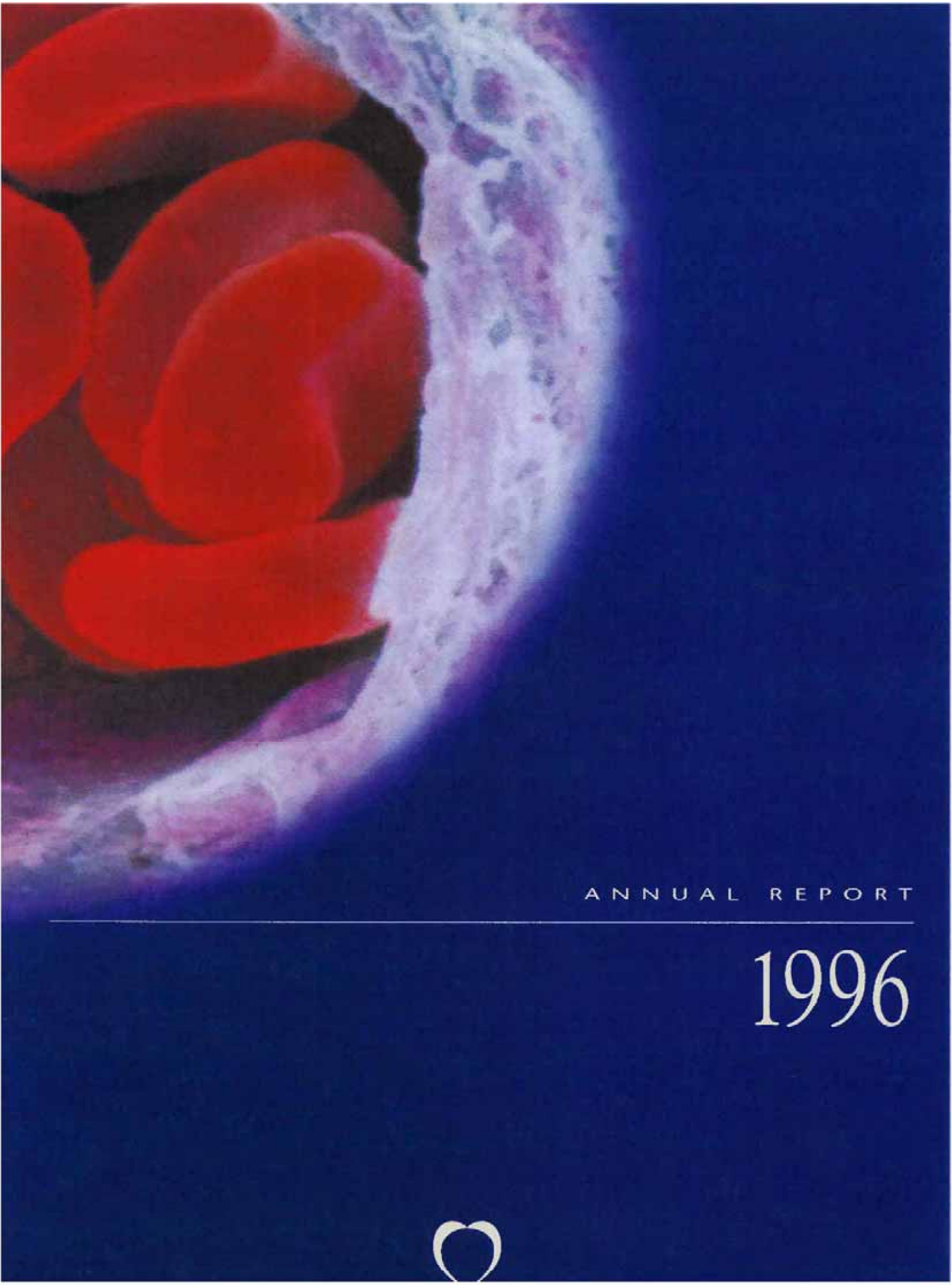
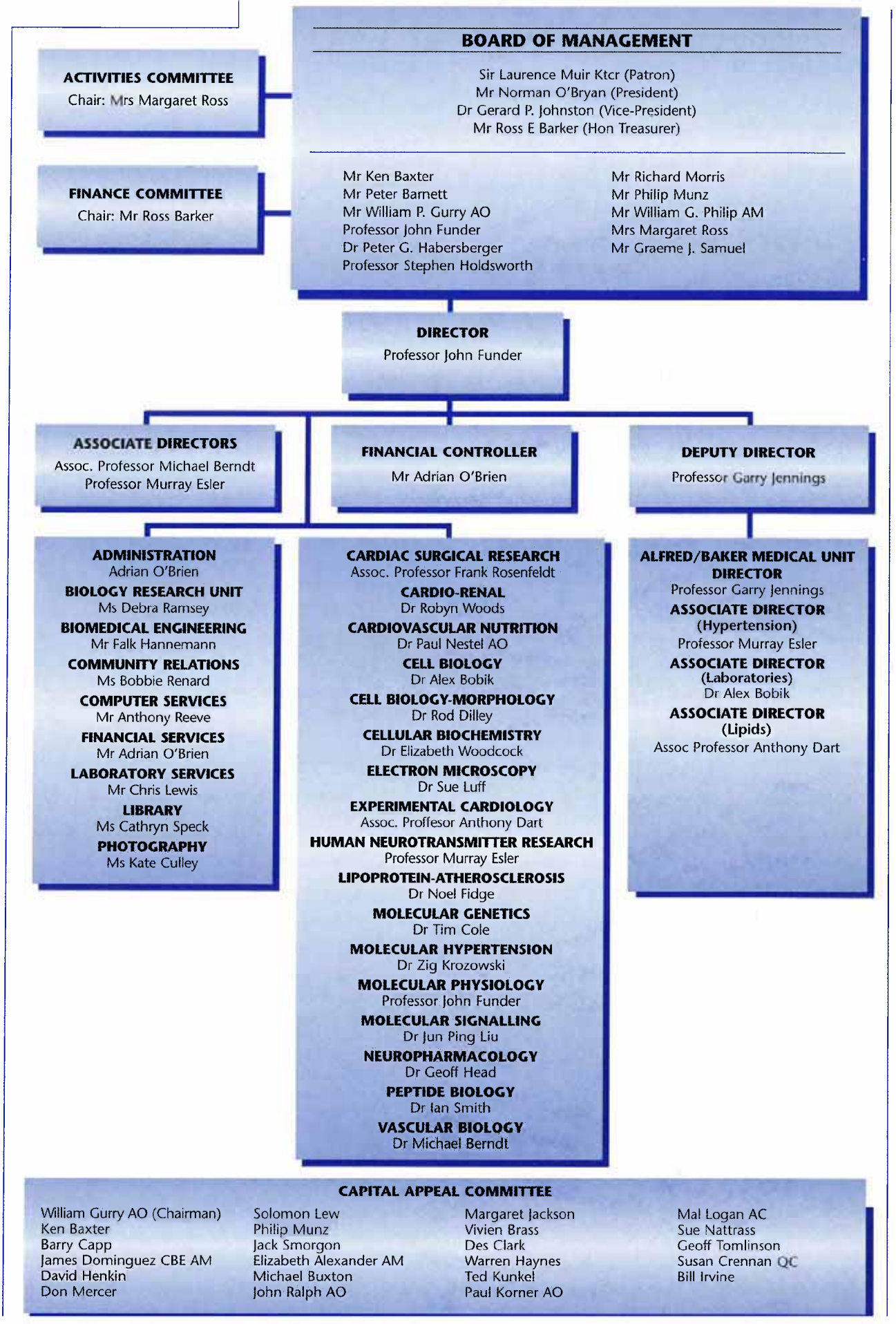


ANNUAL REPORT

1996

The Baker Institute is a block funded institute of the National Health and Medical Research Council of Australia, and is also supported by the Victorian Government and the Baker Benefaction. The institute is affiliated with Monash University and the Alfred Hospital, and Baker staff hold appointments in both of these institutions. In addition, it is a World Health Organisation collaborating centre for research and training in cardiovascular diseases, the only one in Australia.

MANAGEMENT STRUCTURE



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Major Donations in Lieu of Gifts

To celebrate the following occasions:

Mr George & Mrs Gita Smorgon (50th Wedding Anniversary)
Mr Bernie Robertson (70th Birthday)
Mr & Mrs J Sulan (Joint 50th Birthday)
Mr W A Craven (60th Birthday & 1989 Heart Transplant)

Certificates of Appreciation

In addition to the various Charitable Trusts, Foundations and Estates listed in the 1995 Annual Report, a Certificate of Appreciation was also presented to the following at the 1996 Annual General Meeting:

Abbott Stillman & Wilson
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Mr W J Bailey
Mrs D H Behrend
Mrs M Blanchard
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The Institute is grateful for major contributions to its work from:

National Health & Medical Research Council of Australia
Baker Benefaction
Victorian Government
High Blood Pressure Research Council
National Heart Foundation
Victorian Health Promotion Foundation
National Institutes of Health (USA)
Australian Research Council

Merck Sharp & Dohme
IRI Servier & Compagnie Developpement

Capital Appeal

Alfred Healthcare Group
Amcor Limited
Baker Medical Research Staff
Mr Ross Barker
Dame Beryl Beaurepaire
Ms Leonie T Burke
Ms Susan Crennan QC
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Founding Members, Baker Institute Foundation

GSA Group Pty Ltd
Mr William P Gurry AO
Kodak (Australasia) Pty Ltd
Mr Norman O'Bryan
Mrs Margaret Ross

Trusts & Foundations

Alfred Healthcare Foundation Research Grants
William Angliss (Vic) Charitable Fund
William Buckland Foundation
Rebecca L Cooper Foundation
Marion & E H Flack Trust
Elisabeth Murdoch Trust
Clive & Vera Ramaciotti Foundation
George & Edith Ramsay Charitable Trust
R E Ross Trust
Edward Wilson Charitable Fund

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Bell Charitable Trust
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William Buckland Research Fund
Thomas, Annie & Doris Burgess Charity Trust
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Bequests

Estate Lindsay J Baldy
Estate Garnet M Carpenter
Estate Elsie Ehrenfeld
Estate Harry Douglas Greig
Estate Miss Ella B Jones
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RACV
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Royal Australian Air Force Showband
Sandoz Australia Pty Ltd

BAKER MEDICAL RESEARCH INSTITUTE

INDEPENDENT AUDIT REPORT TO THE BOARD OF MANAGEMENT

Scope

We have audited the financial statements of the Institute for the year ended 31st December, 1996 as set out on pages **36** to **42**. The Directors are responsible for the preparation and presentation of the financial statements and the information contained therein. We have conducted an independent audit of the financial statements in order to express an opinion on them to the Board of the Institute.

Our audit has been conducted in accordance with Australian Auditing Standards to provide reasonable assurance as to whether the financial statements are free of material misstatement. Our procedures included examination, on a test basis, of evidence supporting the amounts and other disclosures in the financial statements, and the evaluation of accounting policies and significant accounting estimates. These procedures have been undertaken to form an opinion as to whether, in all material respects, the financial statements are presented fairly in accordance with Accounting Standards, other mandatory professional reporting requirements, being Urgent Issues Group Consensus Views, and the Corporations Law, so as to present a view which is consistent with our understanding of the Institute's state of affairs, the results of its operations and its cash flows.

The audit opinion expressed in this report has been formed on the above basis.

Audit Opinion

In our opinion, the financial statements of the Institute are properly drawn up:

(a) so as to give a true and fair view of the state of affairs of the Institute as at 31 December 1996 and its results and cash flows for the financial year ended on that date

(b) in accordance with provisions of the Corporations Law, and

(c) in accordance with applicable accounting standards and other mandatory professional reporting requirements.

Price Waterhouse
Chartered Accountants

Melbourne
18th April 1997

EA Alexander
Partner

STATEMENT BY BOARD MEMBERS

In the opinion of the Board Members of the Baker Medical Research Institute:

(a) The financial statements and notes to the accounts set out on pages **36** to **42** are drawn up so as to present a true and fair view of the state of the Institute's affairs as at 31st December, 1996 and of its results for the year ended on that date;

(b) As 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; and

(c) The consolidated financial statements have been made out in accordance with applicable Accounting Standards.

Signed at Melbourne this 18th day of April 1997 in accordance with a resolution of the Board.

Norman O'Bryan
President

John W Funder
Director

NOTES TO AND FORMING PART OF THE ACCOUNTS

12. Remuneration of Board Members

(a) The names of each person who held office as a Board Member of the Baker Medical Research Institute during the financial year ended 31 December 1996 are:

Mr N O'Bryan
Dr G P Johnston
Mr R E Barker
Professor J W Funder
Mr P C Barnett
Mr K P Baxter (from Oct'96)
Mr W P Gurry AO
Dr P G Habersberger
Professor S Holdsworth
Mr P Liehne (from Mar - Nov '96)
Dr C Mead (until Feb '96)
Mr R Morris (from Dec '96)
Mr P Munz
Mr W G Philip AM
Mrs M Ross
Mr G Samuel (until Dec '96)

(b) Other than one Board Member who is a Director of a firm of Stockbrokers which has received, or become entitled to receive, fees for services rendered to the Institute on normal commercial terms, no Board Member has received or has become entitled to receive any benefit, by reason of a contract made by the Institute or a related corporation with any Board Member or with a firm of which a Board Member is a member or with an entity in which any Board Member has a substantial financial interest other than the Director of the Institute, Professor J.W. Funder, who receives a salary.

