishimura H and Saltis J. Hormonal regulation of glucose transport in rat adipose cells. Bioch Soc Trans 18:1123-1125, 1991.
129. Skinner MP, Lucas CM, Burns GF, Chesterman CN and Berndt MC. GMP-140 binding to neutrophils is inhibited by sulfated glycans. J Biol Chem 266:5371-5374, 1991.
130. Smith AI, Clarke IJ, Wallace CA, Tetaz T, Glucksman M and Roberts JL. Post-secretary processing of Gn-RH in the ovine hypothalamo-pituitary axis. In: Serono Symposia Publications, in press.
131. Smith JA, Salamonsen RF, Rosenfeldt FL and Esmore DS. Initial clinical experience with the haemopump left ventricular assist device. ANZ J Surg 61:271-275, 1991.
132. Smith RE and Funder JW. Renal 11beta-hydroxysteroid dehydrogenase activity: effects of age, sex and altered hormonal status. J Steroid Biochem Mol Biol 38:265-267, 1991.
133. Sviridov D and Fidge N. The effect of lovastatin on the interaction between high density lipoprotein and cultured rat adrenocortical cells. Atherosclerosis 88:235-242, 1991.
134. Vadas MA, Lopez AF, Gamble JR, Lucas C, Shannon MF, Skinner MP and Berndt MC. The regulation of human eosinophil function. Atopic Disease and Cellular Immunity :119-127, 1991.
135. Vadivaloo P, Morrison J, Fidge N, Lorent G, Brasseur R and Rosseneu M. Characterization of the discoidal complexes formed between apo AI CNB1 fragments and phosphatidyl choline. J Lipid Res 32:1253-1264, 1991.
136. Vadiveloo PK and Fidge NH. The role of apoproteins AI and AII in binding of high density lipoproteins to membranes derived from bovine aortic endothelial cells. Biochem J, in press.
137. Van Den Buuse M, Head GA and Korner PI. Contribution of forebrain noradrenaline innervation to the central circulatory effects of alpha-methyldopa and 6-hydroxydopamine. Brain Research 541:300-308, 1991.
138. Vinogradov DV, Vlasik TN, Agafonova OG, Vasiliev SA, Lagutina NY, Makarov VA, Berndt MC and Masurov AV. Inhibition of platelet Fc-receptor-dependent aggregation by a monoclonal antibody against glycoprotein IIb-IIIa complex. Biochemistry (USSR) 56:787-797, 1991.
139. Wallin BG, Esler MD, Dorward PK, Eisenhofer G, Ferrier C, Westerman R and Jennings GL. Simultaneous measurements of cardiac norepinephrine spillover and sympathetic outflow to skeletal muscle in humans. J Physiol, in press.
140. Warren RJ, Ebert DL, Barter PJ and Mitchell A. The regulation of hepatic lipase and cholesteryl ester transfer protein activity in the cholesterol-fed rabbit. Biochim Biophys Acta, in press.
141. Warren RJ, Ebert DL, Mitchell A and Barter PJ. Rabbit hepatic lipase cDNA sequence: Low activity is associated with low messenger RNA levels. J Lipid Res 32:1333-1339, 1991.
142. Wilcox GR, Berndt MC, Mehrabani PA, Exner T and Trudinger BJ. An improved method for measuring plasma glycocalicin in the investigation of causes of thrombocytopenia. Platelets 2:45-50, 1991.
143. Wong CS, Gamble JR, Skinner MP, Lucas CM, Berndt MC and Vadas MA. Adhesion protein GMP 140 inhibits superoxide anion release by human neutrophils. Proc Natl Acad Sci USA 88:2397-2401, 1991.
144. Woodcock EA and Anderson K. Isolation of adult cardiomyocytes initiates a return of inositol trisphosphate phosphorylating activity. Clin Exp Pharmacol Physiol, in press.
145. Woodcock EA, Fullerton M, Land S and Kuraja IJ. Culturing rat neonatal myocytes causes changes in the phosphatidylinositol turnover pathway. Clin Exp Pharmacol Physiol 18:331-337, 1991.
146. Woodcock EA and Land S. Endothelin receptors in rat renal papilla with a high affinity for endothelin-3. Eur J Pharmacol, in press.
147. Woodcock EA and Land S. Interaction between vasopressin and endothelin in renal papillary tubules: uncoupling following all isolation and culture. Clin Exp Pharmacol Physiol, in press.
148. Woodcock EA, Tanner JK, Fullerton M and Kuraja IJ. Different pathways of inositol phosphate metabolism in intact neonatal rat hearts and isolated cardiomyocytes. Biochem J, in press.
149. Zuzel M, Walton S, Burns GF, Berndt MC and Cawley JC. A monoclonal antibody to a 67 kD cell membrane glycoprotein directly induces persistent platelet aggregation independently of granule secretion. Brit J Haematol, in press.

