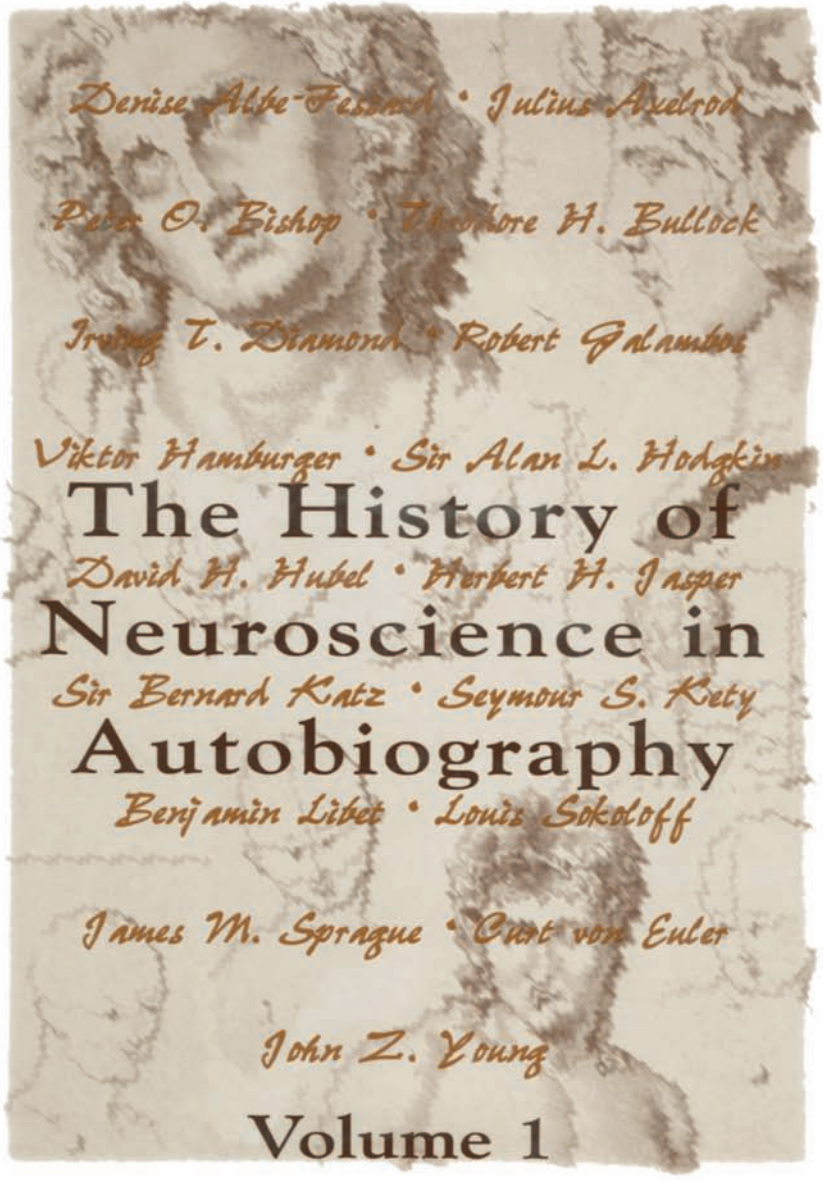


Edited by Larry R. Squire

The History of Neuroscience in Autobiography

VOLUME 1

Edited by Larry R. Squire

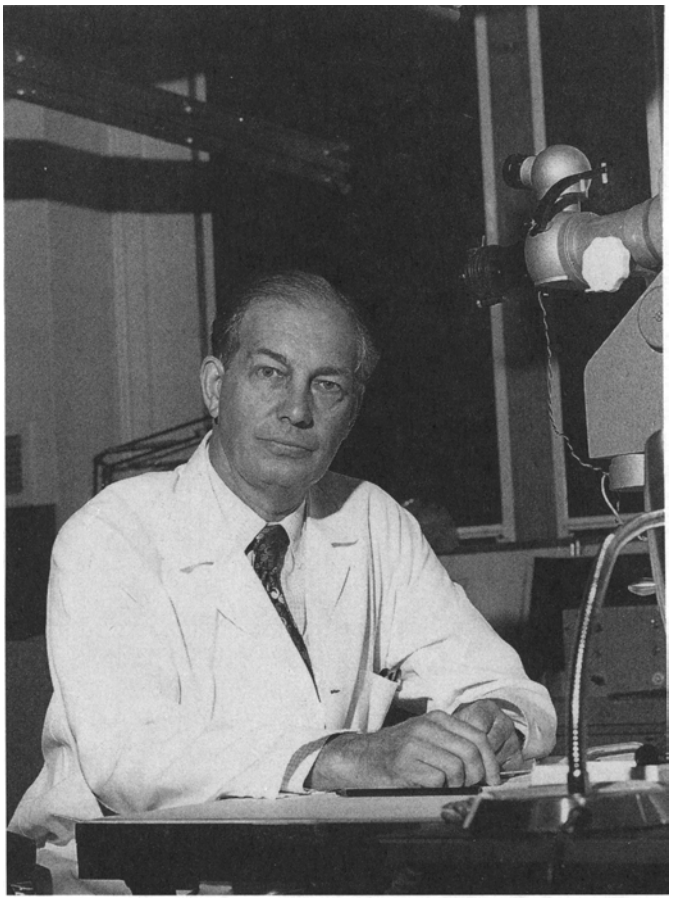
SOCIETY FOR NEUROSCIENCE 1996
Washington, D.C.

Society for Neuroscience
1121 14th Street, NW., Suite 1010
Washington, D.C. 20005

Printed in the United States of America.

Library of Congress Catalog Card Number 96-70950
ISBN 0-916110-51-6

Contents



Peter O. Bishop

BORN:

Tamworth, New South Wales, Australia
June 14, 1917

EDUCATION:

University of Sydney, M.B., B.S., 1940
University of Sydney, D.Sc., 1967

APPOINTMENTS:

Royal Prince Alfred Hospital, Sydney (1941)
University of Sydney (1946)
Australian National University, Canberra (1967)
Professor Emeritus, Australian National University (1983)

HONORS AND AWARDS (SELECTED):

Fellow, Australian Academy of Science (1967)
Fellow, Royal Society of London (1977)
Officer of the Order of Australia (1986)
Australia Prize (Jointly, 1993)

Peter Bishop is best known for his pioneering neurophysiological work on the cat optic nerve, lateral geniculate body, and striate cortex, where he characterized neurons involved in stereopsis. In addition, he developed some of the first mathematical models of the eye itself, which were essential in guiding the neurophysiological work.

Peter O. Bishop

Family History[1]

My forebears, both paternal and maternal, lived in southern England. For a time immediately after World War II, I also lived in England and was able to get in touch with my Bishop relatives and, off and on over the years since then, I have kept up the association. My grandfather, Herbert Orlebar Bishop, was born at Barnstaple in Devon. The name Orlebar, originally Orlingberga, is of Norman origin. I am descended from Richard Orlebar (1736–1803) of Hinwich in Bedfordshire. The Orlebar name came into the Bishop family when Richard Orlebar's granddaughter married into the family in 1812.