13. Superannuation

The Institute operates an accumulation type 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 employee 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.

14. Notes to the Statement of Cash Flows

(a) Reconciliation of Cash

For the purpose of the statement of cash flows, cash includes cash on hand and in the bank and investments in money market instruments, net of outstanding bank overdrafts.

Cash at the end of the financial year as shown in the statement of cash flows is reconciled to the related items in the balance sheet as follows:

	1996	1995
	$	$
Cash	1,196,111	1,305,135
Deposits at call	4,166,256	3,724,225
Total as above	**5,362,367**	**5,029,360**
(b) Reconciliation of Net Cash provided by Consolidated Activities to Surplus		
Operating Surplus from Consolidated Activities	369,109	685,825
Effects of exchange rate changes on cash held in foreign currencies	13,994	76
Depreciation and Amortisation	573,341	592,305
Net assets disposed of	0	39,056
(Profit) on sale of non-current assets	(130,689)	(286,735)
Change in accounting policy	0	(368,792)
Changes in net assets and liabilities		
(Increase) / Decrease in debtors	(25,804)	78,645
(Increase) / Decrease in inventories	(22,800)	777
Decrease / (Increase) in prepayments	40,551	(61,397)
(Increase) in accrued interest	(4,456)	(77,163)
Increase in creditors	67,848	26,538
(Decrease) / Increase in prepaid grant	(39,703)	2,341,637
(Decrease) / Increase in provisions	(199,120)	359,007
Net cash from consolidated activities	**642,271**	**3,329,779**

(c) Non-cash Financing Activities

Motor Vehicles

During the year the Institute provided motor vehicles for staff under salary sacrifice arrangements with a value of $273,182 by means of finance leases. These acquisitions are not reflected in the statement of cash flows.

NOTES TO AND FORMING PART OF THE ACCOUNTS

8. Investments (at cost)	1996	1995
	$	$
(a) Current		
Short term deposits	4,166,256	3,724,225
Total Current Investments	**4,166,256**	**3,724,225**
(b) Non - Current		
Shares and Debentures	4,167,355	4,307,908
Trust Units	65,033	65,032
Total Non - Current Investments	**4,232,388**	**4,372,940**
Total Investments	**8,398,644**	**8,097,165**

The Institute's investments are shown at cost. As at the 31 December 1996 the market value of the Institute's non-current investments was $6,067,897 (1995:$5,873,525)

9. Plant and Equipment		
Plant and Equipment (at cost or Board's valuation)	3,717,541	3,194,260
Less: Accumulated Depreciation	2,036,667	1,521,074
	1,680,874	**1,673,186**
Motor Vehicles under finance leases	273,182	296,522
Less: Accumulated Amortisation	98,659	50,246
	174,523	**246,276**
Written Down Value	**1,855,397**	**1,919,462**

10. Prepaid Grant

To date capital works grants of $3.6m have been received from the Federal Government for the redevelopment of the Institute. In accordance with our accounting practices, income and expenditure associated with the redevelopment project will be brought to account in the period to which they relate.

Prepaid Grant	**3,901,934**	**3,941,637**

11. Provisions		
(a) Current		
Annual Leave	354,381	322,951
Long Service Leave	242,608	299,632
Total Current Provisions	**596,989**	**622,583**
(b) Non - Current		
Long Service Leave	325,742	429,611
Deferred Maintenance	139,044	208,701
Total Non - Current Provisions	**464,786**	**638,312**
Total Provisions	**1,061,775**	**1,260,895**

NOTES TO AND FORMING PART OF THE ACCOUNTS

3. Government and Statutory Bodies

	1996	1995
	$	$
National Health & Medical Research Council	3,848,610	3,862,822
Victorian State Government	715,435	752,291
National Heart Foundation	469,094	473,975
Victorian Health Promotion Foundation	120,000	0
	5,153,139	5,089,088

4. Operating Fund

Balance at beginning of year	(3,032,717)	(1,956,505)
Deficit for year	(412,760)	(707,420)
Adjustment from change in accounting policy	0	(368,792)
Balance at end of year	(3,445,477)	(3,032,717)

5. Capital Fund

The Institute's Capital fund comprises the capital donations, bequests, receipts from fundraising activities and capital grants from government carried forward. Each year the Board allocates a proportion of these funds to supplement the research operations of the Institute. From time to time the Institute is the beneficiary under various wills and trust agreements. Such bequests and legacies are an unpredictable source of income each year. The amounts shown as income in the Income and Expenditure Statement represents the amounts transferred from this fund. The current balance is:

Balance at beginning of year	6,557,215	4,942,863
Surplus for year	523,095	1,659,612
Transfer to ANZ Trustees	0	(45,260)
Balance at end of year	7,080,310	6,557,215

6. Specific Purpose Fund

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 donors. Institute accounting records are kept so as to identify expenditure charged against income from these funds. All such income and expenditure is incorporated in the consolidated Income and Expenditure Statement. The current fund balance is:

Balance at beginning of year	475,549	741,916
Surplus / (Deficit) for year	258,774	(266,367)
Balance at end of year	734,323	475,549

7. Fund Balances

Balance at 1 January 1996	6,001,535	5,729,762
Transfer to ANZ Trustees	0	(45,260)
Adjustment from change in accounting policy	0	(368,792)
Surplus / (Deficit) for year -		
Operating Fund	(412,760)	(707,420)
Capital Fund	523,095	1,659,612
Specific Purpose Fund	258,774	(266,367)
	369,109	685,825
Balance at 31 December 1996	6,370,644	6,001,535

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 1996. These policies have been consistently applied unless otherwise indicated.

(a) Accrual basis

The accrual basis of accounting has been used with revenues and expenses being recognised as they are incurred, and brought to account in the period to which they relate.

(b) Historical cost

The financial statements have been prepared on a historical cost basis and except where stated do not take into account current valuations of non-current assets.

(c) Fund accounting

The Institute operates on a fund accounting basis and maintains three funds; Operating, Specific Purpose and Capital Funds. The work of the Institute is financed from grants, 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 convenants. The amount of grants received for specific purposes during the year but unspent at year end, will be generally expended in the next financial year. The Institute's capital fund comprises the capital donations, bequests and receipts from fundraising activities carried forward.

(d) Principles of consolidation

The Institute's accounts have been prepared on a consolidated basis. All inter-fund transactions have been eliminated on consolidation.

(e) Plant and equipment

Items of plant and equipment are recorded at cost or Board's valuation and are depreciated over their useful lives using the straight line method. Profits and losses on disposal of property, plant and equipment are taken into account in determining the result for the year.

(f) Leased Assets

Assets acquired under finance leases are included as property, plant and equipment in the balance sheet. Finance leases effectively transfer from the lessor to the lessee substantially all the risks and benefits incidental to ownership of the leased property. Where assets are aquired by means of finance leases, the present value of the minimum lease payments is recognised as an asset at the beginning of the lease term and amortised on a straight line basis over the expected useful life of the asset. A corresponding liability is also established and each lease payment is allocated between the liability and finance charge.

(g) Land and building

The land and 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 this building is $13m.

(h) Stocks

Stocks of consumable scientific and administrative items are stated in the Balance Sheet at the lower of cost and net realisable value. Cost is determined by the average cost method from computerised stock records.

(i) Tax status

The income of the Institute is exempt from income tax pursuant to the provisions of section 23(e) of the Income Tax Assessment Act. The Institute is also exempt from other government levies such as payroll tax and sales tax but not fringe benefits tax.

(j) Employee entitlements

Annual Leave

The Institute has fully provided for accrued annual leave entitlements for all employees as at balance date.

Long Service Leave

The liablilty to employee entitlements to long service leave represents the present value of the estimated future cash outflows to be made by the Institute resulting from employees' services up to the balance date. Liabilities for employee entitlements which are not expected to be settled within twelve months are discounted using rates based on government guaranteed securities, which most closely match the terms of maturity of the related liabilities. In determining the liability for employee entitlements, consideration has been given to future increases in salary rates, and the Institute's experience with staff departures. Related on-costs have also been included in the liability. It is Institute policy that employees with ten or more years of service qualify for long service leave entitlements.

(k) 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.

(l) Comparative figures

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

BAKER MEDICAL RESEARCH INSTITUTE

STATEMENT OF CASH FLOWS FOR YEAR ENDED 31 DECEMBER 1996

	Note	1996 $	1995 $
Cash Flows from Consolidated Activities			
Receipts from Granting Bodies		5,155,218	7,659,611
Donations and Bequests		4,143,256	3,527,235
Payments to Suppliers & Employees		(10,015,620)	(8,985,678)
Dividends Received		222,989	240,907
Interest Received		373,854	258,020
General Income		762,574	629,684
Net Cash from Consolidated Activities	14(b)	**642,271**	**3,329,779**
Cash Flows from Investing Activities			
Payment for Investment Securities		(1,514,690)	(1,707,629)
Proceeds from sale of Investment Securities		1,783,131	1,360,381
Payment for Plant & Equipment		(523,281)	(489,428)
Net Cash used in Investing Activities		**(254,840)**	**(836,676)**
Cash Flows from financing activities			
Principal Repayments under finance leases		(40,430)	(35,182)
Net Cash used in financing activities		**(40,430)**	**(35,182)**
Net Cash Increase in cash held		**347,001**	**2,457,921**
Cash at beginning of the financial year		5,029,360	2,571,515
Effects of exchange rate changes on cash held in foreign currencies		(13,994)	(76)
Cash at the end of the financial year	14(a)	**5,362,367**	**5,029,360**

The accompanying notes form an integral part of these financial statements

BAKER MEDICAL RESEARCH INSTITUTE

CONSOLIDATED BALANCE SHEET AS AT 31 DECEMBER 1996

	Note	1996 $	1995 $
ASSETS			
Current Assets			
Cash at bank and in hand		1,196,111	1,305,135
Debtors		214,773	188,969
Stock on hand		159,531	136,731
Prepayments		119,042	159,593
Accrued Interest		81,619	77,163
Investments (at cost)	8(a)	4,166,256	3,724,225
Total Current Assets		**5,937,332**	**5,591,816**
Non - Current Assets			
Plant & Equipment	9	1,855,397	1,919,462
Investments (at cost)	8(b)	4,232,388	4,372,940
Total Non - Current Assets		**6,087,785**	**6,292,402**
TOTAL ASSETS		**12,025,117**	**11,884,218**
LIABILITIES			
Current Liabilities			
Creditors		486,660	418,812
Lease Liability	2(f)	38,252	41,519
Prepaid Grant		3,901,934	3,941,637
Provisions	11(a)	596,989	622,583
Total Current Liabilities		**5,023,835**	**5,024,551**
Non - Current Liabilities			
Lease Liability	2(f)	165,852	219,820
Provisions	11(b)	464,786	638,312
Total Non - Current Liabilities		**630,638**	**858,132**
TOTAL LIABILITIES		**5,654,473**	**5,882,683**
NET ASSETS		**6,370,644**	**6,001,535**
FUNDS			
Accumulated Funds			
Operating Fund	4	(3,445,477)	(3,032,717)
Capital Fund	5	7,080,310	6,557,215
Specific Purpose Funds	6	734,323	475,549
Asset Revaluation Reserve - 1/1/93		2,001,488	2,001,488
TOTAL FUNDS	7	**6,370,644**	**6,001,535**

The accompanying notes form an integral part of these financial statements

BAKER MEDICAL RESEARCH INSTITUTE

CONSOLIDATED INCOME AND EXPENDITURE STATEMENT
YEAR ENDED 31 DECEMBER 1996

	Note	1996 $	1995 $
INCOME			
Government and Statutory Bodies	3	5,153,139	5,089,088
Baker Benefaction		1,050,000	911,569
Alfred Hospital		122,715	136,386
Fundraising, Corporate & Private Support		3,093,256	2,615,666
Investment Income		599,055	573,795
Clinical Services		268,370	232,108
General Income		546,571	700,384
Total Income		**10,833,106**	**10,258,996**
EXPENDITURE			
Salaries and Wages		6,425,975	5,995,624
Consumable Supplies		1,434,802	1,231,198
Minor Scientific Equipment		53,593	38,288
Depreciation / Amortisation		573,341	592,305
Laboratory Support Costs		665,816	649,374
General Overheads		889,513	626,565
Administration		330,789	387,650
Public Relations/Fundraising		90,168	52,167
Total Expenditure		**10,463,997**	**9,573,171**
CONSOLIDATED SURPLUS FOR YEAR	7	**369,109**	**685,825**
Represented by:			
Deficit from Operations		(412,760)	(707,420)
Surplus from Capital Fund		523,095	1,659,612
Surplus / (Deficit) from Specific Purpose Fund		258,774	(266,367)
Consolidated Surplus for Year		**369,109**	**685,825**

The accompanying notes form an integral part of these financial statements

Board Members Benefits

Since the end of the previous financial year, other than one Board Member who is a Director of a firm of Stockbrokers which has received, or become entitled to receive, fees for services rendered to the Institute on normal commercial terms, no Board member has received or has become entitled to receive any benefit, by reason of a contract made by the Institute or a related corporation with any Board Member or with a firm of which a Board Member is a member or with an entity in which any Board Member has a substantial financial interest other than the Director of the Institute, Professor J W Funder, who receives a salary.

Dated at Melbourne this 18th day of April 1997

Signed in accordance with a resolution of the Board of Management

Norman O'Bryan
President

John W. Funder
Director

BOARD MEMBERS REPORT

FOR THE YEAR ENDED 31 DECEMBER 1996

The Board of Management present their report together with the financial statements of the Institute for the year ended 31 December, 1996 and the auditors' report thereon.