BAKER MEDICAL RESEARCH INSTITUTE

CONSOLIDATED INCOME AND EXPENDITURE STATEMENT

YEAR ENDED 31 DECEMBER 1991

	NOTES	1991 $	1990 $
INCOME			
Government and statutory bodies	3	4,157,870	3,789,164
Baker Benefactions		1,021,340	926,404
Alfred Hospital		168,312	202,779
Donations, Corporate & Private Support		1,800,401	2,009,461
Investment income		426,672	560,798
Clinical services		195,243	130,385
General income		24,113	303,257
Total Income		**7,793,951**	**7,922,248**
EXPENDITURE			
Salaries and wages		4,849,640	4,059,486
Consumable supplies		1,302,109	1,127,592
Scientific equipment		376,181	277,769
Laboratory support costs		703,145	693,878
Administration and general overheads		411,169	652,960
Public relations/fundraising		70,729	146,923
Total Expenditure		**7,712,973**	**6,958,608**
Surplus	5	**80,978**	**963,640**

The accompanying notes form an integral part of these accounts

BAKER MEDICAL RESEARCH INSTITUTE

CONSOLIDATED BALANCE SHEET AS AT 31 DECEMBER 1991

	NOTES	1991 $	1990 $
CURRENT ASSETS			
Cash at bank and in hand		91,063	182,307
Debtors		192,328	165,849
Prepayments		68,564	-
Investments (at cost)	6(a)	2,507,915	2,265,898
Total current Assets		**2,859,870**	**2,614,054**
NON CURRENT ASSETS			
Investments (at cost)	6(b)	1,787,301	1,047,620
Total non-current assets		**1,787,301**	**1,047,620**
Total Assets		**4,647,171**	**3,661,674**
CURRENT LIABILITIES			
Creditors		326,417	83,405
Prepaid income		693,405	-
Total current liabilities		**1,019,822**	**83,405**
NON CURRENT LIABILITIES			
Provisions	7	737,861	769,759
Total non-current liabilities		**737,861**	**769,759**
Total liabilities		**1,757,683**	**853,164**
Net Assets		**2,889,488**	**2,808,510**
FUNDS			
Accumulated Funds			
Operating fund		(1,171,721)	(1,202,237)
Capital fund		2,288,749	2,088,240
Specific purpose funds	4	1,772,460	1,922,507
	5	**2,889,488**	**2,808,510**

The accompanying notes form an integral part of these accounts

BAKER MEDICAL RESEARCH INSTITUTE

NOTES TO AND FORMING PART OF THE ACCOUNTS

1. INCORPORATION

The Thomas Baker, Alice Baker and Eleanor Shaw Medical Research Institute was incorporated as the 'Baker Medical Research Institute' ("the Institute") under the Baker Medical Research Act 1980.

2. SUMMARY OF SIGNIFICANT ACCOUNTING POLICIES

Set out hereunder are the significant accounting policies adopted by the Institute in the preparation of its accounts for the year ended 31 December 1991. These policies have been consistently applied unless otherwise indicated. The accounts have been prepared using the historical cost convention and on a normal accrual basis.

(a) Institute Funds, Income and Expenditure

The work of the Institute is financed from grants, endowments, bequests, investment income and donations of both general and specific natures. Income of a specific nature is used in accordance with the terms of any relevant covenants. The amount of grants received for specific purposes during the year but unspent at year end, is carried forward to the next financial year.

The Institute's accounts have been prepared on a consolidated basis. The results of particular funds in relation to the consolidated surplus are set out in note 5.

(b) Fixed Assets and Depreciation

(i) Fixed assets are not shown in the accounts. Grants are provided or allocated for the purchase of items of equipment. It is Institute policy that all such capital expenditure is written off in the year of purchase through the income and expenditure account, consequently no depreciation is charged in the accounts. The amount written off in the year ended 31 December 1991 amounted to $376,181.