In 1870 at the age of 19 my grandfather migrated to Australia, where he was employed as a "line repairer" in the Department of Post and Telegraph in Queensland. Even at that time, Queensland was sparsely populated. Free European settlers had arrived only in the 1840s, and Queensland was the last of the Australian states to become a separate colony. Herbert subsequently became officer-in-charge of various post offices in remote settlements and later in country towns. He remained with the department for the remainder of his working life.

Herbert married Amy Cowan in 1876; my father, Ernest, born in 1877, was the eldest of their six children. With Herbert posted to the settlements of Cunnamulla and Port Douglas, both remote from Brisbane, my father had little, if any, formal education in his early years. At about age 12, he was sent from Port Douglas to the state school at Yeppoon near Rockhampton, 640 miles to the south. At age 14, he was a state scholar and became a boarder at the grammar school in Toowoomba. After leaving school, my father served as an "apprentice," training as a surveyor for entry into the New South Wales Department of Lands. He spent his early years in the department camping in the field, mostly in fairly wild country carrying out surveys for roads and settlements in the northeastern parts of New South Wales. He remained with the department, finally becoming district surveyor for the land district of Armidale from 1924 until his retirement in 1941.

[1]I thank W. Burke and W.R. Levick for checking my draft against their recollections.

My maternal great-grandfather, George Vidal, was born in 1815 in Spanish Town, Jamaica, of English parents. For his schooling, he was sent to Eton in England, and subsequently to Trinity College, Cambridge. He graduated with a B.A. in 1839. He was determined to become an Anglo-Catholic missionary and, with that in mind, migrated to Australia in 1840. Soon after his arrival in Sydney, he was ordained into the Church of England. He made a brief visit to England in 1845, where he married Jane Creak before returning to Sydney. My grandfather, Henry Vidal, was the eighth of my great-grandparents' 10 children. Henry was a public servant in the New South Wales Harbours and Rivers Department. My mother, Mildred, was the fourth of nine children.

I was born at Tamworth, New South Wales, in 1917, the second of my parents' five children. I was seven years old when my father became the district surveyor in Armidale, a town some 360 miles north of Sydney. The family moved to Armidale, and I attended the state primary and high schools there. At age 14, I became a boarder at Barker College, Hornsby, on the outskirts of Sydney. The Depression was then at its height and the school was small, with only 78 pupils. I enjoyed mathematics and physics the most and my original intention was to study engineering at the university. I was not particularly attracted to medicine. As a result of my mother's influence, however, I finally decided to enter the medical school at Sydney University.

Medical School and Hospital, 1935–1942

In the 1930s, the medical school was dominated largely by clinicians in private medical practice, and relatively little research was done. Biochemistry became a separate department only in 1938, and pharmacology in 1949. Lectures in the various disciplines were of an introductory nature, hardly suited to form the basis for a career in research. However, I have never regretted my decision to enter medical school, although I always wished I could have had a better grounding in mathematics.

During the medical course, I was attracted to anatomy, particularly neuroanatomy. In the third year, I dissected a brain. I will never forget the fascination of actually holding a human brain in my hands and realizing that it once belonged to a person like myself with the same sorts of thoughts and feelings as I had. This experience had a tremendous impact on me, and from then on I never questioned that I would try to make a career in brain research.

In the 1920s and 1930s, most of the exciting brain research was done by anatomists rather than physiologists, at least it seemed so to me. I read all I could of the works by people like Arthur Keith, Grafton Elliot Smith, W.E.

Le Gros Clark, and F. Wood Jones. As a result of my third year neuroanatomical dissections and my general reading, I wrote an article, "The Nature of Consciousness," that was published in the Sydney University medical journal. This article brought me to the attention of the professor of anatomy, A.N. Burkitt, and to A.A. Abbie. Dr. Abbie, senior lecturer in anatomy, subsequently published a reply to my article in the medical journal. His paper, also titled "The Nature of Consciousness," was largely a refutation of the ideas I had put forward, but his criticism was kindly. I became friendly with him and, through him, with Burkitt. Abbie subsequently became professor of anatomy in Adelaide, and I saw little of him after my undergraduate days. However, I kept up my friendship with the members of the department of anatomy in Sydney until Burkitt's retirement from the chair in 1955.

When England declared war on Germany in September 1939, many of the resident medical officers in the hospitals immediately joined the armed services. Consequently, the medical course was shortened and my class graduated early, in 1940. In the final year, although we had yet to graduate, we worked in hospitals in place of those who had gone to war. I was in residence in the Royal Prince Alfred Hospital, where I spent a great deal of my final year in the operating theaters giving open ether anesthesia.

By that time, my interest in neurology was fairly well known and, after graduation, I was offered the position of resident medical officer in charge of neurosurgery, a position that would ordinarily have been taken by a senior resident. In neurosurgery I was under Professor (Sir Harold) Dew and Gilbert Phillips, both of whom were to have an important influence on my career. Dew was one of the pioneers of neurosurgery in Australia, and Phillips was a rising star in the field. In 1941, I was made neurological registrar responsible for both neurosurgery and psychiatry.

Another major event took place at this time that was to have a profound effect on my life. I met Hilare Louise Holmes, a member of the nursing staff in the neurosurgical operating theater. We were married in February 1942, just after I was called up for service in the navy.

World War II, 1942–1946

As a surgeon lieutenant, I served at sea in the Atlantic, Indian, and Pacific Oceans, first on the cruiser *Adelaide* and then on the destroyer *Quiberon*. Toward the end of the war, I was stationed at Madang on the north coast of New Guinea. Although the war with Japan ended in August 1945, I was unable to return to Sydney until early 1946. While I was in Madang, I applied for, and was awarded, a fellowship of the Postgraduate Committee in Medicine of the University of Sydney. As a result of my association with Gilbert Phillips, an arrangement was made for me to go to Oxford to work

under Sir Hugh Cairns. During the war, Phillips had joined Cairn's neurosurgical unit in the course of the North African Campaign, so he was well known to Sir Hugh.

By the time Hilare and I sailed for England in July 1946, we had two small children, one about two and one-half years and the other just over a year old. Our ship, the *Stirling Castle*, was still under troopship conditions, with many service personnel returning to England from duty in the Far East. Men and women had separate accommodations. I shared a 14-berth cabin, but fortunately my wife had a separate cabin for herself and the children. We sailed nonstop from Fremantle, in Western Australia, to Southampton, England. With Oxford full of returning servicemen and women, we were unable to find suitable accommodation in the city, and we finally leased an old cottage in Wiltshire on top of the Downs not far from the picturesque village of Ham. On the National Grid, the ordnance survey for England and Wales, the cottage was appropriately called Bishop's Barn! I trained in London during the week and traveled down to Wiltshire at the weekend. Later the family moved to London.