Board Members

The Board Members in office at the date of this report are:

Mr N O'Bryan, President
Dr G P Johnston, Vice-President
Mr R E Barker, Hon. Treasurer
Professor J W Funder, Director
Mr P C Barnett
Mr K Baxter
Mr W P Gurry AO
Dr P G Habersberger
Professor S Holdsworth
Mr R Morris
Mr P Munz
Mr W G Philip AM
Mrs M Ross
Mr G Samuel

Principal Activities

The principal activities of the Institute are medical research into the basic causes of cardiovascular disease, to use this knowledge to help prevent heart and vascular disease in the community, and to improve medical and surgical treatment. No significant change in the nature of these activities occurred during the year.

Operating Result

The consolidated surplus of the Institute for the year amounted to $369,109 (1995:surplus $685,825) Income tax is not applicable.

Review of Operations

A review of the operations of the Institute during the year has been included in the President's and Director's report. The Institute's activities continued to be dedicated to medical research into the basic causes of cardiovascular disease. The Institute is a body corporate under an Act of Parliament and has no share capital.

State of Affairs

In October 1996 the final plans for reconfiguration of health services provided by the Inner and Eastern Health Care Network were released, including a continued major role for Alfred Hospital on its present site. The Board of this Institute anticipates the rebuilding of the Baker on the Commercial Road site, to re-establish our link with Alfred Baker Medical Unit. Negotiations with the Network Board on details and timing are currently in progress. It is not expected that the institute will move within the next 2-3 years.

Events Subsequent to Balance Date

There has not arisen in the interval between the end of the financial year and the date of this report any item, transaction or event of a material and unusual nature likely, in the opinion of the Board of Management of the Institute, to affect significantly the operations of the Institute, the results of those operations or the state of affairs of the Institute in subsequent financial years.

36. Jennings GL, Reid CM, Kay S, Jennings JA, Dart AM, Anderson WP. Animals and cardiovascular disease. In: ***Companion to animals in human health,*** C Wilson and DC Turner eds, Sage Publ, (in press)
37. Jennings GRL, Chin-Dusting JPF, Kingwell BA, Dart AM, Cameron J, Esler M, Lewis TV. Modulation of vascular function by diet and exercise. ***Clin Exp Hypertension*** (in press).
38. Kingwell BA, Sherrard B, Jennings GL, Dart AM. Four weeks of cycle training increases basal production of nitric oxide from the forearm. ***Am J Physiol*** (in press).
39. Komesaroff PA. Concepts of measurement of quality of life in health care. ***Bioethics*** (in press).
40. Komesaroff PA. The ethical conditions of modernity. In: ***Ethical Intersections,*** J Daly ed. Sydney, Allen and Unwin, (in press).
41. Komesaroff PA. Medicine and the moral space of the menopausal woman. In: ***Reinterpreting Menopause: Philosophical and Ethical Issues,*** PA Komesaroff, et al. eds, Routledge, (in press).
42. Komesaroff PA, Rothfield P, Daly J, eds. ***Reinterpreting menopause: philosophical and ethical issues.*** Routledge, (in press).
43. Krozowski ZS, Stewart PM, Obeyesekere VR, Li KXZ, Ferrari P. Mutations in the 11beta-hydroxysteroid dehydrogenase type II enzyme associated with hypertension and possibly stillbirth. ***Clin Exp Hypertens*** (in press).
44. Lambert G, Esler M. Origin of homovanillic acid in plasma. ***Am J Psychiatry*** (in press).
45. Liu J-P. Protein phosphorylation events in exocytosis and endocytosis. ***Clin Exp Pharmacol Physiol*** (in press).
46. Malpas SC, Groom AS, Head GA. Cardiac baroreflexes and cardiac hypertrophy in angiotensin II induced hypertension in rabbits. ***Hypertension*** (in press).
47. Meijer OC, Cole TJ, Schmid W, Schutz G, Joels M, de Kleot ER. Regulation of hippocampal 5-HT_{1A} receptor mRNA and binding in transgenic mice with a targeted disruption of the glucocorticoid receptor. ***Mol Brain Res*** (in press).
48. Mok SS, Even G, Li Q-X, Smith AI, Beyreuther K, Masters CL, Small DH. A novel metalloprotease in rat brain cleaves the amyloid precursor protein of alzheimers disease generating amyloidogenic fragments. ***Biochemistry*** (in press).
49. Morris MJ, Cox HS, Lambert GW, Kaye DM, Jennings GL, Meredith IT, Esler MD. Region-specific plasma NPY concentrations and overflows at rest and during sympathetic activation in man. ***Hypertension*** (in press).
50. Nagura H, Krozowski ZS, Silverberg SG. Localization of mineralocorticoid receptor and 11ß-hydroxysteroid dehydrogenase type II in human breast and its disorders. ***Anti-cancer Res*** (in press).
51. Nestel P, Simons L, Barter P, Clifton P, Colquhoun D, Hamilton-Craig I, Sikaris K, Sullivan D. A comparative study of the efficacy of simvastatin and gemfibrozil in combined hyperlipoproteinemia; prediction of response by baseline lipids, apo E genotype, lipoprotein(a), and insulin. ***Atherosclerosis*** (in press).
52. Nestel PJ, Pomeroy SE, Sasahara T, Yamashita TY, Dart AM, Jennings GL, Abbey M, Cameron JD. Arterial compliance in obese subjects is improved with dietary plant n-3 fatty acid from flaxseed oil despite increased LDL oxidizability. ***Arteriosclerosis Thromb Vasc Biol*** (in press).
53. Peters WS, Stevens JH, Smith JA, Rosenfeldt FL, Siegel LC, Burdon TA, Reitz BA. Port-access right cardiotomy: techniques for peripheral cardiopulmonary bypass with bicaval occlusion and cardioplegia. ***Eur J Cardiothoracic Surg*** (in press).
54. Rosenfeldt FL, Meldrum-Hanna WG. Saphenous vein grafts. In: ***Ischemic heart disease: surgical management,*** BF Buxton, et al. eds, Mosby Wolfe, (in press).
55. Rosenfeldt FL, Ostberg BN, Tcherkas DD, Pastoriza-Pinol JV, Southwell JE. A novel valveless pump for cardiac assist. ***Artificial Organs*** (in press).
56. Sasahara T, Yamashita T, Sviridov D, Fidge N, Nestel P. Altered properties of high density lipoprotein subfractions in obese subjects. ***J Lipid Res*** (in press).
57. Sato A, Canny BJ, Autelitano DJ. Adrenomedullin stimulates cAMP accumulation and inhibits atrial natriuretic peptide gene expression in cardiomyocytes. ***Biochem Biophys Res Commun*** (in press).
58. Sheppard KE, Wallace CA, Tetaz TJ, Smith AI. Studies on the hyper-activation and apparent glucocorticoid insensitivity of the hypothalamic-pituitary-adrenal axis of the genetically obese mouse. In: ***Stress: Molecular, Genetic and Neurobiological Advances,*** R McCarty, et al. eds, New York: Gordon and Breach Science Publishers, (in press).
59. Smith JA, Rosenfeldt FL. Ventricular free wall rupture. In: ***Ischemic heart disease: surgical management,*** BF Buxton, et al. eds, Mosby Wolfe, (in press).
60. Smith RE, Li KXZ, Andrews RK, Krozowski Z. Immunohistochemical and molecular characterization of the rat 11 beta-hydroxysteroid dehydrogenase type II enzyme. ***Endocrinology*** (in press).
61. Sviridov D, Sasahara T, Pyle L, Nestel P, Fidge N. Antibodies against HDL binding proteins enhance HDL uptake but do not affect cholesterol efflux from rat hepatoma cells. ***Int J Biochem Cell Biol*** (in press).
62. Thomas CJ, Rankin AJ, Head GA, Woods RL. ANP enhances bradycardic reflexes in normotensive but not spontaneously hypertensive rats. ***Hypertension*** (in press).
63. van den Buuse M. Effects of endothelins on the nervous system. In: ***Endothelins in Biology and Medicine,*** RC Miller, et al. eds, Series: ***Pharmacology and Toxicology: Basic and Clinical Aspects,*** MA Hollinger Ed Boca Raton: CRC Press Inc., (in press).
64. Webber KM, van den Buuse M. Intrastriatal injection of endothelin evokes dopaminergic turning behaviour in rats through activation of the ETB receptor. ***Brain Res*** (in press).
65. Wolfson AJ, Shrimpton CN, Lew RA, Smith AI. Differential activation of endopeptidase EC 3.4.24.15 toward natural and synthetic substrates by metal ions. ***Biochem Biophys Res Commun*** (in press).
66. Wong J, Rauhoft C, Dilley R, Agrotis A, Jennings G, Bobik A. Angiotensin converting enzyme inhibition abolishes medial smooth muscle PDGF-AB biosynthesis and attenuates their proliferation in injured carotid arteries. ***Circ*** (in press).
67. Woodcock EA. Inositol phosphates and inositol phospholipids: how big is the iceberg? ***Mol Cell Endocrinol*** (in press).
68. Woodcock EA. Analysis of inositol phosphates in heart tissue using anion-exchange high-performance liquid chromatography. ***Mol Cell Biochem*** (in press).
69. Woodcock EA, Lambert KA, Pham T, Jacobsen AN. Inositol phosphate metabolism during myocardial ischemia. ***J Mol Cell Cardiol*** (in press).

PUBLICATIONS

BAKER PUBLICATIONS IN PRESS

1. Andrews RK, Lopez JA, Berndt MC. Molecular mechanisms of platelet adhesion and activation. ***Int J Biochem Cell Biol*** (in press).
2. Autelitano DJ. Role of immediate early genes in the regulation of corticotroph function during stress. In: ***Stress: molecular, genetic and neurobiological advances,*** R McCarty, et al. eds, New York: Gordon and Breach Science Publishers, (in press).
3. Autelitano DJ, van den Buuse M. Concomitant up-regulation of proopiomelanocortin and dopamine D2-receptor gene expression in the pituitary intermediate lobe of the spontaneously hypertensive rat. ***J Neuroendocrinol*** (in press).
4. Bendle RD, Malpas SC, Head GA. Role of endogenous angiotensin II on sympathetic reflexes in conscious rabbits. ***Am J Physiol*** (in press).
5. Black J, Niklaus A, Bertram J, Dilley R, Bobik A. Vascular growth responses in SHR and WKY during development of renal (1K1C) hypertension. ***Am J Hypertens*** (in press).
6. Bobik A. Atherosclerosis resistance in rats correlates with lack of expansion of an immature smooth muscle population. ***J Vasc Res*** (in press).
7. Bobik A, Agrotis A, Little PJ. Vascular derived growth factors: potential role in the development of the tumour vasculature. In: ***Tumour Angiogenesis,*** Lewis, et al. eds, Oxford: Oxford University Press, (in press).
8. Bobik A, Dilley R, Kanellakis P. Cardiovascular hypertrophy in genetic hypertension. Regulation by the sympathetic nervous system and rilmenidine. ***J Hypertens*** (in press).
9. Bobik A, Dilley R, Wong J, Krushinsky A. Cytokines, neointimal hyperplasia and atherosclerosis. In: ***Lower Limb Ischaemia,*** KA Myers ed. London: Med-Orion Publishing, (in press).
10. Cameron JD, Jennings GL, Dart AM. Systemic arterial compliance is decreased in newly diagnosed patients with coronary heart disease: implications for prediction of risk. ***J Cardiovasc Risk*** (in press).
11. Cameron JD, Jennings GL, Kay S, Wahi S, Bennet KE, Reid C, Dart AM. The results of a self-administered questionnaire for the detection of unrecognised coronary heart disease. ***Austr NZ J Public Health*** (in press).
12. Challinor-Rogers JL, Rosenfeldt FJ, Du X-J, McPherson GA. Anti-ischaemic and anti-arrhythmic activities of some novel alinidine analogues in the rat heart. ***J Cardiovasc Pharm*** (in press).
13. Clifton PM, Noakes M, Nestel PJ. LDL particle size and lipoprotein response to dietary fat and cholesterol. ***Atherosclerosis*** (in press).
14. Curnow KM, Mulatero P, Emeric-Blanchouin N, Aupetit-Faisant B, Corvol P, Pascoe L. The amino acid substitutions Ser288Gly and Val320Ala convert the cortisol producing enzyme, 11ß-hydroxylase (CYP11B1), into an aldosterone producing enzyme. ***Nat Struct Biol*** (in press).
15. Dart A, Sherrard B, Salem H. Protein S, von Willebrand factor, and euglobulin lysis time as markers of coronary heart disease. ***Ann Intern Med*** (in press).
16. Dart AM, Sherrard B, Simpson HF. Influence of Apo E phenotype on postprandial triglyceride and glucose responses in subjects with and without coronary heart disease. ***Atherosclerosis*** (in press).
17. Douglas AM, Goss GA, Sutherland RL, Hilton DJ, Berndt MC, Nicola NA, Begley CG. Expression and function of members of the cytokine receptor superfamily on breast cancer cells. ***Oncogene*** (in press).
18. Du X-J, Bobik A, Little PJ, Esler MD, Dart AM. Role of Ca^{2+} in metabolic inhibition-induced norepinephrine release in rat brain synaptosomes. ***Circ Res*** (in press).
19. Esler M. High blood pressure management: potential benefit of I_1 agents. ***J Hypertension*** (in press).
20. Esler M. Sympathetic activity in experimental and human hypertension. In: ***Pathophysiology of hypertension,*** G Mancia and A Zanchetti eds, Series: ***Handbook of hypertension,*** Elsevier: Amsterdam, (in press).
21. Esler M, Kaye D, Lambert G, Esler D, Jennings G. Adrenergic nervous system in heart failure. ***Am J Cardiol*** (in press).
22. Esler M, Lambert G, Kaye D, Jennings G, Vaz M. Human obesity-related hypertension; brain neurotransmitters and regional noradrenaline spillover. ***Fundam Clin Pharmacol*** (in press).
23. Esler M, Lambert G, Vaz M, Thompson J, Kaye D, Kalff V, Kelly M, Turner A, Jennings G. Central nervous system monoamine neurotransmitter turnover in primary and obesity-related human hypertension. ***Clin Exp Hypertens*** (in press).
24. Evans RG. Current status of putative imidazoline (I_1) receptors and renal mechanisms in relation to their antihypertensive therapeutic potential. ***Clin Exp Pharmacol Physiol*** (in press).
25. Evans RG, Ludbrook J. Opioids: physiology, pharmacology and therapeutics. In: ***Scientific Foundations of Trauma,*** G Cooper, et al. eds, Oxford: Butterworth Heineman, (in press).
26. Fennessy PA, Godwin S, Head GA, Campbell JH, Campbell GR. Short-term and long-term cardiovascular actions of different doses of perindopril in the rabbit. ***Pharmacol Res*** (in press).
27. Fitzgerald SM, Stevenson KM, Evans RG, Anderson WP. Low dose angiotensin II infusions into the renal artery induce chronic hypertension in conscious dogs. ***Blood*** (in press).
28. Funder JW. Glucocorticoid receptors and mineralocorticoid receptors ; biology and clinical relevance. ***Ann Rev Med*** (in press).
29. Funder JW. Mineralocorticoid receptors. In: ***Receptors in Cardiovascular Disease,*** B Kobilka ed. (in press).
30. Funder JW, Krozowski Z, Myles K, Sato A, Sheppard KE, Young M. Mineralocorticoid receptors, salt and hypertension. ***Recent Progress in Hormone Research*** (in press).
31. Gaudet EG, Godwin SJ, Lukoshkova E, Head GA. Effect of central endogenous angiotensin II on sympathetic activation induced by hypoxia. ***Clin Exp Hypertens*** (in press).
32. Head GA, Burke SL, Chan CKS. Site and receptors involved in the sympathoinhibitory actions of rilmenidine. ***J Hypertens*** (in press).
33. Head GA, Burke SL, Chan CKS. Central imidazoline receptors and centrally acting anti-hypertensive agents. ***Clin Exp Hypertens*** (in press).
34. Head GA, Chan CKS, Godwin SJ. Central cardiovascular actions of agmatine, a putative clonidine-displacing substance, in conscious rabbits. ***Neurochem Int*** (in press).
35. Isenberg DA, Menon S, Rahman MAA, Ravirajan CT, Kandiah D, Longhurst C, McNally T, Williams WM, Latchman DS. The production, binding characteristics and sequence analysis of four human IgG monoclonal antiphospholipid antibodies. ***Arth Rheum*** (in press).