To date the Institute has acquired and purchased out of grant income, assets which at balance date have an estimated cost of approximately $3.9m. The building occupied by the Institute is not included as an asset as the Institute does not have title to the property. The estimated replacement cost of these assets and the building is $16.8m.

(ii) The writing-off of assets in the year of purchase is contrary to generally acceptable accounting standards. The Board believes that the policy adopted is appropriate to a research institute where grants are provided or allocated for the purchase of assets.

(c) Stocks

Stocks of consumable scientific and administrative items purchased in the course of normal operations out of grant income are not taken into account at the balance date as assets but are written off at the time of purchase.

(d) Income Tax

The income of the institute is exempt from income tax pursuant to the provisions of section 23(e) of the Income Tax Assessment Act.

(e) Employee Entitlements

The Institute has fully provided for accrued annual leave for all staff employed as at 31 December 1991. Long service leave entitlements are provided for staff with ten or more years of service.

(f) Foreign Exchange Transactions

The Institute maintains bank accounts in the USA and UK for the purpose of receiving donations and for the purchase of equipment and supplies. Foreign currency at balance date is translated at exchange rates at balance date. Exchange gains and losses are brought to account in determining the surplus or deficit for the year.

(g) Comparative Figures

Where necessary comparative figures have been adjusted to conform with changes in presentation in the current year.

3. INCOME

Government and statutory bodies	1991 $	1990 $
National Health & Medical Research Council	2,795,267	2,364,118
Victorian State Government	640,060	706,740
National Heart Foundation	400,783	346,506
Victorian Health Promotion Foundation	321,760	371,800
	4,157,870	**3,789,164**

4. SPECIFIC PURPOSE FUNDS

Specific purpose funds comprise funds provided to the Institute for special purposes other than through normal fund raising activities. The funds are used in accordance with the wishes of the donors. Institute accounting records are kept as to identify expenditure charged against income from these funds. All such income and expenditure is incorporated in the consolidated Income and Expenditure Statement.

General Restricted Funds include major contributions from Glaxo Australia Pty. Ltd. and I.R.I. Servier & Compagnie - Developpement.

General Restricted Funds	1,186,785	1,349,979
Ethel Mary Baillieu Fund	134,180	131,810
Bertalli Family Research Fund	117,279	114,933
William Buckland Research Fund	41,418	39,888
Lang Research Scholarship Fund	105,357	103,249
Laura Nyulasy Scholarship Fund	4,108	2,892
Edgar Rouse Memorial Scholarship Fund	93,897	91,863
Ruby Wallace Travel Scholarship Fund	89,103	87,893
Integrity Trust	333	-
	1,772,460	1,922,507

5. FUND MOVEMENTS

	1991 $	1990 $
Balance at 1 January 1991	2,808,510	1,844,870
Surplus/(deficit) for year -		
operating fund	30,516	(28,818)
capital fund	200,509	218,627
specific purpose funds	(150,047)	773,831
Balance at 31 December 1991	**2,889,488**	**2,808,510**

6. INVESTMENTS (at cost)

(a) CURRENT		
Short term deposits	2,507,915	2,265,898
	2,507,915	2,265,898
(b) NON CURRENT		
Shares and debentures	1,511,914	440,594
Trust units	65,032	379,609
Government and semi-government stock	202,600	202,600
Mortgage loan	7,755	24,817
	1,787,301	**1,047,620**
	4,295,216	**3,313,518**

7. PROVISIONS

Employee entitlements:		
Annual leave	230,425	210,003
Long service leave	216,944	269,264
Deferred maintenance	290,492	290,492
	737,861	**769,759**

8. REMUNERATION OF BOARD MEMBERS

The Board Members of the Baker Medical Research Institute during the year were:

J.D. Moir	M. Ross	G.P. Johnston (appointed 15/4/91)
D.F. Hogarth	J. Loy	G.B. Ryan
R.J. Barcham	W.D. McPherson	D.J. Butler
J. Grimwade	W.G. Philip	W.A. Kricker
J.W. Funder	R. Porter	D. Wittner (resigned 15/4/91)

No Board Member has received or become entitled to receive a benefit other than the Director of the Institute, Professor J.W. Funder, who receives a salary.

9. SUPERANNUATION

The Institute operates a superannuation plan under which all employees are entitled to benefits on retirement, disability or death. Employees contribute to the plan at various percentages of their salaries. The Institute also contributes to the plan at rates related to employer contributions and pursuant to an award set down under a national wage case.