Oxford and London, 1946–1950

In my application to the postgraduate committee at Sydney University, I proposed to study the neuropsychiatric defects in persons who had suffered relatively localized cerebral gunshot wounds. By the time I arrived in Oxford, my original plans had become rather hazy. Cairns certainly had the impression that I had come to train as a neurosurgeon. His idea was that I should spend some time training in clinical neurology at the National Hospital at Queen Square in London before going back to Oxford to resume my neurosurgical career. With this in mind, he arranged for me to be clinical clerk to Sir Charles Symonds. I found the clinical work and intellectual environment of Queen Square tremendously stimulating. In addition to Symonds, people like F.M.R. Walshe and Macdonald Critchley were there, and Gordon Holmes, although retired, was still coming in regularly. At that time, I was still thinking in terms of a clinical career. However, one day I happened into a laboratory in the basement of the hospital, where I met George Dawson working away with electronic equipment. I asked if I could come in and watch the experiment.

Dawson was one of the first people in Britain to build and use electroencephalograph (EEG) amplifiers in a clinical setting and, when I first met him, he was making EEG recordings from patients with myoclonic epilepsy. He was also trying to find out whether it was possible to record potentials from the scalp of normal subjects after electrical stimulation of the ulnar nerve at the wrist or elbow. I soon became the normal subject, with stimulating electrodes at my wrist

and elbow. Dawson is universally recognized as the pioneer of averaging techniques in the recording of biological potentials. However, the potentials he recorded from my scalp were particularly marked, barely needing the photographic averaging by the superimposition of the cathode-ray traces. So, when Dawson (1947) published the first records of evoked potentials to be obtained from a normal subject, the records he used for the illustrations came from my scalp. This experience made me realize that I was better suited to laboratory than clinical research and, about the middle of 1947, I gave up any idea of going back to Oxford to pursue a neurosurgical career.

University College of London, 1947–1950

With my stipend paid from Australia, I approached Professor E.A. Carmichael, director of the research unit at the National Hospital, about the possibility of getting a research appointment at the hospital. Perhaps not surprisingly, he showed little enthusiasm when I told him I was 30 years old and had never done any research. However, he did arrange for me to see C. Lovatt Evans, professor of physiology at University College of London. Lovatt Evans was soon to retire, so he in turn referred me to J.Z. Young, "that young man from Oxford" who just the year before had been appointed to the chair of anatomy at the college. Young took me on immediately and gave me a big empty room on the top floor of the anatomy building in a section that seemed to form part of A.V. Hill's Biophysics Research Unit.

Bernard (later Sir Bernard) Katz had a laboratory just across the corridor from my room. I remember going to see him when I first arrived. He was excited about a little response from a stimulated medullated nerve that he had just observed for the first time, now called the local response that precedes and initiates the nerve action potential. He pointed out to me the little wiggle on the cathode-ray tube trace, but I could neither see nor understand what he was so excited about; with fast single sweeps of a short persistence cathode-ray tube trace, one has to be trained to see such things.

Professor Young suggested that I investigate the claim that changes in the EEG record had been obtained in rabbits as a result of some learning procedures. So I had a research project, an empty room, no research training, and no knowledge of electronics. I was grateful to Professor Young for the generous, but general, support he gave me while I was at University College, but I was never given a research supervisor, so I worked entirely on my own.

It seemed to me that a direct-coupled amplifier was needed to record both resting potentials and the low-frequency EEG waves. Knowing no electronics, I enrolled in a course at the Northampton

Polytechnic and attended classes two or three nights a week for two years. Unfortunately, the course dealt with amplifiers, pulse generators, and related equipment only in the second year. I could not wait for this background theoretical knowledge because I had to start building equipment I needed for the research project. Fortunately I had the occasional, but nevertheless considerable, help from E.J. Harris, a member of the biophysics research unit. In building the equipment, I gained a reasonably good grounding in electronics and the ability to use the tools and equipment in the mechanical workshop. The first seven papers I published all concerned electronics, some of which I wrote in collaboration with Harris.

Beginning Vision Research

Meanwhile, as a result of all the reading I had been doing, I decided not to go on with the original research project. Instead, by using the rather primitive DC amplifier I had assembled, I attempted to determine whether resting potentials were associated with the highly stratified cell layers in the optic tectum of the frog. I quickly realized that the large potentials I recorded had little to do with neural activity but were due mainly to polarization potentials associated with the steel microelectrodes I was using and to the injury potentials caused by tissue damage. However, my recording from the optic tectum in the frog was the beginning of my lifelong association with the visual system.

As work on the frog might seem rather remote to the practical concerns of a hospital in Sydney, I decided to work on the mammalian visual system. My acquaintance with the tectal visual system in the frog prompted me to consider investigating the visual system in the cat. The leading investigators in the field at that time were George Bishop and James O'Leary at Washington University, St. Louis. I read their papers and decided to begin by attempting to repeat their main observations.

Work in neurophysiology at that time centered largely on problems relating to nerve conduction and neuromuscular and synaptic transmission. Little work was done on systems neurophysiology. This situation was true particularly at Washington University where Bishop was associated with J. Erlanger and H.S. Gasser in work for which the latter two received the Nobel Prize in 1944. Erlanger and Gasser used electrical stimulation to produce a compound action potential in a frog's sciatic nerve. They established the classification of the various types of fibers to be found in a peripheral nerve, designated A, B, and C in descending order of conduction velocity and, in the case of myelinated nerve, also in descending order of fiber diameter. It was, therefore, not surprising that Bishop and O'Leary were using electrical stimulation of the optic nerve in the cat to study the differ-

ent groups of fibers in the nerve and tract based on their conduction velocities and were attempting to interpret the field potentials associated with synaptic transmission in the lateral geniculate nucleus and cerebral cortex. As I had already decided to work on the visual system in the cat, it was also not surprising that I came to work on the same problems and use much the same general techniques as those of Bishop and O'Leary.

Use of the cat as the experimental animal required the development of a range of new equipment, most of which was not commercially available at the time. This additional equipment included an electronic stimulator, a suitable slow time base for the oscilloscope, and a camera for recording the cathode-ray tube trace. In addition, I had to design and build a stereotaxic cat head holder and a micromanipulator for directing the recording microelectrode into the lateral geniculate nucleus. Fortunately, the recording of the nerve action potentials did not require a DC amplifier. However, the development of such an amplifier had become an obsession with me, and I continued to work on the DC amplifier design throughout much of my stay at University College. The final design was published in the American journal, *Review of Scientific Instruments*. Despite the effort needed to develop all this equipment, I managed to make a study of the field potentials associated with synaptic transmission in the lateral geniculate nucleus after electrical stimulation of the optic nerve. The paper was published in the *Proceedings of the Royal Society*.