108. Sherrard B, Simpson H, Cameron J, Wahi S, Jennings G, Dart A. LDL particle size in subjects with previously unsuspected coronary heart disease: Relationship with other cardiovascular risk markers. ***Atherosclerosis*** 1996; 126: 277-87.

109. Smith RE, Krozowski ZS. 11 beta-hydroxysteroid dehydrogenase type I enzyme in the hearts of normotensive and spontaneously hypertensive rats. ***Clin Exp Pharmacol Physiol*** 1996; 23: 642-7.

110. Smith RE, Little PJ, Maguire JA, Stein-Oakley AN, Krozowski ZN. Vascular localisation of the 11beta-hydroxysteroid dehydrogenase type II enzyme. ***Clin Exp Pharmacol Physiol*** 1996; 23: 549-51.

111. Smith RE, Maguire JA, Stein-Oakley AN, Sasano H, Takahashi K, Fukushima K, Krozowski ZS. Localization of 11 beta-hydroxysteroid dehydrogenase type II in human epithelial tissues. ***J Clin Endocrinol Metab*** 1996; 81: 3244-8.

112. Smolich JJ, Cox HS, Eisenhofer G, Esler MD. Increased spillover and reduced clearance both contribute to rise in plasma catecholamines after birth in lambs. ***Am J Physiol*** 1996; 270: H668-77.

113. Speed CJ, Little PJ, Hayman JA, Mitchell CA. Underexpression of the 43kDa inositol polyphosphate 5-phosphatase is associated with cellular transformation. ***EMBO J*** 1996; 15: 4852-61.

114. Stevenson KM, Gibson KJ, Lumbers ER. Effects of losartan on the cardiovascular system, renal haemodynamics and function and lung liquid flow in fetal sheep. ***Clin Exp Pharmacol Physiol*** 1996; 23: 125-33.

115. Stewart PM, Krozowski ZS, Gupta A, Milford DV, Howie AJ, Sheppard MC, Whorwood CB. Hypertension in the syndrome of apparent mineralocorticoid excess due to mutation of the 11 beta-hydroxysteroid dehydrogenase type 2 gene. ***Lancet*** 1996; 347: 88-91.

116. Stomski FC, Sun Q, Bagley CJ, Woodcock J, Goodall G, Andrews RK, Berndt MC, Lopez AF. Human interleukin-3 (IL-3) induces disulfide-linked IL-3 receptor alpha-and beta-chain heterodimerization, which is required for receptor activation but not high-affinity binding. ***Mol Cell Biol*** 1996; 16: 3035-46.

117. Sudhir K, Jennings GL, Funder JW, Komesaroff PA. Estrogen enhances basal nitric oxide release in the forearm vasculature in perimenopausal women. ***Hypertension*** 1996; 28: 330-4.

118. Suzukawa M, Abbey M, Clifton P, Nestel PJ. Enhanced capacity of n-3 fatty acid-enriched macrophages to oxidize low density lipoprotein: mechanisms and effects of antioxidant vitamins. ***Atherosclerosis*** 1996; 124: 157-69.

119. Sviridov D, Pyle L, Fidge N. Identification of a sequence of apolipoprotein A-I associated with the efflux of intracellular cholesterol to human serum and apolipoprotein A-I containing particles. ***Biochemistry*** 1996; 35: 189-96.

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121. Thomas CJ, Woods RL, Evans RG, Alcorn D, Christy IJ, Anderson WP. Evidence for a renomedullary vasodepressor hormone. ***Clin Exp Pharmacol Physiol*** 1996; 23: 777-85.

122. Thomas WG, Thekkumkara TJ, Baker KM. Molecular mechanisms of angiotensin II (AT_{1A}) receptor endocytosis. ***Clin Exp Pharmacol Physiol*** 1996; 23 Suppl. 3: S74-80.

123. Thomas WG, Thekkumkara TJ, Baker KM. Cardiac effects of angiotensin II; AT_1 mediated actions in primary cardiac and CHO-K1 cells stably transfected with the AT_{1A} receptor. In: ***Recent adances in angiotensin II receptors,*** MK Raizada, et al. eds, New York: Plenum Press, 1996: 59-69.

124. Thomas WG, Thekkumkara TJ, Booz GW, Baker KM. Evidence against a role for protein kinase C in the desensitization of the angiotensin II (AT_{1A}) receptor. ***Eur J Pharmacol*** 1996; 295: 119-22.

125. Tipping PG, Huang XR, Berndt MC, Holdsworth SR. P-selectin directs T lymphocyte-mediated injury in delayed-type hypersensitivity responses: studies in glomerulonephritis and cutaneous delayed-type hypersensitivity. ***Eur J Immunol*** 1996; 26: 454-60.

126. Tkachuk V, Stepanova V, Little PJ, Bobik A. Regulation and role of urokinase plasminogen activator in vascular remodelling. ***Clin Exp Pharmacol Physiol*** 1996; 23: 759-65.

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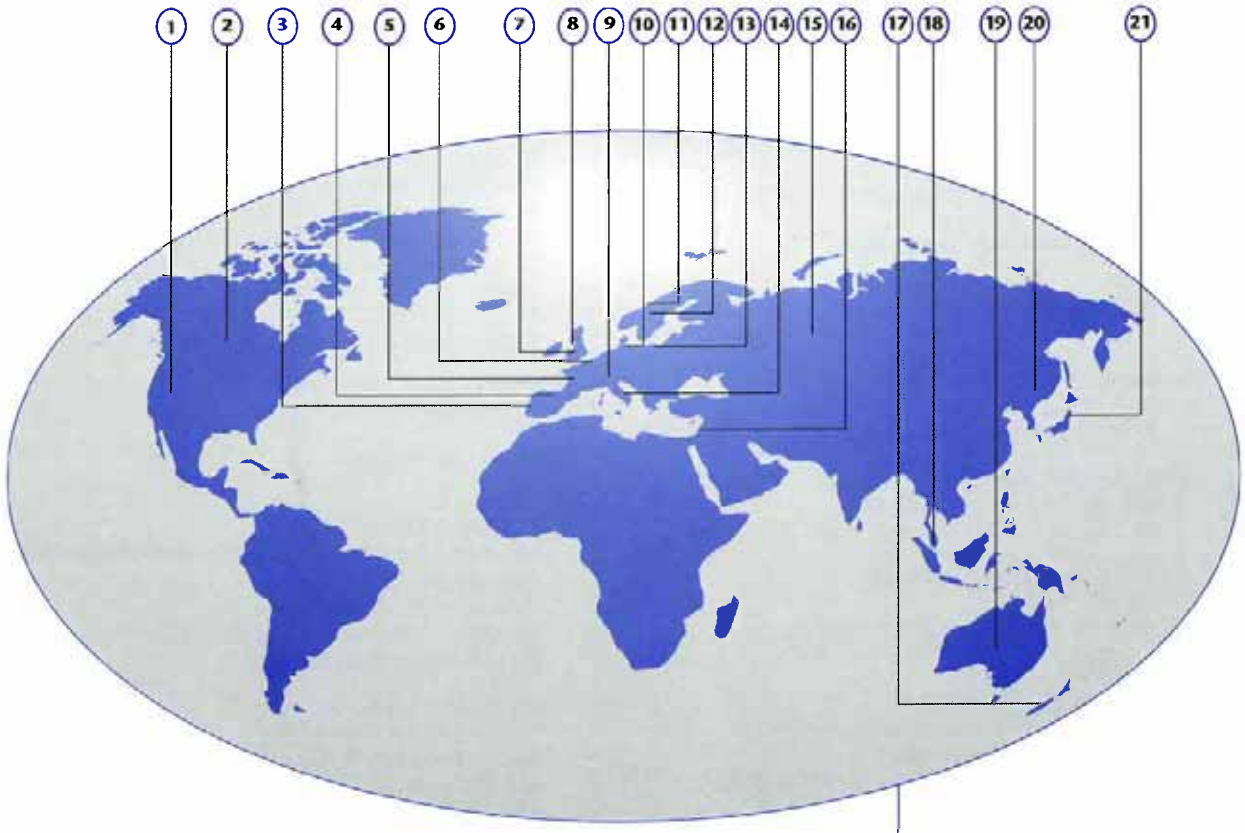
Ms Elizabeth Thomas SRN
Ms Rhonda Gerwing SRN
Ms Paulette Gardner SRN
Ms Barbara McComas SRN

AND WHERE WE WENT TO TELL THE NEWS



1996 - SEMINARS, MEETINGS AND LAB VISITS BY STAFF

1 USA
Houston, San Francisco, Orlando, New York, New Orleans, Washington, Keystone, Seattle, Monterey, Ann Arbor, Chicago, Boston, Indianapolis, Madison, Denver, Asilomar, Boulder, Detroit, Nevada, Reno, Los Angeles, Cleveland, Durham, St. Louis, Snowbird, Philadelphia, Salt Lake