Funds are available to satisfy all benefits that have been vested under the plan in the event of termination of the plan or voluntary or compulsory termination of employment of each employee.

10. CONTINGENT LIABILITY

A contingent liability exists where the Institute has indemnified a former employee in a libel action brought against him in circumstances where he was representing the Institute. The action is presently pending and it is the opinion of the solicitors of the Institute and the Board of Management that the result of this action cannot be assessed at this time.

11. STATEMENT OF SOURCES AND APPLICATION OF FUNDS

	1991 $	1990 $
SOURCES OF FUNDS		
Funds from Operations		
Inflow of funds from Operations:		
Grant income	5,187,121	4,888,542
Interest	426,672	560,798
Other income	2,180,158	2,472,908
Outflow of funds to Operations	(7,744,871)	(6,958,608)
	49,070	963,640
Reduction in Assets		
Current Assets		
Cash	91,244	
Debtors	-	87,807
Non Current Assets		
Investments	-	3,735
Increase in Liabilities		
Creditors	243,012	-
Provisions	-	229,040
Prepaid income	693,405	-
	1,076,741	**1,284,222**
APPLICATION OF FUNDS		
Increase in Assets		
Current Assets		
Cash	-	326,837
Debtors	26,479	-
Prepayments	68,564	-
Investments	242,017	777,630
Non Current Assets		
Investments	739,681	-
Reduction in Liabilities		
Creditors	-	179,755
	1,076,741	**1,284,222**

AUDITORS' REPORT TO THE BOARD OF MANAGEMENT

BAKER MEDICAL RESEARCH INSTITUTE

We have audited the accounts set out on pages 34 to 39 in accordance with Australian Auditing Standards.

As indicated in note 2(b), it is the Institute's policy to write off all capital expenditure as incurred.

In our opinion, with the exception of the effect of the omission of these assets and the related depreciation charge, the attached accounts are drawn up so as to give a true and fair view of the accumulated funds of the Institute and the net assets representing those funds at 31st December 1991 and the movements in these funds for the year to 31st December 1991, and have been made out in accordance with Australian Accounting Standards applicable to non business entities.

Price Waterhouse

EA Alexander

A member of the firm

Chartered Accountants.

Melbourne

1st April, 1992

BAKER MEDICAL RESEARCH INSTITUTE

STATEMENT BY BOARD MEMBERS

In the opinion of the Board Members:

(a) the accounts set out on pages 34 to 39 are drawn up so as to give a true and fair view of the state of the Institute's affairs as at 31st December, 1991 and of its results for the year ended on that date;

(b) at the date of this statement there are reasonable grounds to believe that the Institute will be able to pay its debts as and when they fall due;

(c) the accounts have been compiled in accordance with Australian Accounting Standards, except in relation to the treatment of capital expenditure and depreciation of scientific assets as set out in the notes to the accounts and referred to in the Report of the Auditors. Signed at Melbourne this 1st Day of April, 1992 in accordance with a resolution of the board.

John Moir

President

John Funder

Director

MAJOR DONATIONS

The Institute is grateful for major contributions to its work from:

MAJOR CORPORATE SUPPORTER

Glaxo Australia Pty Ltd

CONTRACT RESEARCH

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Estate Myrtle Margaret Dobbie
Estate Irma Heller
Estate Alison Mary Hooke
Estate Phyllis Latham Perry
Estate Mary Aglaia Venue
Estate Dorothy H Walker
Estate Mrs A M White
Estate Edward Wilson
Estate Ivy Alma Wood
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The William Angliss Charitable Fund
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A special thanks must go to all our many other donors who, through their regular support, are equally important to the ultimate success of our research programme. They are too numerous to list here but their support has been essential in the pursuit of our goals.

ANNUAL DINNER

The Institute is especially grateful to the Sponsors of the Baker Medical Research Institute Annual Dinner held in December 1991. They were:

Australia & New Zealand Banking Group Ltd
Kodak (Australasia) Pty Ltd
Lustig & Moar Group
Hyatt on Collins Hotel

Without their support and generosity this function could not have been held.