In my last year in England, I became a fellow of the (Australian) National Health and Medical Research Council (NH&MRC). Before that I had been a fellow of the Sydney University Postgraduate Committee in Medicine. The postgraduate committee was extraordinarily supportive over the first three years of my stay in England, always agreeing to my various changes in plan, although the changes were made mostly without reference to the committee in Sydney. I doubt that today such a committee would so readily agree to similar changes in plan when the work was such a radical departure from a career in neurosurgery. Before I returned to Sydney early in 1950, the NH&MRC gave me a grant of £1,000 to buy equipment that enabled me to build up a considerable stock of electronic components that were to stand me in good stead after my return.

Return to Australia, May 1950

One of my main sponsors while I was in England was Professor Dew, professor of surgery at Sydney University. When I returned to Sydney, I joined the department of surgery there. Dew gave me four large rooms that were bare except for tables and chairs so I had, once again, to build all the equipment I needed except for some items that I had brought back from England.

Just the year before, in 1949, the faculty of medicine had introduced a new degree, the Bachelor of Science (Medical) or B.Sc.(Med.). The new

degree allowed selected students, after completing the third or fourth year of the medical course, to spend an extra year working on a research project either with, or supervised by, a senior member of one of the departments. I immediately saw the importance of this innovation, and I determined to give it every support. I called my largely empty rooms in the department of surgery the "Brain Research Unit." Although I was not then a member of the Faculty of Medicine, the faculty approved my application to have students for the new degree work under my supervision. In 1950, my first year back in Sydney, I had four B.Sc.(Med.) students, one of whom, Richard Gye, subsequently became a neurosurgeon and dean of the faculty. As I had only been back for a few months, I had to devise experiments that could be carried out with what little equipment was available. Every year thereafter, until I left Sydney University in 1967, I always had one or more students working with me, not just under my supervision. Without their help, I could not have managed the large administrative load I had when I became head of the department in 1955.

For some years after I came back to Sydney, I continued to use the technique of electrical stimulation of the optic nerve to study the properties of the fiber groups in the nerve and to investigate the field potentials associated with synaptic transmission in the lateral geniculate nucleus. In 1951, my second year back, Jim Lance and Brian Turner, both recent medical graduates, came to work with me as research fellows. Lance later founded the first academic department of neurology in Australia and became the foundation professor of neurology at the University of New South Wales. That year I also had four B.Sc.(Med.) students, David Jeremy, Bill Levick, Jim McLeod, and Annette Walshe, and we accomplished a fair amount of research.

The nerve fibers in the central nervous system had been assumed to have the same general properties as those in the periphery. The optic nerve preparation provided a unique opportunity for determining the properties of a central tract, as developmentally and structurally, the optic nerve must be considered a central tract. We (Bishop et al., 1953) showed that all the fibers in the optic nerve of the cat had the same properties as the group A fibers in the periphery. In a similar study of the pyramidal tract, David Jeremy, Jim Lance, and I showed that all the myelinated fibers in that tract probably also belonged to the A group. Subsequently, Lance made a series of independent studies on the pyramidal tract. By recording field potentials, Jim McLeod and I studied the two main groups of fibers in the optic nerve, as well as the properties of their synaptic potentials in the lateral geniculate nucleus. McLeod was later to become a full professor of medicine in the University of Sydney, as well as Bushell Professor of Neurology.

That same year, Bill Levick and I studied saltatory conduction in the single isolated fiber from the tibial nerve of the cane toad. Levick carried

out all the single fiber dissections and became proficient at isolating single nerve fibers up to 15 mm in length. Some years later, after he completed the medical course and carried out his hospital residency, Levick came back to work with me as a research fellow and, as will be detailed later, he made a singular contribution to my research.

In 1951, I was appointed to my first tenured position as a senior lecturer in the department of physiology. Professor F.S. Cotton, the professor of physiology, treated me generously. He allowed me to retain my laboratories in the department of surgery, and the few formal teaching commitments I had did not interrupt my research activities to any great extent. However, Cotton retired at the end of 1954, and I was appointed to succeed him as professor and head of the department.

University in Crisis

Beginning in 1955, my life was to undergo a radical change. In the early 1950s, Sydney was the only university in New South Wales and, consequently, it had the only medical school. The department of physiology, and the university in general, were in poor shape because of years of financial neglect and the large influx of students in the years immediately after World War II. I had had a sound training in neuroanatomy and neurophysiology, but I knew relatively little about the other bodily systems. The department at that time was responsible for 14 different courses in physiology, including those in the faculties of dentistry, medicine, science, and veterinary science. In addition to these standard undergraduate courses, the department had a separate series of lectures for each of the postgraduate medical diplomas, such as those for gynecology and obstetrics and dermatology; it also had courses for the various allied medical personnel, including occupational therapy, physiotherapy, speech therapy, and so on.

Apart from me, there were only four full-time members on the academic staff of the department, two of whom resigned during my first year. That left only a senior lecturer (William Lawrence) and a teaching fellow (Arthur Everitt). Lawrence had had considerable experience with the physiology practical classes and, while he organized these classes, I took the responsibility for organizing the various courses of lectures. It was possible to maintain reasonable academic standards only by having a large number of part-time lecturers, most of whom were fairly recent medical graduates in the early stages of developing a practice. Inevitably, I had to do a great deal of the lecturing myself, mostly on systems other than the nervous system. With such a heavy administrative and teaching load, I was able to devote much less time to my research activities. Even so, I still was able to supervise B.Sc.(Med.) students but at a much reduced level of involvement.

In 1956, I induced Paul Korner and William (Liam) Burke to accept appointments to senior lectureships, and subsequently both became full professors. In the following years, many more staff appointments eased my load considerably. But the overall teaching load increased even faster than the staff increased. In my first year as head of the department, the medical course had little more than 200 second-year students. In every year from then on nearly 100 more were added; by 1961 there were 620 students in the second year. Numbers increased in the other faculties as well, but less dramatically than in medicine. Nevertheless, we finally had a total of about 1,500 students taking physiology in the various faculties and courses.

From the start, I pressed for the introduction of a quota system to limit student numbers, particularly in the faculty of medicine, but for some years the university offered little support. Two main events finally led to the introduction of a quota system in the faculty of medicine, the first such quota in the university. The Federal Government set up a Committee of Inquiry into Tertiary Education in Australia. Among the far-reaching recommendations of the committee was one relating to the problem of student numbers. In particular, the committee recommended the establishment of a second medical school in New South Wales. Then, in 1963, after the second medical school at Kensington in Sydney was established, it finally was possible for Sydney University to have a limit of 300 entrants to its medical school. Since then, the quota has been set at 240. The above account provides a background against which to set my research activities during my early years as head of the department.