2 Canada
Vancouver

3 Portugal
Lisbon

4 Spain
Barcelona

5 France
Lyon, Strasbourg, Paris

6 Netherlands
Groningen

7 UK
London, Birmingham, Nottingham, Cardiff, Leeds, Dublin,

8 Scotland
Edinburgh
Glasgow

9 Switzerland
Basel, Zurich, Interlaken

10 Germany
Berlin, Heidelberg, Stuttgart, Frankfurt

11 Norway
Oslo

12 Sweden
Gothenburg, Stockholm, Uppsala

13 Poland
Zacopane, Posnan

14 Italy
Naples, Pisa, Florence, Milan, Ancona

15 Russia
Moscow

16 Israel
Jerusalem

17 New Zealand
Christchurch

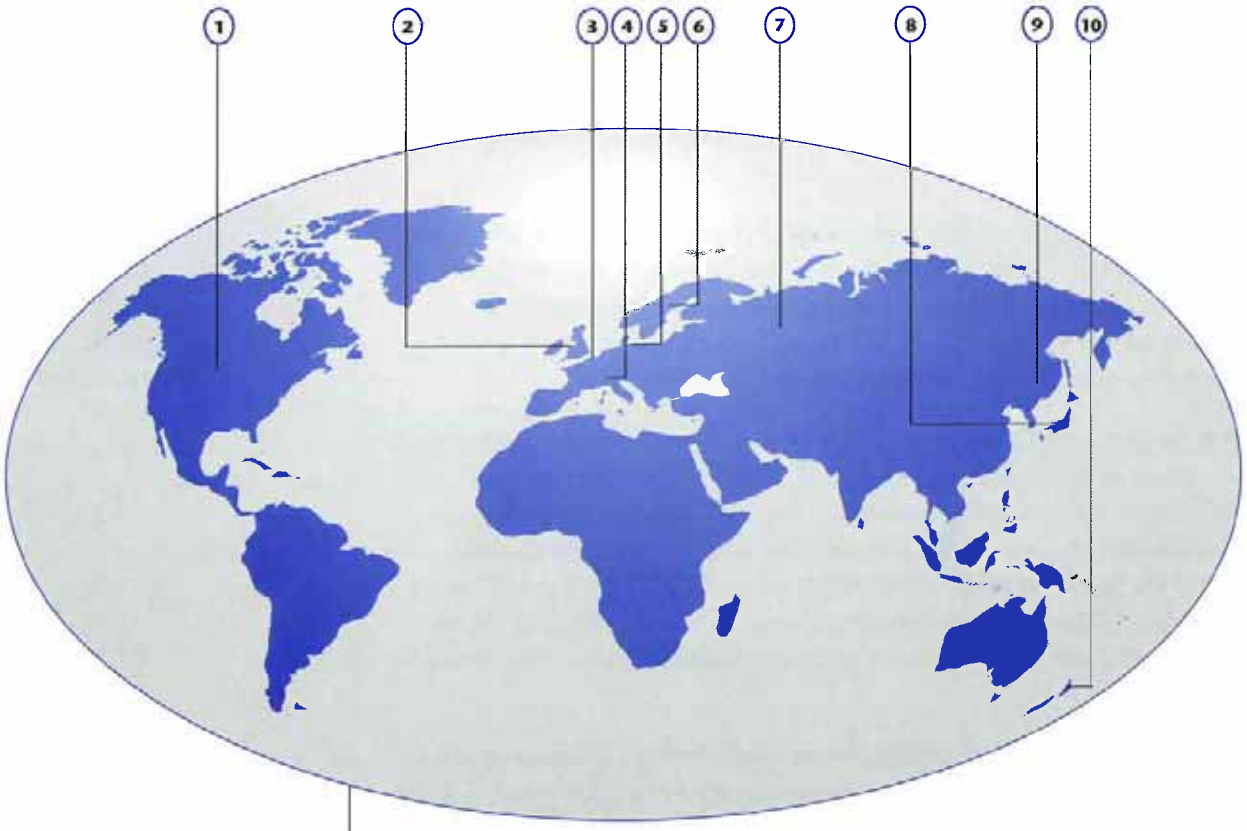
18 Singapore
Singapore

19 Australia
Melbourne, Lorne, Marysville, Hobart, Hervey Bay, Sydney, Adelaide, Fraser Island, Thredbo, Canberra, Brisbane, Perth, Cairns, Broome, Newcastle, Noosa

20 China
Hong Kong

21 Japan
Tokyo, Tokushima, Osaka

OUR WORLD HEALTH ROLE...



1996 - VISITING SCIENTISTS AT THE BAKER INSTITUTE

1	Dr Barbara Roland	Wisconsin	USA
	Dr Adele Wolfson	Boston	USA
2	Dr Tracey McNally	London	UK
3	Dr Ben J A Janssen	Maastricht	Netherlands
4	Dr Paolo Ferrari	Berne	Switzerland
5	Dr Claudia Rauhoft	Hamburg	Germany
6	Dr Goran Bergstrom	Goteborg	Sweden
7	Dr Alexei Mazurov	Moscow	Russia
	Dr Elena Lukoshkova	Moscow	Russia
	Dr Elena Parfenova	Moscow	Russia
8	Dr Takayugi Sasahara	Kumamoto	Japan
	Dr Takeshi Yamashita	Saitama	Japan
	Dr Fumihiro Tomoda	Toyama	Japan
	Dr Yoko Fujiwara	Ochanomizu	Japan
	Dr Atsuhisa Sato	Tokyo	Japan
9	Prof Chide Han	Beijing	China
	Prof Ming Yan	Beijing	China
10	Dr Zaw Lin	Dunedin	New Zealand

Paul Nestel - An Ounce of Prevention

'We are what we eat', like most five word sentences, is a gross oversimplification: eating rye bread doesn't give you hay fever, or chewing tobacco lung cancer. On the other hand, there's no denying the increasing realization of the role of nutrition in health and well-being. For centuries we have focused on famine and malnutrition, deficiencies of calories or essential nutrients. Today these remain world-wide problems, producing disease and death; but what is also now clear is that you can have too much of a good thing, and that malnutrition includes over- as well as under-nutrition. At the Baker, nutrition is Nestel.

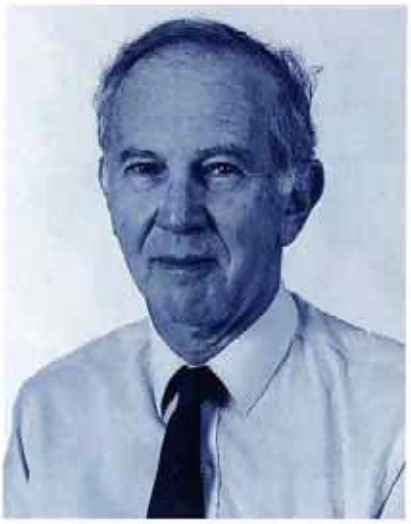
Paul Nestel is officially in retirement, from his position as Director of the CSIRO Division of Human Nutrition in Adelaide. Before his decade in Adelaide, he was Deputy Director of the Baker, and an internationally recognized clinical investigator in lipids and atherosclerosis. For the last two years, he has been back at the Baker, officially half time, in practice running from early morning to late afternoon each day.

What Paul runs is the Cardiovascular Nutrition Laboratory at the Institute, applying the techniques of modern investigative medicine to establishing how and why good nutrition works. Much nutritional research has been based on painstaking analysis of dietary differences in various ethnic and population groups - think of the protective effects of 'the Mediterranean diet'. Such studies are highly persuasive, but don't tell us whether what is important is the high carbohydrate, the low fat, the type of fat, the plant rather than animal protein, the red wine in moderation - or all of these, alone and in combination.

The Cardiovascular Nutrition Laboratory includes Sylvia Pomeroy, a research dietitian, and two Japanese postdoctoral fellows, Takayuki Sasahara and Takeshi Yamashita. Some of the studies they do have predictable results (like weight loss follows low fat diets), but are important for charting the cardiovascular effects of such a diet - how blood pressure falls, how plasma lipid profiles change, and how blood vessels respond, both to the body's own signals and administered drugs, over the period of low fat intake.

Other studies address different types of fat in the diet, comparing the effects on cardiovascular risk factors of saturated (animal) fats, versus fish oils versus diets high in linoleic acid. Other constituents of ethnic diets - tofu - also appear protective in a cardiovascular sense. Paul is exploring how tofu works in clinical studies, and similarly examining the effects of phyto (plant derived) estrogens as cardiovascular protection for post-menopausal women.

Thirty years ago cardiovascular disease accounted for over half of our death rate: now it stands at 40%, thanks to improved lifestyle and much better drugs. Medical care is not just doing tests and writing prescriptions; it's also about promoting good health, conveying the appropriate lifestyle messages on diet, exercise, and smoking etc. Such messages need to be scientifically grounded, and the ways in which the body responds clearly demonstrated: people with high blood pressure or cholesterol levels generally feel no pain until it's far too late. Paul Nestel, physician-scientist, is both explorer and translator, as an acknowledged expert in both nutrition and medicine. We expect him to contribute to the Institute for at least another decade post 'retirement' - as long as he remembers to eat and drink properly.



Paul Nestel, enjoying his retirement

Other studies address different types of fat in the diet, comparing the effects on cardiovascular risk factors of saturated (animal) fats, versus fish oils versus diets high in linoleic acid.

William Peters, a new generation of surgeons

Where Smaller is Better

The Alfred Hospital is the major trauma centre for the State of Victoria, for victims of road accidents and people requiring similar acute emergency care. While seat belts and safety helmets mean many people can walk away from an otherwise fatal accident, others are saved from death but not serious injury. In such circumstances wide-exposure surgery is often the only way to stabilize such a patient, commonly in a state of shock. With multiple fractures and lacerations, the surgeon's knife adds little to the pain, but very much to the gain.

In non-emergency situations, however, the patient is not commonly in a state of shock, and the less intrusive surgery is the better. Modern anaesthesia is itself minimally stressful, but the first incision through the skin sets off all the body's response systems. The bigger the wound, the more severe the reaction, the flatter the patient postoperatively, the longer the hospital stay. Both good medicine and health economics have an interest in day surgery, in keyhole approaches to removing gall bladders or ovarian cysts, and to arthroscopic surgery on troublesome knees.

Both good medicine and health economics have an interest in keyhole approaches.

For cardiac surgery you're not looking at an abdominal incision, which however extensive is through soft tissue. In cardiac surgery the sternum (breastbone) is split, and the two halves retracted to allow access to the heart. While artificial heart valves and coronary artery bypass grafts clearly benefit those who receive them, they come at a short-term (and sometimes medium-term) cost to the patient - of an often very difficult post-operative few weeks, and sometimes of months of nagging bone and cartilage pain as a result of wedging the chest open.

In the foreseeable future there are clearly still going to be reasons where this will remain necessary: It's not easy, for example, to envisage a heart transplant without this sort of access; for more common procedures, however, 'keyhole' approaches can and should be developed. In the procedure of angioplasty a long tube (catheter) is inserted into an artery in the thigh, fed up under video guidance into the narrowed coronary artery, and the artery stretched by inflating a tiny balloon at the catheter tip. Pretty invasive, you say - but nothing compared with sternum splitting. While not all narrowed arteries are amenable to angioplasty, most are: and the challenge over the next five years is to reduce the restenosis (renarrowing) rate post-angioplasty to zero, and thus drastically reduce the need for open chest, bypass graft surgery.

There's no substitute for turning painstaking research into evidence-based practice.

What William Peters has been doing in 1996, in Franklin Rosenfeldt's Cardiac Surgical laboratory at the Baker, is exploring the possibility of a different sort of keyhole cardiac surgery. In minimally invasive cardiac surgery the surgeon approaches the heart via an incision between the ribs, rather than by splitting the sternum. In 1996, with our collaborators at the Royal Children's Hospital, Australia's first keyhole atrial septal defect repair was performed. William and his colleagues also trialled over two dozen keyhole cardiac surgery procedures in greyhounds, testing not only the incision and approach to the heart, but also the way in which cardiopulmonary bypass can be achieved - so that the rest of the body gets properly oxygenated blood, even though the heart is stopped.

On the basis of these dog studies, in 1997 the Alfred cardiac surgical team will do a clinical trial of keyhole cardiac surgery in patients with single vessel disease unsuitable for angioplasty. These patients will be randomized to either the keyhole or conventional surgery, and the outcomes compared - in terms of postoperative pain, complication rate and duration of hospital stay. While we can predict the answers, there's no substitute for turning painstaking research into evidence-based practice - and people like William Peters are the crucial linkage in this process.

One of the things that Liz established early is that heart cells in tissue culture produce IP_3 (inositol with three phosphates), normally in the whole heart they produce IP_2, which blocks the generation of abnormal rhythms. This means that studies on whole rat hearts are useful (and less misleading) than on the more convenient cells grown in culture - but that's life. What Liz also noted was that when a rat heart was denied oxygen - or more specifically, when the oxygen supply was restored after it had been cut off - the heart responded by generating a burst of IP_3, and going into ventricular fibrillation, an arrhythmia characterized by very rapid, asynchronous, useless-for-pumping contractions. What she also found was that if she blocked the generation of IP_3 with a range of compounds - including the antibiotics neomycin and gentamycin - she also blocked the arrhythmia which otherwise followed.

Currently in clinical practice we don't have any very effective antiarrhythmic agents, which is a great pity. Most people who die acutely from a heart attack have ventricular fibrillation: you can survive a massive heart attack, with very high levels of tissue death, as long as the rate at which your heart beats stays reasonable. In the rat experiments the reperfusion-induced IP_3 peak and arrhythmia depend on the level of adrenaline released by the sympathetic (accelerator) nerves to the heart. Interestingly, the effect of adrenaline can be mimicked by the enzyme thrombin, released from the platelets involved in a coronary thrombosis (clot).

Now we need to fill in all the other pieces of the mosaic, at least as best we can. Liz is working with transgenic mice, which have deficient or superactive bits of the machinery linking the particular $alpha_1$-adrenergic receptors activated by adrenaline with the enzymes generating IP_3. Her colleague Dr. Du, appointed an NHMRC Research Fellow at the Baker this year, is tying off coronary arteries in rats and inducing heart failure in rabbits, and looking at the effects of blocking the $alpha_1$-adrenergic receptors with drugs, or agents that interfere with the action of thrombin. Tony Dart is the physician, awaiting the ability to translate these laboratory findings into clinical practice, and acting as the interface with industry in terms of the eventual development of clinically effective antiarrhythmic agents. Such agents, potentially based on the Woodcock/Du/Dart research, would prove of immeasurable value to millions of patients in alleviating premature mortality in patients with a history of heart disease, and equally importantly making a major contribution to their quality of life.

You can survive a massive heart attack, with very high levels of tissue death, as long as the rate at which your heart beats stays reasonable.

Tony Dart, in the laboratory and in the clinic

Elizabeth Woodcock, with her beloved high pressure liquid chromatograph

What Elizabeth Woodcock studies, at the level of cellular and molecular biology, is how the normal rhythm generator and the sympathetic (accelerator) nerves interact in very abnormal situations, like angina and heart failure.