EDGAR ROUSE MEMORIAL FUND 1991

In memory of	*From*
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"Loved Ones"	Winfred Chambers
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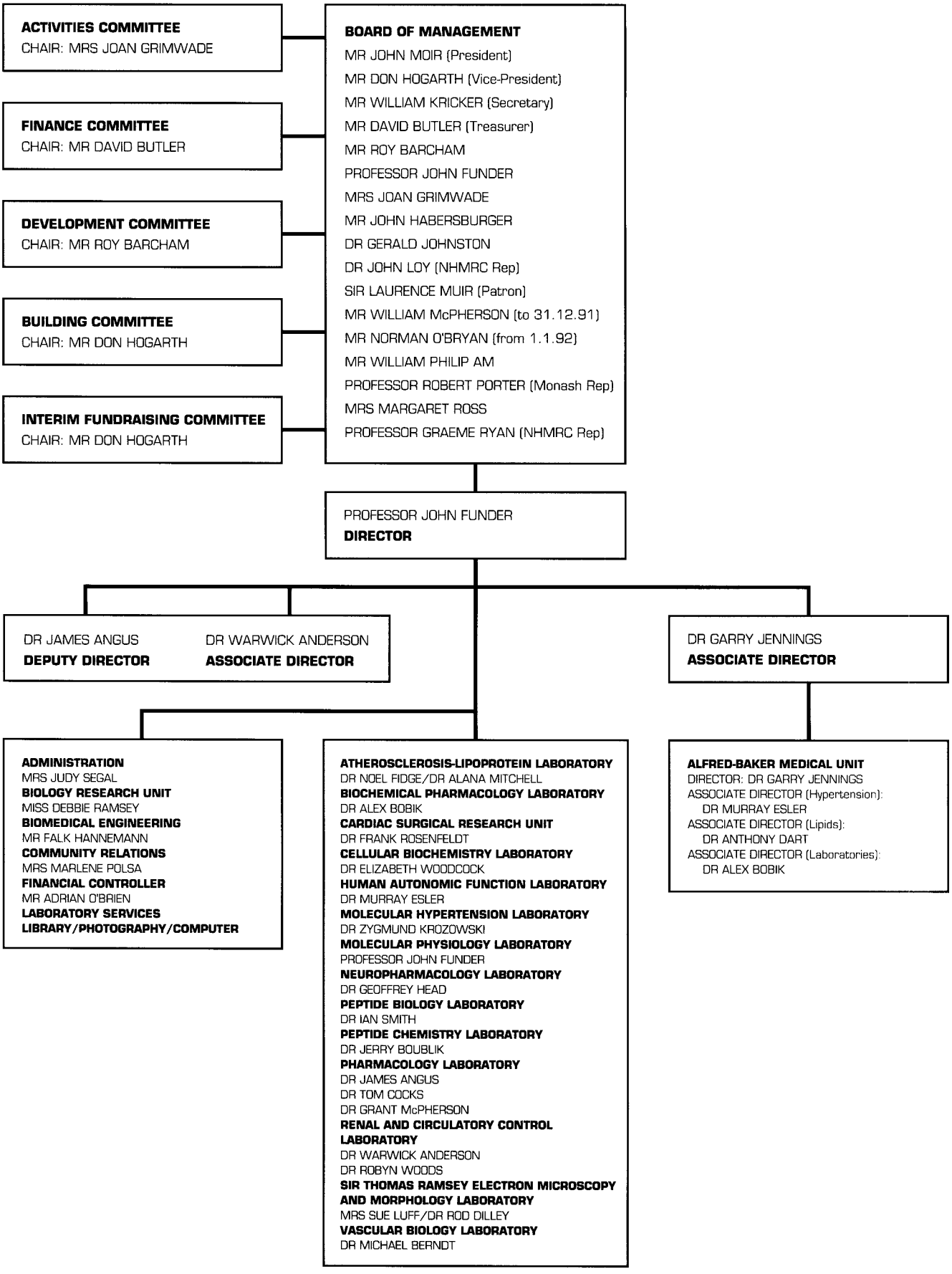

DIRECTORY

AUDITORS

PRICE WATERHOUSE

215 SPRING STREET, MELBOURNE, VIC 3000

SOLICITORS

BLAKE DAWSON WALDRON

140 WILLIAMS STREET, MELBOURNE, VIC 3000

ANNUAL GENERAL MEETING

MONDAY 13th APRIL

BAKER MEDICAL RESEARCH INSTITUTE

5:00 PM

BAKER MEDICAL RESEARCH INSTITUTE

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