Research Activities, 1955–1967

Aside from the above diversion we can now return to the account of my research activities. In 1954, Ross Davis, then a medical student, and I used electrical stimulation of the optic nerve and field potential recordings to study the recovery of responsiveness and other aspects of synaptic transmission in the lateral geniculate nucleus. Then, in 1958, after medical graduation and a year in a hospital as an intern, Davis returned to work with me as a research fellow. In the mid-1950s, we had made many attempts to obtain intracellular records but, using the techniques available to us at that time, the recordings we achieved were always too brief to be of practical use. Unlike the large motoneurons in the spinal cord, the relatively small geniculate cells could not withstand the injury caused by the insertion of the microelectrode. Nevertheless, we were able to make good extracellular records from single units even over quite long recording times.

While still using electrical stimulation of the optic nerve, but now recording from single units extracellularly, we again studied the synaptic events in the lateral geniculate nucleus (Bishop et al., 1962). In a series of

three papers, Burke, Davis, and I provided a detailed description of the various waveforms of the responses of single optic tract and radiation axons and of the responses from geniculate cell bodies when they are activated either orthodromically via the optic tract or antidromically via the optic radiation. The responses from the cell bodies could be fractionated into three components, namely the slow S-potential, considered to be the extracellularly recorded excitatory postsynaptic potential (EPSP) evoked by the retinal afferents; the A-potential, apparently derived from the initial segment of the geniculate cell body-axon region; and the B-potential, believed to represent the invasion of the soma-dendritic membrane. Many geniculate synapses have a high safety factor. At times, a single retinal afferent axon can be found that leads to a single all-or-none S-potential which could, in turn, occasionally be sufficient to discharge the cell. These papers are still relevant today, and they are regularly cited in the literature. Subsequently, the concept of a transfer ratio (proportion of afferent S-potentials that generate geniculate action potentials) has been used as a way to study the efficacy of signal transmission through the lateral geniculate nucleus.

In 1958, I was invited to attend a symposium in Paris in honor of Henri Piéron. The trip gave me the opportunity to visit vision laboratories in Denmark, Sweden, the United Kingdom, and the United States. While in Baltimore, Maryland, I visited Steve Kuffler in the Wilmer Institute at The Johns Hopkins University, and I had the opportunity to watch an experiment by David Hubel and Torsten Wiesel. At that time they were at the start of their career together and were recording from single units in the cat cerebral cortex. They were using the multibeam ophthalmoscope that S.A. Talbot and S.W. Kuffler had designed and built some years before in 1952. At that earlier time, the instrument represented an important technical advance because small flashing lights could be focused on the retina under direct viewing with the eye intact and, except for the introduction of the microelectrode, its optics preserved. Watching their experiments had a profound effect on me and, when I returned to Sydney, Hubel and Wiesel soon appreciated the marked constraints that the multibeam ophthalmoscope imposed. Instead, for stimuli, they turned to the use of small targets moved by hand over the surface of a tangent screen placed in front of the cat. On my return to Sydney, I immediately set to work to design and build a cat multibeam ophthalmoscope. The instrument was finally assembled, but it was used only for the one set of experiments that Tetsuro Ogawa, Levick, and I did. By that time, we had recognized the same experimental constraints that Hubel and Wiesel had appreciated a year or so before.

The department of surgery was located in a building some distance from the department of physiology, and by the late 1950s I had completed the move from one building to the other, giving me two new fully equipped laboratories and associated facilities. The experience with the multibeam

ophthalmoscope had been a powerful influence in directing my research toward the use of more natural stimuli and intact visual optics, and the new laboratories, fitted with tangent screens, had already been designed with this new approach in mind. Furthermore we had, by then, gained considerable experience in the use of extracellular single unit recording in the lateral geniculate nucleus and later in the visual cortex.

In retrospect, 1959 can be seen as a watershed year in the history of visual neurophysiology, as most of our knowledge of the visual system dates from that time. That was the year Hubel and Wiesel (1959) published their first report on the receptive fields of simple cells in the visual cortex. They found the stimulus features important for striate neurons to be straight lines, bars, and edges, having an orientation and, usually, a direction of movement that were characteristic and critical for the discharge of the cell. In the same year, Lettvin et al. (1959) published a paper with the title "What the frog's eye tells the frog's brain." The title of the paper and the speculations it contained undoubtedly caught the imagination of the time. The authors proposed to present the frog with as wide a range of visible stimuli as they could, including things it would be disposed to eat, things from which it would flee, sundry geometrical figures, stationary and moving about, and so on.

In many ways, the years 1959 to 1967 were the most exciting and fruitful of my career. Liam Burke had worked with me for some time before that, and now I had a further succession of able collaborators, each of whom was to bring to bear their own experience and expertise. In addition to Ross Davis and Bill Levick, there were George Vakkur, the Sydney medical graduate; Tetsuro Ogawa and Tosaku Nikara from Japan; Bob Rodieck from the Massachusetts Institute of Technology (MIT) in Cambridge, Massachusetts; and Wlod Kozak from Warsaw, via the Eccles' laboratories in Canberra. In addition to their collaboration with me, many of these researchers also had other independent projects.

Visual Optics and Neuro-ophthalmology

There was a further factor that drove the direction of my research toward a consideration of visual optics and neuro-ophthalmology. We had begun a study to determine the projection of the visual field onto the lateral geniculate nucleus. It became clear to us that, for this project, we would need a detailed knowledge of the cat's optics. A thorough search of the literature failed to reveal a sufficiently detailed account of the visual optics of the cat or, indeed, of any other animal. So we (Vakkur and Bishop, 1963) began the preparation of a cat schematic eye. Whereas our main concern with the schematic eye was the practical need to provide a quantitative framework for neurophysiologic studies, the project appears to have been the first example where the information derived from a schematic eye was

used in an explicitly comparative manner to shed light on the possible adaptive significance of ocular structures (Martin, 1983). Thus, in effect, we pioneered the new field of comparative neuro-ophthalmology.

A schematic eye is a self-consistent mathematical model of the optical system of the average eye. We arrived at a final schematic eye model by two independent methods. Vakkur, Kozak, and I made an initial examination of the eye as a whole that provided a measure of the posterior nodal distance and the out-of-focus distance. Then, assuming the refractive index of the vitreous humor, the values as measured above fixed the positions of the posterior three cardinal points (principal, nodal, and focal) of the optical system with respect to the receptor layer of the retina. Established in this way, the cardinal points do not require information about the cornea and lens. The second method, independent of the first, is the reverse of the above procedure (Vakkur and Bishop, 1963). The development of the cornea-lens optical system fixes the position of the cardinal points with respect to the plane of the anterior corneal surface. Then, by measuring the overall length of the eyeball and estimating the combined thickness of the sclera and choroid, these cardinal points can also be referred to the receoptor layer of the retina as was done by the first method. The two sets of data showed a remarkable level of agreement.