Keeping the Beat

As most people realize, the heart has an intrinsic or built-in rhythm generator: a transplanted heart, for example, beats steadily before any of the nerves that control heart rate reconnect. Normally the heart accelerates in response to the release of noradrenaline from sympathetic nerves, and is slowed when the vagus nerve fires off and releases acetylcholine. What Elizabeth Woodcock studies, at the level of cellular and molecular biology, is how the normal rhythm generator and the sympathetic (accelerator) nerves interact in very abnormal situations, like angina and heart failure.

Liz came into the area as an expert in inositol phosphates, a family of molecules which act as part of the signalling apparatus between messages received at the cell surface, and the intracellular operations and machinery which they affect. The family members vary in terms of the number of phosphate groups they have, which in turn determines who they can talk to and what they can do in the cell. Inositol phosphates are generated in response to noradrenaline by the action of a series of enzymes, which release the inositol from the membrane lipid and then start adding and/or subtracting phosphates. Nobody's yet sure how complicated it is, except that it's very, very complicated.

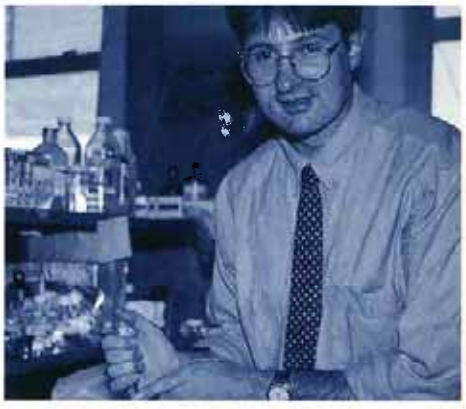
Tim Cole, the rest is up to the mice

omnipresent control system works, in areas as diverse as stress, obesity and blood vessel contractility. Just before he left Germany, Tim and a student successfully knocked out the mineralocorticoid receptor, the keyhole for the salt-retaining steroid aldosterone. Since his return he has cloned the enzyme 11ßHSD2 in the mouse, and is in the last steps of generating a knockout (touch wood: it's now up to the mice); at this stage, put it down as a technical knockout.

Kathleen Curnow is also a Melbourne University Ph.D, who forsook immunology for the Department of Pediatrics at Cornell in New York for six years as a postdoc, with another three in Paris at the College de France. KC (as she's known) is an expert in the enzymes in the adrenal gland responsible for shaping cholesterol into the stress hormone hydrocortisone and the salt-retaining hormone aldosterone. Over 90% of people with elevated blood pressure are said to have 'Essential Hypertension', which is medical for we don't know why it's raised. In a substantial percentage of this population there are tantalizing clues that all is not absolutely AOK in the adrenal gland, and that subtle alterations may wind up as elevated blood pressure.
The painstaking, patient-based, highly internationally collaborative studies on the adrenal gland, in which KC is a key player, may prove the key to what has become an increasingly embarrassing area of ignorance over the years.

Like KC, David Kaye is a Fellow of the High Blood Pressure Research Foundation of Australia. Unlike KC, Jun-Ping or Tim, he is an active clinician, a specialist cardiologist who then did a Ph.D here at the Baker. He then went off as a National Heart Foundation Overseas Fellow to Harvard for two years, and spent his time immersed in the molecular biology of the heart in general, and in particular how nerves talk to the heart (and interestingly, vice versa). He's now back in the clinic and the lab, where his projects run from cells through mice and rats to reversible models of heart failure in dogs: like the other three, there's no such thing as an eight hour day, or a forty hour week.

Send them away, to Heidelberg and New York and Paris and Boston, but bring them back home. Ring in the new: the future is yours.



David Kaye, home from Harvard

Jun-Ping Lui, He Li and the next generation

Send them away, to

Heidelberg and New York

and Paris and Boston, but

bring them back home.

Ring in the new:

the future is yours.

Kathy Curnow,
Melbourne, New York, Paris, Melbourne

Ring in the New

For many years most of the senior scientific staff of the Baker have been National Health and Medical Research Council Fellows, two year appointments, non-tenured, publish or perish. In 1996, for the first time, two of our senior staff - Dr. Jun-Ping Liu and Dr. Tim Cole - won Australian Research Council Fellowships. Because NHMRC funds a lot of very basic research, but ARC has responsibility for the biochemistry and cell biology which increasingly underpin biomedical research, the demarcation lines are increasingly blurred. What having ARC Fellows means, in short, is that the Baker is increasingly recognized as a place now focusing on very basic research, as well as the disciplines required for translating such research into clinical practice.

Jun-Ping Liu is a physician from Beijing, and a Monash Ph.D, who returned to Melbourne after four years as a postdoctoral fellow in Newcastle (NSW, not UK). Jun-Ping's area of expertise is in the way cells receive signals, particularly the way little bits of cell membrane are pinched off and internalized, so that the hormones and receptors can be disposed of or recycled. Many such cell processes are switched on and off by the addition of a phosphate group (phosphorylation), or its removal by enzymes called phosphatases. Jun-Ping charts these very basic waters, in cells from mouse pituitary and rat heart and human breast cancer, and for this work has collected a very impressive string of national prizes and awards over the past three years.

Tim Cole's Ph.D is from Melbourne, rather than Monash. On graduation Tim went to Heidelberg (FRG, not 3084) for a postdoctoral fellowship that extended to six years and two children. In the laboratory Tim 'knocked out' the glucocorticoid receptor, the keyhole in which cortisone-like stress hormones fit to produce their extraordinary range of effects throughout the body. This procedure is done in mice, rather than people, for the excellent reason that most (but not all) homozygous knockout mice (with a defective gene from both parents) die within hours of birth from poor lung inflation. Studies on the surviving homozygotes, and on heterozygotes (with one good and one defective gene), are enormously useful tools in working out how this

Hydrocortisone and corticosterone belong to a class of hormones called 'glucocorticoids', reflecting their ability to raise blood glucose and mobilize energy-providing mechanisms in response to stress. Glucocorticoids (like other hormones) circulate in the blood after their release, acting like keys in a lock by fitting into receptors (keyholes) in target tissue cells. Most hormones talk to a relatively restricted group of tissues, but glucocorticoid receptors are found in all the cells in the body except red blood cells and platelets.

What Karen has spent the last decade doing is looking through the keyhole, to try and see what happens on the other side. We've known since 1985 that the glucocorticoid receptor (or GR, for short) is a protein of 777 amino acids, and that it is found in the cytoplasm or cell sap. When the key fits in, however, things start to move - and the GR goes into the nucleus, to seek (and find) a particular run of DNA, which is usually just upstream of target genes. Once in place, the GR attaches itself to a whole raft of accessory molecules - in some of the pictures it looks like a fully equipped scuba diver - and then the whole lot triggers an enzyme called RNA polymerase to read heaps of copies of working drawings (RNA) off the blueprints (DNA). Providing there's enough bricks-and-mortar in the cell (and there usually is), working drawings are the rate-limiting step: so that glucocorticoids, like other steroid hormones, and thyroid hormone, and many vitamins, have their effect by altering the amount of particular proteins that are manufactured off the RNA working drawings.

Sometimes keys fit into more than one lock, as anyone who has broken off their boot key in the ignition knows only too well. For instance, glucocorticoids fit equally well into the receptors for aldosterone, which is a big problem. To overcome this, we make an enzyme in aldosterone target tissues that metabolizes glucocorticoids so that they no longer fit into the receptors for aldosterone - or, as it happens, into GR either. Recently, Karen has discovered a brand new receptor, a keyhole which fits modified (technically, 11-keto) glucocorticoids, which may be the way in which these very important stress hormones talk to cells that would otherwise be deaf to them. The story, in one sense, has just begun all over again: and now the challenge for Karen, and her students, is what does this new keyhole open, in terms of stress, and blood pressure, and all of the other things that glucocorticoids are known to do in our bodies.

The clue is in the coping, and nature has given us a complex array of mechanisms - in the nervous, immune and endocrine systems - to rise to challenges.

Karen Shepperd, looking remarkably unstressed

Hydrocortisone and corticosterone belong to a class of hormone called 'glucocorticoids', reflecting their ability to raise blood glucose and mobilize energy-providing mechanisms in response to stress.

Stress and the Steroid Receptor

Karen Shepperd

Stress is a word we all use but might have some difficulty in defining. Stress can be acute or chronic, personal or social, physical or psychological. We can be stressed by the challenges we face, and probably more by the feeling of not being able to cope with such challenges. While commonly we think of stress as 'a bad thing', it enables us to do things we would otherwise never believe possible. The clue is in the coping, and nature has given us a complex array of mechanisms - in the nervous, immune and endocrine systems - to rise to challenges.

In the adrenal gland two of these mechanisms meet and mingle. The adrenal gland is both nervous and endocrine system, an outpost of the brain and a factory making hormones. From the centre (medulla, in Latin) comes adrenaline, released in response to acute stress; from the outer part (the cortex) come steroid hormones, most notably hydrocortisone and aldosterone. For over 30 years the salt retaining hormone aldosterone, and its effect on blood pressure, has kept John Funder more or less out of mischief. For the last ten years, what Karen Sheppard has done is to study the stress hormone hydrocortisone (or more commonly corticosterone, its rat equivalent), and how it helps us cope in a whole range of ways.

These genes can be additional copies of mouse genes, or from rat, and so on. In addition, by hooking them up to the right homing signal we can increasingly confine their activity to particular tissues - brain, heart, whatever - which allows us to ask, and increasingly often answer, very exciting questions.

Gene knockouts are the opposite. Rather than 'overexpressing' (making too much of) a particular protein, in the KO mouse the gene coding for that particular protein has been disrupted in the embryo. If a page of aircraft carrier turns up in the blueprints for a 747, it won't fly, and it won't float. Many gene KOs are lethal-in-utero, showing that that particular gene is necessary for normal development. Other KOs have a mixture of predictable and unpredicted effects; still others show little if any effect, testimony to the resourcefulness of biology in improvisation.

If a page of aircraft carrier turns up in the blueprints for a 747, it won't fly, and it won't float.

When it is important to have rapid increases in animal numbers, mice are hard to beat, as most Mallee farmers can tell you. The current analytical tools available mean that we can address many questions on tiny tissue samples - like those from mice. For other questions - like how best to access the heart from between two ribs, rather than splitting the breastbone, or how can we keep coronary arteries open after surgery - models more or less of human size are still needed.

Experiments on animals are thus important stepping stones in health promotion and disease prevention. The Institute is very lucky to have Debra Ramsey, and her staff, to run the BRU. They do it productively and economically, and with a true respect for the animals under their care. They are the people who allow the Institute to bridge between benchtop and bedside, by translating theory into practice.

People might think of faster greyhounds or leaner pigs but, for human and veterinary medicine the current action is in what are known as transgenic and knockout mice.

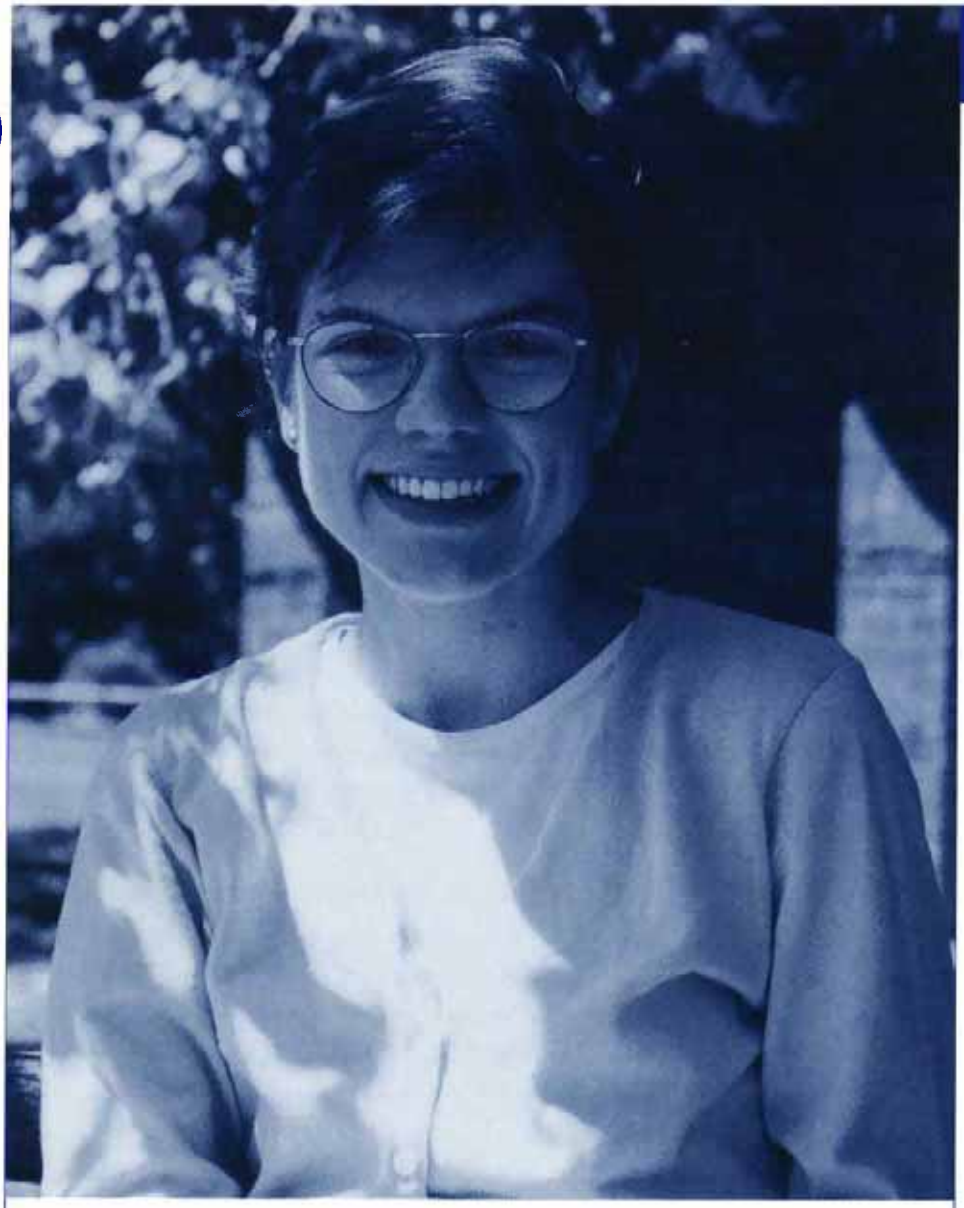
The Biological Research Unit: Bridging the Gap

We sometimes say that the Baker goes from benchtop to bedside, from cloning to clinic. True, but such a description emphasizes the ends of the spectrum, and pays little attention how the two ends can (and should) be linked. One of the critical steps between benchtop and bedside is experiments on animals, an area which has seen as many challenges and changes as any in medicine. Most of the laboratories at the Baker use experimental animals in one way or another. This section of the Research Report, therefore, deals with the changing nature of the use of animals in medical research in general, and at the Baker in particular.