Although the paraxial lens equation (Gauss) used to develop the schematic eye treats only rays close to the optic axis, the observations and measurements that we made were far more extensive and useful than those provided by the paraxial system. The additional information included a complete metrological treatment of the globe and its components, together with their average values, the positions and sizes of the entrance and exit pupils, and the extent of the monocular and binocular visual fields. For our later studies, particularly in relation to binocular vision, it was important to establish the accuracy with which the center of the area centralis and the visual axis could be determined, as well as the relationship of the visual axis with respect to both the positions of the optic disk and the blind spot. A further important experimental consideration concerns the positions the eyes assume when the anesthetized animal is completely paralyzed—the so-called position of paralysis. Our schematic eye studies are now regularly cited in the literature, and the data they contain continue to be used widely.

The study that Kozak, Levick, Vakkur, and I did on the projection of the visual field onto the lateral geniculate nucleus was the first attempt to establish in any animal the details of the projection by electrophysiologic methods. Of the possible systems of coordinates for defining directions in the visual field, we finally decided on a particular system of spherical coordinates. Using single unit recording, the visual direction of the center of a receptive field of a neuron was expressed in terms of two angles, azimuth and elevation, of the coordinate system, the polar axis of which passed through the nodal point of the eye at right angles to the fix-

ation plane. This coordinate system is now universally used to specify the visual field locations of the receptive fields of cells in the central nervous system. An important concept to arise from this study was the projection line. This concept refers to a column of cells, the receptive fields of which all have a common visual direction in the visual field so that each column can be regarded as representing a particular direction. In the cat lateral geniculate nucleus, a projection line is approximately confined to a parasagittal plane and passes downward and backward through all the separate cellular layers of the nucleus.

Binocular Vision and Stereopsis

In 1964, Jack Pettigrew, then a B.Sc.(Med.) student, came to work with Tetsuro Nikara and me on the problem of binocular interaction on single cells in the cat's striate cortex. As will be described later, this study led to the discovery that most of the striate cells were stimulus-disparity-selective. The experimental techniques and observations that were made over the previous few years provided the essential ingredients that led to this discovery. By then, Levick had been able to modify a commercial RIDL 256-Channel Analyzer for the computation of poststimulus time histograms, which were later to prove essential for our quantitative assessment of the level of binocular facilitation. The binocular project also involved further essential innovations (Bishop and Pettigrew, 1986). The development of a more effective intravenously administered drug mixture, as well as other associated techniques, made it possible to reduce the residual eye movements in the paralyzed cat preparation to an acceptably low level. Further, the use of a specially adapted Risley counter-rotating prism assembly enabled the positions of the two receptive fields of a striate cell to be moved in small steps over the surface of the tangent screen.

In early November 1965, I attended the Caltech symposium on "Information Processing in Sight Sensory Systems," where I met Horace Barlow. Just before the symposium, Pettigrew had, as part of his thesis for the B.Sc.(Med.) degree, included our work on the disparity-selectivity of striate cells, and I took the thesis with me to the meeting. When I showed it to Barlow, he found that the work was similar to the project he had planned for Colin Blakemore's Ph.D. thesis. Soon afterwards, Barlow invited Pettigrew to visit Berkeley and spend some time in mid-1966 working with Blakemore and himself. By then, I had begun working with another B.Sc.(Med.) student, Doug Joshua, along the same general lines. With the continuing collaboration between the two departments in Sydney and Berkeley, progress was rapid. We already knew that each of the two receptive fields of a cortical cell has the same highly specific stimulus requirements, and Barlow made an important contribution by suggesting that the cortical cells could be acting as feature detectors with a high probability of

responding to a particular feature in the two retinal images that corresponded to one and the same object feature in the external world.

At this stage it will be helpful to give a brief account of our work on binocular depth discrimination, or stereopsis, an activity for which the two eyes are essential. Because the two eyes are horizontally separated in the head, each eye sees a given object feature from slightly different vantage points, leading to a small horizontal difference in the relative positions of their images on the retinas of the two eyes. The images of the fixation point, by occupying the same relative positions on the two retinas, are by that token exactly corresponding. The plane through the fixation point that is orthogonal to the visual axis constitutes a reference surface for expressing the relative positions of image points on the two retinas. Of the image points that are noncorresponding, some are closer to the reference plane than their companion image in the other eye. The various object points therefore have a range of different retinal image locations or disparities and so are detected by the nervous system as representing varying intervals in depth to one or the other side of the reference plane. The neural theory of binocular depth discrimination requires that binocular cells in the striate cortex have at least two properties. First, because of the differing directions or positions of its two receptive fields, each binocular neuron should respond selectively to the position disparity that corresponds to the particular depth interval at which the two receptive fields are in spatial register. In addition, each cell should be capable of a fine discrimination of that stimulus disparity within its narrow responsive range and should be either inhibited or ineffective outside this range. Second, a population of such cells should show a range of different receptive field position disparities, so that a range of different horizontal stimulus disparities can be detected. It therefore was natural that we should give particular attention to these properties.

The neural theory of binocular depth discrimination as outlined above is now widely accepted. The theory is based on the concept that the two receptive fields of a binocular cell are to be regarded as feature detectors and as such must have an identical structure and spatial organization. It is, however, still undecided just how object features are represented in the brain, and it is possible that they are actually represented in terms of their spatial Fourier components. On this basis, DeAngelis et al. (1995) proposed that horizontal disparities are encoded by binocular cells not in terms of the position disparities of their left and right receptive fields but rather in terms of the differences in the shapes (or phases) of their receptive fields.

By early 1967, the Berkeley group had been able to complete its analysis of the disparity data and to present them for publication later that year (Barlow et al., 1967). At that time, I was working with two other B.Sc.(Med.) students, Warren Kinston and Matthew Vadas, on a somewhat unrelated problem concerned with the nuclei medial to the lateral

geniculate nucleus. Then a further complicating event arose. In January 1967, I was invited to accept the chair of physiology in the John Curtin School of Medical Research in succession to Sir John Eccles who had, the previous year, resigned to go to the United States. However, I was not able to make the move to Canberra until June. We had a problem, therefore, in getting our disparity data published. By working with B.Sc.(Med.) students, much of the final analysis of the data and the task of writing the paper for publication were my responsibility. Hence our papers on binocular interaction were not published until 1968, and one even later (Pettigrew et al., 1968; Joshua and Bishop, 1970).

The Australian National University, 1967–1984

I was sad to leave Sydney University and to sever my association with B.Sc.(Med.) students.[2] I always felt that, with the means available, the university had treated me generously.

Within the Australian National University, the John Curtin School is one of the schools that forms the Institute of Advanced Studies. The institute is a center for research and postgraduate training without involvement in undergraduate teaching. The emphasis on research, coupled with the departmental structure that existed in the John Curtin School at the time of my appointment, provided the head of a department with the ability to redirect the department's research effort. An essential element of the redirection process was the school's policy of keeping the number of tenured members of the academic staff to 50 percent or less. The intention always was that about half of the research personnel in the institute would be visitors coming from elsewhere in Australia or from abroad and staying for three to five years. On this basis, the necessary research fellowships were provided and, subject to the departmental budget, the head of the department made the recommendations for the award of fellowships. As a result, the systems neurophysiology of vision became the dominant interest of the department.