If you had to sum it up in one sentence, where it used to be dogs and rabbits, it's now rats and mice. We still use dogs - for instance, in the development of keyhole techniques for cardiac surgery. We still use rabbits, for example to raise antibodies. Recently we have begun studies on minipigs, which have very human-like coronary arteries. There's no question, however, that rats and mice are the growth area: and it's worth reflecting on why this is the case.

The first reason is that researchers are generally fond of animals, and like most other people are much less unhappy about killing rats than dogs, however humanely. Second is the economics: the costs of looking after minipigs, in terms of staff time, are the same as for hundreds of rats. Third, and most recently, is our current ability to manipulate genes in animals. People might think of faster greyhounds or leaner pigs but, for human and veterinary medicine the current action is in what are known as transgenic and knockout mice.

In a transgenic mouse an extra gene (blueprint) coding for a particular protein is injected into the very early embryo, so that when the mouse develops it will make rather more of that particular protein than normal. There are lots of pitfalls - getting the blueprints into cells in good shape, and in such a way that the working drawings (RNA) can be read off them properly, and so on - but currently it's a very powerful technique to look at the consequences of having too much of what is normally a good thing.

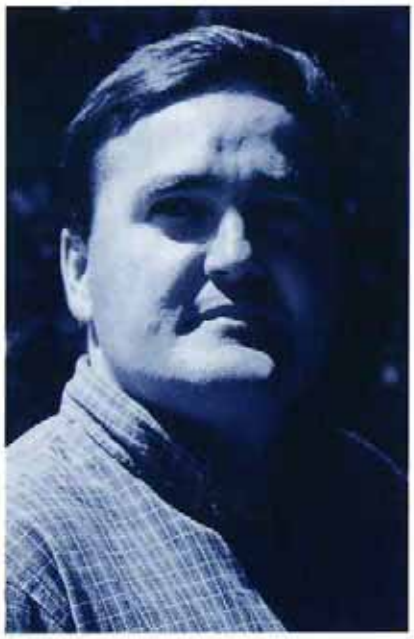


Morag Young, facing life in Dallas

In November 1996 our latest CJM's were announced. Karen Anderson did her Ph.D on cardiac arrhythmias with Liz Woodcock and Tony Dart, and has spent this year in the Monash Department of Medicine at Box Hill, where she will return after her two years in Cambridge. Morag Young did her Ph.D under John Funder's supervision, in the most elastic sense of the word, on how excess salt and salt-retaining hormones cause the heart to enlarge and become infiltrated with fibrous tissue. In 1997 she is off to Dallas, to study how to make mice with various gene knockouts in this or that tissue, before returning to Tim Cole's laboratory at the Baker and her work on cardiac fibrosis.

Ave atque vale, CJM's - and, on very bad days, *morituri vos salutamus.*

Cathie Coulter, asking questions of the placenta



Wally Thomas, gazing steadfastly into the future

C J Martins.

Currently at the Baker

we have two 'back here',

one 'over there', and

two about to go.

C J Martin Fellowships – Launch and Re-entry

Each year the National Health and Medical Research Council awards around half a dozen CJ Martin Fellowships to our brightest and best young Ph.D graduates. CJM's are a four year proposition, two overseas and two back in Australia. Currently at the Baker we have two 'back here', one 'over there', and two about to go.

Cathie Coulter is a Monash Ph.D, who took off for San Francisco in 1990 and started her CJM in 1992. Cathie is a reproductive biologist from her time at Monash, through Professor Bob Jaffe's laboratory at UCSF, to an additional couple of years on staff in London, Ontario: why the Baker, you might ask, for her two years back in Australia? The answer is the enzyme 11ßHSD2, cloned by Anthony Albiston and Zig Krozowski here in 1994, and the subject of intense continuing investigation in several Institute laboratories. Zig and his group are interested in what it does in the kidney, Karen Sheppard in the colon, Barb Roland in the brain - all directly related to its role in salt handling and blood pressure. But it's also very active in the placenta, which is Cathie's area of expertise. In the placenta it does other things, crucial for the normal development of the foetus - which is why a reproductive biologist has taken her time back here at the Baker.

Wally Thomas did his Ph.D at the University of Queensland on the hormone angiotensin, which constricts blood vessels and stimulates secretion of the salt retaining hormone aldosterone. These are key events in blood pressure control, in hypertension we use ACE inhibitors, to block the conversion of inactive precursor to active angiotensin. Wally went off in 1993 to the Weis Research Center in Pennsylvania, to work on the receptors for angiotensin, the keyholes in the cell membrane into which the hormone fits like a key. His hosts in the USA persuaded (and paid) him to stay for a third year, and early in 1996 he finally arrived back in Australia, where he rapidly established his laboratory at the Baker with a portfolio of projects on angiotensin action.

Gavin Lambert took off in 1996 for two years in Uppsala as the overseas component of his CJM. Gavin had worked as a research assistant with Murray Esler before doing his Ph.D, on the way that chemical messengers in the brain can be monitored in jugular vein blood, and how these are very much altered in a variety of cardiovascular disorders. The laboratory in Sweden is primarily 'neuro' where the Baker is 'cardio' - but Murray Esler can't wait for him to return, to expand the work of the Human Neurotransmitter laboratory at the leading edge of the interface between brain function and the cardiovascular system.

In the old days such studies were done by pharmaceutical firms, to sell a product and to make money; ANBP2 is a collaboration between the pharmaceutical industry and the Commonwealth, to save lives and to save money. Where the taxpayer kicks in is that all the pathology testing is done under Medicare - a very considerable contribution to knowledge, as well as to individual patient well-being. Where the industry money goes is in supporting the research workers involved, and in providing gratis the drugs used. It's a partnership, and a model for how drug trials can and should be run.

From the point of view of laboratory research, pipettes and test tubes are where it starts, and ANBP2 is where it finishes up. From the point of view of the consumer, ANBP2 might often be where it starts, and we need to take it back from there. People in the community with no background in biology or medicine often have a very keen appreciation of testing, of quality control, of post-marketing surveillance.

The processes of ANBP2 - setting targets, recruiting, getting out in the field, making sure everyone stays in the loop and happy - are common to a whole range of human activities; the curiosity-driven laboratory worker in a sense has more affinity with a poet or a prospector. The important thing is to do both, to do both well, and to do both where the differences in approach can build something bigger than the sum of the parts. That's what ANBP2 is doing, and that's why we're proud it's part of the Baker.

Dedicated research nurses go out into general practitioner's surgeries all over Australia, working with hundreds of local doctors in the city and country.

Australian National Blood Pressure Study

Our stereotype of a medical research institute, tends to be pipettes and test tubes and microscopes. In real life it's rather more complex, and some of the most necessary research is done long after the pipettes and test tubes are thrown away, the microscope turned off. The second Australian National Blood Pressure Study, or ANBP2, is an example of this latter sort of research. It's one of the areas in which the Baker does applied research, under the direction of Chris Reid - whose Ph.D. is, appropriately, in community medicine rather than test tubes and microscopes.

ANBP2 is looking at not just how effective the two measures are in lowering blood pressure, but much more importantly in preventing heart attacks and strokes.

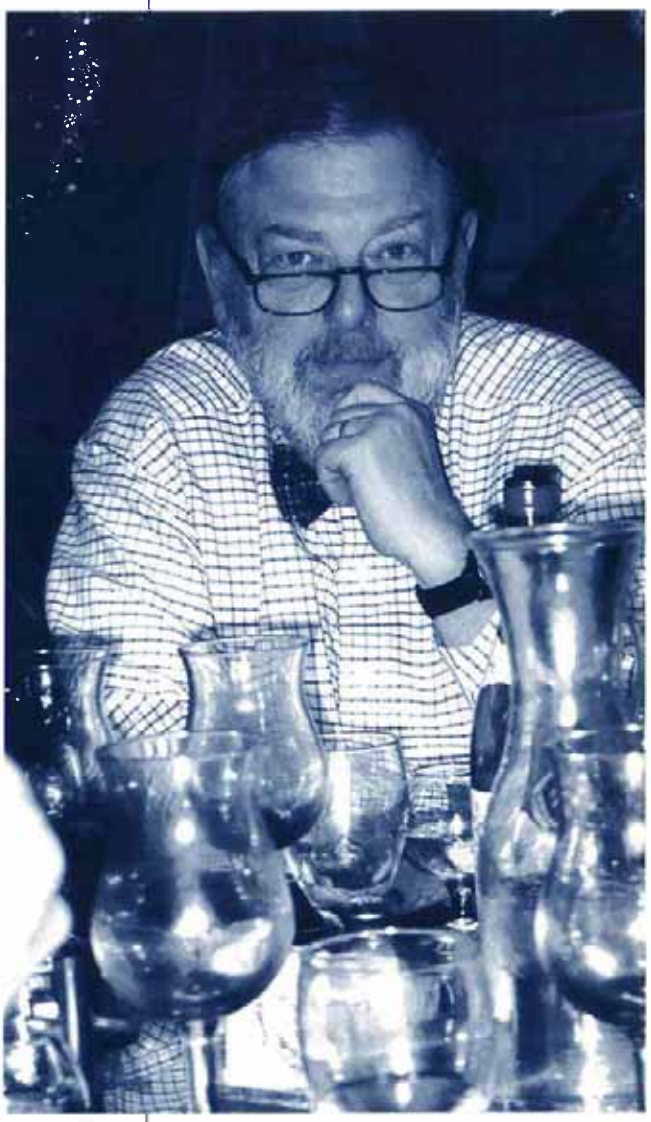
ANBP2 sounds deceptively simple. It looks at 6,000 relatively senior citizens over a period of five or more years, half of whom are on standard diuretic therapy for their high blood pressure, with the other half on the newer ACE inhibitors. The question is straightforward, whether ACE inhibitors (which are relatively expensive) are more effective than diuretics (which have more side effects). What distinguishes it, among other things, from previous comparisons is the spin on the word 'effective': ANBP2 is looking at not just how effective the two measures are in lowering blood pressure, but much more importantly in preventing heart attacks and strokes, the actual outcomes or 'end-points'.

In the old days it might have been possible to attract 6,000 mature age hypertensives to specialist clinics at the Baker, although on average for every patient seen only one in five is actually enrolled in the trial, and we'd have to turn half the Institute into waiting rooms. Today high blood pressure is routinely managed in general practice, so that to be relevant the study has to be in a general practice setting, rather than in the specialist clinic - and so that's where ANBP2 is.

What this means is dedicated research nurses going out into general practitioner's surgeries all over Australia, working with hundreds of local doctors in the city and country, accumulating suitable patients, randomizing the treatment they are to receive, taking histories and sending blood samples off for tests, and finally then entering the results onto diskettes for accumulation and statistical analysis at the University of Adelaide.

I would also like to thank with equal sincerity the Board of the Institute, and in particular the President Norman O'Bryan. 1996 for the first time saw an all day 'retreat' for the Board members and senior staff, an extremely productive exercise and one which we will repeat annually. The Board has been a constant source of guidance, support and outreach into the wide Victorian and Australian community: for their enthusiasm for, and commitment to, the Baker I and the Institute staff are enormously grateful. In addition to our current Board, our Past Presidents John Habersberger, Laurie Muir, John Moir and Don Hogarth have continued their very active support of and involvement in the work of the Institute.

The outlook for 1997, the year of our next quinquennial review (1998-2002), looks very good. Scientific productivity, gauged by numbers of publications and their impact factor, has risen progressively and impressively over the past five years. In 1996 our funding from the National Heart Foundation jumped to almost double that in previous years. In 1992 we had a relatively narrow bridge, from predominantly physiology and pharmacology to clinical investigation; at the end of 1996 our span is much wider, from the Australian National Blood Pressure trial and Paul Nestel's public health studies to an unprecedented emphasis on the cellular and molecular biology that underpins contemporary biomedical research. The Institute staff are increasingly taking on leadership roles - locally, nationally and internationally - and the star of the Baker has never stood higher. The next five years should see the cloning of the entire human genome, a period of unparalleled efflorescence for human biology and medicine. For the Baker it is a period of excitement and anticipation, in terms of the research that we can do, and the return we can thus make on the investment made in the Institute by the people of Australia.