The Ph.D. degree was first introduced in Australia about 1949, and the Australian National University originally was established in Canberra to provide the necessary graduate research training. However, at the time, most graduates from Australian universities preferred to continue their training either at their home university or abroad. As a result of that preference, coupled with the specialized nature of the work we were doing in what was then a fairly new field, the visitors we attracted tended to be mostly postdoctoral scientists from abroad.

[2] Alphabetically, they were: D.S. Bell, R. Davis, W.A. Evans, G.B. Field, D.C. Glenn, C.S. Grace, J.G. Grudzinskas, R.S. Gye, B.L. Hennessy, D. Jeremy, D.E. Joshua, B.R. Kelly, W.J. Kinston, J.G. McLeod, W.R. Levick, J.D. Pettigrew, J. Scougall, J.R. Smith, J. Stone, M.A. Vadas, and A.M. Walshe.

In the late 1940s, when the Australian National University was founded, Canberra was a small and isolated community. As a result, the John Curtin School had to be largely self-sufficient, having readily available its own full range of workshop facilities, including fitting and turning, instrument making, joinery, and so on. When I arrived in Canberra, these workshop facilities were still largely intact. In addition, the head technical officer of the department, Lionel Davies, who had remained after Eccles' departure for the United States, had considerable expertise as an instrument maker. Furthermore, Robert Tupper, who had come with me from the department in Sydney, now was responsible for the development and maintenance of the electronic equipment. I had, therefore, an unparalleled opportunity to design and construct laboratories suitable for the systems neurophysiology of vision that we were now contemplating. Before too long, three of what were eventually seven fully equipped research laboratories were ready for occupation.

Early in 1968, G.H. Henry joined me in Canberra after spending the previous year working abroad as a Churchill fellow. In collaboration with various colleagues, Henry and I worked together for the next seven years. Our colleagues included J.C. Coombs, I. Darian-Smith, and K.J. Sanderson, all from Australia, and C.J. Smith (New York), A.W. Goodwin (South Africa), and B. Dreher (Poland). Toward the end of 1967, Bill Levick came to the department from the University of California, Berkeley, and soon afterwards Brian Cleland joined him from Northwestern University, Chicago. With separate laboratory facilities, Levick and Cleland were able to work independently of Henry and me. As additional laboratories were fitted out, two relatively long-term appointments were made, first Jon Stone and somewhat later, Austin Hughes. Again, with separate laboratory facilities, they were each able to work independently although mostly in collaboration with colleagues from abroad.

First Experiments in Canberra

The first experiment we did in Canberra (Henry et al., 1969) was to study the binocular interaction on those simple cells in the striate cortex that were considered to be exclusively monocular. Up to that time, binocular influences of an inhibitory nature had been largely neglected, particularly in relation to cells considered exclusively monocular. This neglect was not surprising because inhibition can be observed only in the presence of some form of excitatory activity.

Simple neurons usually have a low or absent maintained discharge. However, such a discharge can be produced by controlled stimulation of the dominant eye using the activated-discharge technique. To do this, the dominant eye was stimulated by small amplitude oscillations of an optimally oriented light bar moving continuously to and fro in the optimal direction

over the excitatory region of the receptive field. At the same time as this background discharge was produced, the suspected position of the nondominant eye receptive field was tested by a stimulus considered to be optimal for the dominant eye. Though approximately optimal in each case, the conditioning and testing stimuli were driven at different and asynchronous frequencies by separate and independent function generators. As the spikes were collected in phase with the testing stimulus while those due to the activated discharge were collected randomly, the analyzer bins were filled relatively uniformly when the nondominant eye was occluded. We found that, despite being ostensibly monocular, all the cells showed clear binocular effects. A predominantly inhibitory receptive field for the nondominant eye could usually be found in the contralateral hemifield at a position approximately corresponding to the receptive field for the dominant eye.

The above technique revealed the receptive field for the nondominant eye to be mainly suppressive. However, a small region of subliminal excitation was commonly found within the subliminal receptive field. This excitatory region was located in the contralateral hemifield in close correspondence to the excitatory region in the receptive field of the dominant eye, and it had approximately the same relatively small size as the latter region. Particularly striking was the steep transition from strong inhibition at one position to a peak of facilitation at another all in the space of a few minutes of arc. The peak of binocular facilitation provided by the nondominant eye, together with the surrounding inhibition, is clearly important for the discrimination of retinal image position disparities.

The experiments described above were important also because they provided a test for two further methods of examining the nature of binocular interaction. One was the prism displacement procedure that we had already used in Sydney, in which the two receptive fields were stimulated as the receptive field of one eye was moved stepwise into and out of exact correspondence by prisms placed in front of the dominant eye. The other or phase shift method is, in effect, the equivalent to the prism displacement procedure. However, this time the prisms are used to separate the two receptive fields widely on the rear projection screen so they can be stimulated, separately but optimally in each case, by light bars moving over their respective receptive fields. With the two stimulus sweeps at first in synchrony, advancing or retarding the stimulus sweep for one eye is then equivalent to the prism displacement procedure. Considerable precision is possible with this second method because the start of the stimulus sweep can be controlled in small steps. All three methods gave identical results, each demonstrating the same excitatory and inhibitory effects on the part of the nondominant eye. The activated-discharge technique is a relatively fast procedure and has the important advantage that it produces a continuous profile of the response across the receptive field.

According to the general belief at the time of these experiments, there is little, if any, binocular interaction in the lateral geniculate nucleus. This premise gave us an early opportunity to test for binocular interaction in the nucleus using the activated-discharge technique (Sanderson et al., 1971). Contrary to general belief, we found that the great majority of the cells in all the laminae were, in fact, binocularly activated and that, of these cells, the great majority of the receptive fields for the nondominant eye was purely inhibitory. Of the few nondominant cells that had receptive fields that were excitatory, the effect was so weak that, with only one or two exceptions, it could not be appreciated by hand plotting. The location of the nondominant eye receptive field was always in approximate correspondence with the receptive field for the dominant eye.

Most of the experiments that Henry and I did over the ensuing years were concerned with the receptive field properties of the various types of cell in the striate cortex, although we gave special attention to the property of selectivity in relation to orientation and the direction of movement. One early observation that Henry, Dreher, and I made concerned the hypercomplex property of end-inhibition. End-inhibition refers to the observation that the excitatory response from a cell can be reduced if the length of the stimulating bar is extended beyond some optimal value. It was a property thought only to be found in cells at a relatively high level in a simple, complex, and hypercomplex hierarchical sequence. Our finding was that the property was not a later acquisition by complex cells but a general property of all the various cell types in the striate cortex. Some time later, Orban, Kato, and I made a particularly detailed study of the inhibitory properties of the end-zone region.