Two glasses of red wine a day...
John Funder, Director of the Baker Medical Research Institute

John Funder
Director

biology in Harvard, as a National Heart Foundation Overseas Fellow: David is also a specialist cardiologist, and as such ideally placed to continue the Institute's remit of from benchtop to bedside. In March we welcomed two returning C.J. Martin Fellows, Cathie Coulter to spend two years working in Zig Krozowski's laboratory, and Wally Thomas to continue his molecular biology studies on receptors for the powerful vasoconstrictor (and thus blood pressure raising) hormone angiotensin. In October he was joined by Kathy Curnow, with nine postdoctoral years in New York and Paris, to work on angiotensin receptors and the enzymes that make adrenal steroids. Late in the year Kathy Curnow and David Kaye won High Blood Pressure Research Foundation of Australia Research Scholarships, for their salaries and laboratory support in 1997-98.

Although attracting staff of such calibre is one index of the rude good health of the Institute, another is the contributions made by the Baker as a whole in 1996. In June the Biennial meeting of the International Society for Hypertension in Glasgow followed the quadrennial International Society of Endocrinology in San Francisco. Institute staff gave five plenary lectures/ invited symposium presentations, and over twenty 'free communications', an unprecedented degree of exposure in the two areas. For the past six years one of the service roles of the Institute is to measure levels of hormone receptors in breast cancer specimens, up to 1000/year; in 1996 the Baker is part of the developing 'Institute without walls' concept of the Victorian Breast Cancer Research Consortium, into which the Victorian Government is putting $3m per year for ten to fifteen years. Two of our PhD students (Karen Anderson, supervised by Elizabeth Woodcock, and Morag Young by John Funder) were awarded C.J. Martin Fellowships, the top NHMRC awards for overseas postdoctoral study, in November 1996.

Finally, Michael Berndt - who came to the Baker in 1991, soon after the completion of his Wellcome Senior Research Fellowship in the Sydney University Department of Medicine at Westmead Hospital - was awarded the inaugural GlaxoWellcome medal in November 1996. From 1980 onwards the Wellcome Medal has constituted Australia's most significant acknowledgement for outstanding biomedical research (or research into the prevention of harvested crops) over the previous five years, by someone in mid-career. In 1995 Julie Campbell (BMRI 1979-92) won the last Wellcome award, after John Funder in 1987 and Murray Esler in 1989. Michael is the third or fourth Baker person, depending how you count, to have won the award - and brought great credit to the Institute, both by his research and by its recognition in this way.

1996 has also seen an expansion and strengthening of the Institute executive. Garry Jennings has become Deputy Director of the Baker - in addition to his positions as Director of Cardiology Services, and Director of the Alfred Baker Medical Unit. Murray Esler and Michael Berndt are the new Associate Directors of the Baker, and Noel Fidge and Alex Bobik, as Senior Principal Research Fellows, have also joined the executive. The seventh member, Adrian O'Brien, is the Institute Financial Director and has taken on in addition a number of key administrative roles in 1996. For the contributions of all the members of the executive in general, and to Adrian for his tireless commitment to his expanded role in particular, I would like to offer my very sincere personal thanks.

DIRECTOR'S REPORT

1996 has been a year of high scientific achievements, of growth and evolution in terms of the Institute staff, and of considerable frustration in terms of building 'the new Baker'. Some - but not all - of the scientific achievement is detailed in the research reports that follow; many - but not all - of the people who started at the Baker in 1996 are similarly highlighted; and late in the year, at long last, the Victorian Government accepted the metropolitan hospitals network plans in detail, ensuring the continued operation of the Alfred as a major center for clinical service, teaching and research. This allows us to proceed with our (and the Inner and Eastern Health Care Network's) plan to relocate, holus bolus, to a front-of-house site on Commercial Road.

The additional building initially proposed across Baker Lane was costed at ~$14m, and we currently have $4m from the Commonwealth, $4m from the Victorian Government, and $4m from private and philanthropic supporters. A completely new building, relocating the Institute on Commercial Road back in apposition with ABMU, will cost $21m, and we thus need an additional $3m from each of the above sources of support. It is our very sincere hope that the additional Commonwealth funding will be found in the first half of 1997, that it will be matched by the Victorian Government and on that basis we can start the planning and detailed design phase for the new Baker shortly after the May Budget.

Although in 1996 the Institute welcomed a number of new staff members, we also farewelled others, most notably Warwick Anderson. Warwick worked at the Baker for over twenty years, most recently as Associate Director (1990-2) and thereafter Deputy Director, until taking over as Chairman of the Department of Physiology at Monash at the end of March. Monash Physiology has more NHMRC support than any other Monash department, or any other physiology department in Australia, and in Warwick it has attracted a very broadly based biomedical scientist and an outstanding administrator. In 1994-96 Warwick was deputy chair of the Medical Research Committee of NHMRC, and will chair the MRC in 1997-99. For his contributions over two decades to medical research in a multitude of ways, and to the Institute in particular, the Baker is very much in his debt.

Roger Evans accompanied Warwick to Monash as a senior research officer, with a considerable level of continuing collaboration with Institute staff. Kathy Stevenson, an NHMRC postdoctoral fellow working on hormones and renal function, similarly relocated to Monash. Simon Malpas, who worked with Geoff Head and Warwick, returned to his native New Zealand as a lecturer in Physiology after postdoctoral experience in Oxford, Osaka and Melbourne. Atsuhisa Sato and Barbara Roland, both visiting scientists in the Molecular Physiology laboratory for three years, returned at the end of the year to staff positions in the Mito Red Cross Hospital, Japan, and the Salk Institute, La Jolla, respectively. Judy Segal became the administrator at the Australian Israel Chamber of Commerce, and our photographer Neil Potter opted for private photographic practice.

For each new alumnus, however, there is a new staff member. At the beginning of the year Jun-Ping Liu arrived as an Australian Research Council Queen Elizabeth II Fellow, a five year fellowship appointment and recognition of the Institute's expanding strength in the basic biochemistry and cell biology underpinning biomedical research. In February David Kaye arrived back from two years molecular

Although attracting staff of such calibre is one index of the rude good health of the Institute, another is the contributions made by the Baker as a whole in 1996.

PRESIDENT'S REPORT

Norman O'Bryan,
President of the Baker Board of Management

1996 was another year of great achievement for the Baker Medical Research Institute. The Director has summarized the excellent achievements of our scientists in this year. The Board congratulates all the scientists of the Institute and its support staff, who have contributed so well to the continued success and growing national and international reputation of the Baker during 1996. In particular the Board salutes Michael Berndt, David Kaye, Kathy Curnow, Karen Anderson, Morag Young and the other Institute scientists whose work has received well-deserved public recognition and accolade.

It has been a challenging and exciting year for the Board as well. As members and supporters of the Institute are well aware, the Board has worked many years on a proposal to rebuild the Institute to provide a working environment appropriate for Australia's major cardiovascular research institute. A considerable amount of money has been raised from the Commonwealth and State Governments and from the public. The turmoil and uncertainty surrounding the Victorian hospital system (and in particular the future role of the Alfred Hospital in that system) have meant that it has not been sensible to contemplate spending that money, until we could be certain about the future of the practice of cardiovascular medicine on the Alfred Campus.

I am pleased to say that it appears that at last the clouds have cleared over this issue. We now have confirmation that cardiovascular medicine on a substantial scale will continue to be practiced on the Alfred Campus, and the Network board will grant the Baker a long term lease for a peppercorn rent of the land directly adjacent to the East block of the hospital with a frontage on Commercial Road. This prime piece of real estate will be a part of what is expected to become a major new academic and research precinct at the hospital. That's the good news.

The only difficult remaining part is, of course, raising the money to pay for it. Whereas our 1995 capital campaign had provided the funds to complete our proposed building on the originally chosen site on the RVIB land, we will need to raise approximately $9 million more to build on the new proposed site. We have accordingly requested the Federal and State governments to join in a partnership with our Capital Appeal Committee to provide $3 million each to ensure the new building can go ahead. At the time of writing there is no guarantee that we will receive this additional money. The Board of course cannot recommend proceeding unless the money is pledged. Nevertheless, we are quietly confident that in 1997 we will achieve at last our great objective, and turn the first sod on the rebuilding of the Baker with a view to occupying brand new, purpose built facilities to take the Institute into the 21st century.

I would like to express my gratitude to the Director, the Deputy Director and the staff of the Institute, to my fellow members of the Board and in particular to the members of the Capital Appeal Committee who have worked so tirelessly in an endeavour to resolve this longstanding major issue before the Institute. Our facilities are currently second rate, and it is my fervent hope and that of the Board that by the time we come to report to you next we shall be happily reporting on the successful commencement of our building works.

Norman O'Bryan

Norman O'Bryan
President

BOARD OF MANAGEMENT

Sir Laurence Muir Ktcr
VRD, LLB, FSIA, FIAM
Patron of the Institute and former President of the Board of Management

Mr Norman O'Bryan
BA , LLB, BCL
President, Baker Board of Management
Barrister-at-Law

Professor John Funder
MD, PhD, FRACP
Director of the Baker Medical Research Institute

Photo courtesy of The Herald and Weekly Times

Mr Ken P Baxter
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KPMG

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Member, Australian Stock Exchange

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BSc, PhD
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Past President, Minerals Council of Australia
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Visiting Cardiologist, Alfred Hospital
Director, Cardiac Services
St. Francis Xavier Cabrini Hospital

Professor Stephen R Holdsworth
MD, PhD, FRACP
Director of Clinical Immunology
Monash Medical Centre

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Group Executive Chairman
GSA Industries Pty Ltd

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B.Comm, FCA

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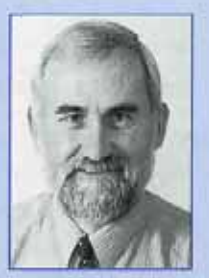
Chairman, Baker Capital Campaign
Deputy Chairman
SBC Warburg Australia Limited

Mrs Margaret Ross
Convenor, Baker Activities Committee
Chairman, Board of Management
Fintona Girls' School



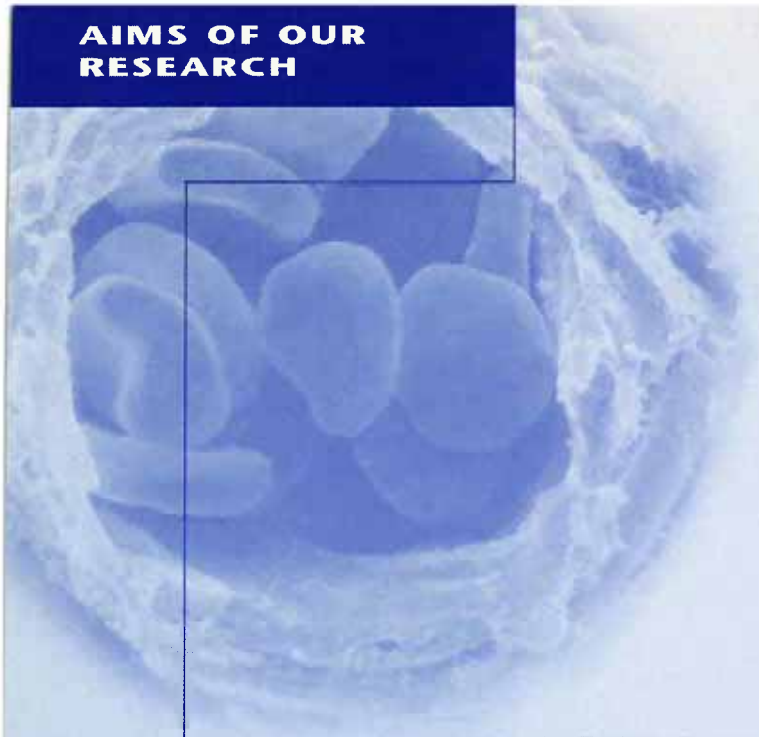
Mr Richard Morris
NHMRC Representative
Secretary to MRC

Mr Graeme J. Samuel LLB, LLM
Chairman, Inner and Eastern Health Care Network Board
Company Director

PAST PRESIDENTS

J. F. Mackeddie	**E. Rouse**	**J. C. Habersberger AO**	**Sir Laurence Muir Ktcr**	**J. D. Moir AM**	**D. F. Hogarth OAM**
BA, MD, BS	CBE, FRACR (Hon)	B. Comm	VRD, LLB, FSIA, FAIM	1987 - 1992	Bsc
1929 - 1944	1944 - 1971	1972 - 1983	1984 - 1986		1992 - 1994

AIMS OF OUR RESEARCH



In Australia, 50% of all deaths and serious illness are due to disease of the heart and circulation.

Most of them are due to Hypertension (High Blood Pressure) and Atherosclerosis (clogging of the arteries with fatty cholesterol-laden plaques) which cause stroke, 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 kind of knowledge to help prevent heart and vascular disease in the community, and to improve medical and surgical treatment.

CONTENTS

DIRECTORY

Auditor

Price Waterhouse

215 Spring Street, Melbourne, Vic. 3001

Solicitors

Blake Dawson Waldron

101 Collins Street, Melbourne, Vic. 3001

Annual General Meeting

Tuesday 6th May

Baker Medical Research Institute

5.00pm

Baker Medical Research Institute

Commercial Road, Prahran

P.O. Box 348, Prahran, Victoria 3181 Australia

Telephone: (03) 9522 4333