After United States President Lyndon Johnson visited Australia in 1966, the respective governments set up the United States–Australia bilateral agreement for scientific and technical cooperation. By that time, the publications on vision from the John Curtin School had attracted fairly wide general interest, particularly in the United States, and Peter Gouras wrote to me from the National Eye Institute in Bethesda, Md., about the possibility of organizing a symposium on vision under the terms of this agreement. He suggested that the meeting be held in Canberra. I responded enthusiastically to his proposal, with the result that the National Eye Institute and the John Curtin School jointly organized a week-long symposium. It was held in Canberra February 7–11, 1972, with Gouras responsible for arrangements in the United States and me in Australia. Among the leading visual scientists invited to attend, some 22 came from the United States. The major emphasis was on the neurophysiology of visual mechanisms using single unit recording at the various levels of the visual pathway. The proceedings of the symposium, including selected parts of the discussions that followed each presentation, were published in two dedicated issues for May and June 1972 of the journal *Investigative*

Ophthalmology (now *Investigative Ophthalmology and Visual Science*) just three months after the meeting.

Binocular Interactions in Relation to Stereoscopic Vision

Among the studies we did in Canberra, I will comment on only a few that seem in hindsight to be of general interest, particularly the role of binocular interactions in relation to stereoscopic vision.

In 1975, Henry spent a sabbatical year at the University of Washington, Seattle, working with Ray and Jenny Lund who were subsequently to make a year-long return visit to work with Henry in Canberra. On his return from Seattle, Henry began working independently of me with his own laboratory facilities. From 1976 onward, all my collaborators except Stjepan Marčelja came from abroad, but a complete list must include those I have already mentioned.[3]

In recent years, two different approaches have developed toward an understanding of the operation of the visual cortex. The usual approach, my own included, has largely concerned the role of simple cells as feature detectors, with attention on the spatial organization of their receptive fields, and with lines and edges regarded as the elementary features extracted by the cells. The alternative approach is based on the application of spatial frequency (Fourier) methods and, by concentrating attention on the sensitivity to sinusoidal gratings of varying spatial frequencies, this approach has tended to neglect the discrimination of spatial position. Not until Janusz Kulikowski came to work with me, did I give serious consideration to the application of spatial frequency methods.

Gabor's analysis (1946) of auditory communication applies equally well to the communication of visual signals, and Marčelja (1980) was the first to appreciate the relevance of Gabor's ideas to the coding of visual signals in the nervous system. With respect to auditory communication, Gabor pointed out that if one wishes to encode a communication signal compactly into a succession of elementary signals or samples spaced in time, one has to accept a compromise between the "spread" of each of the samples, both in the time domain and in the frequency domain. The nature of this compromise can be appreciated by considering the note of a tuning fork. To be sure of the frequency of the note, one has to listen to many cycles of the vibration; but the longer one takes to make a decision about the frequency of the vibration, the more indeterminate becomes the precise time at which one can say the note occurred. Similarly, precision regarding the time of occurrence of the note can be achieved only at the expense of the lack of precision regarding the frequency of the note.

[3] Alphabetically, they were: R.M. Camarda (Italy), A. Harvey (England), H. Kato (Japan), J.J. Kulikowski (England), R. Maske (South Africa), J.I. Nelson (USA), G. Orban (Belgium), E. Peterhans (Switzerland), and S. Yamane (Japan).

Spatial frequency considerations along the lines of a Gabor representation provide an explanation for the shape and organization of the receptive fields of simple cells in the striate cortex in that they contain a varying number of narrow elongated subregions arranged in a side-by-side fashion with subregions that respond at light ON alternating with those that respond at light OFF. Furthermore, a response to high spatial frequencies is needed to discriminate thin lines and sharp edges, and the same compromise exists between the discrimination of these features and their precise location in space. Whereas a detailed exposition of the concepts would be out of place here, a brief outline of certain aspects is needed to place our observations in context.

At the outset, it was clear that our rear-projection methods had to be replaced by stimuli generated on the face of an oscilloscope so we could obtain stimuli that were either lighter or darker than the background, but each equal in contrast. For this series of experiments, only monocular stimulation was used, and our observations were largely confined to the responses of simple cells in the striate cortex. As a basis for the application of Fourier analysis, we carried out the following experimental procedures on a series of simple cells (Kulikowski and Bishop, 1981). As the application of Fourier methods requires that spatial summation over the receptive field be linear, we first confirmed earlier reports concerning the essential linearity of simple cells. Next, we recorded each cell's spatial response profile (receptive field) to narrow stationary and moving bars that were both brighter and darker than the background and we examined the relationship between these responses and those to moving light and dark edges. Then, using the same series of cells, we recorded their responses to stationary and drifting sinusoidal gratings. Finally, on the assumption that simple cells operate linearly, we compared the spatial response profiles recorded experimentally with those predicted by inverse Fourier transformation of the spatial frequency tuning curves. Conversely, the spatial frequency tuning curves recorded experimentally were compared with those predicted from the response profiles to stationary and moving stimuli.

Theoretical considerations indicate that, for any given spatial frequency tuning curve (bandwidth) and optimal spatial frequency, the inverse Fourier transform should predict the spatial response profile (receptive field) modeled as a Gaussian function, as well as the spatial period of the subregions within the Gaussian envelope (number and dimensions of the subregions). The spatial period (combined width of two subregions) is inversely proportional to the optimal spatial frequency. In general, it can be said that the narrower the bandwidth, the greater the number of subregions needed to achieve the required selectivity; and the higher the optimal spatial frequency the narrower the width of the individual subregions in the receptive field.

Our experimental observations indicate that the overall width of the response profiles obtained from a series of simple cells as well as the num-

ber and widths of the individual response peaks in the profiles are all in reasonably good agreement with those to be expected on the basis of Fourier transforms of their respective spatial frequency tuning curves and optimal spatial frequency. The simple cell with the narrowest bandwidth that we have observed (0.94 octave) has an optimal spatial frequency of 2.0 cycles/degree. To achieve such a relatively high degree of spatial frequency selectivity on the basis of a Gabor representation, this cell would require a receptive field profile with an overall width of about 1.2 degrees having 5 response peaks with amplitudes all above the 10 percent level and having a width of about 0.25 degrees for each peak. These values agree reasonably well with those for the profile that we obtained experimentally from this cell in response to moving light and dark bars.

The cell with the highest optimal spatial frequency that we have observed (2.3 cycles/degree) should be adequate to account for the cut-off spatial frequency of 9 cycles/degree determined experimentally for the cat. The same reasonably good level of agreement is found for cells at the other end of the scale, namely those with the broadest spatial frequency tuning curves and the lowest optimal spatial frequencies. However, the Gabor representation suggests that the most common receptive field types are those with three or four subregions, whereas we found that receptive fields with two subregions are much more common than those with three or four. It is possible that the high threshold for discharge in simple cells conceals subregions with a relatively low sensitivity.

Some years later, we again considered the role of simple cells as feature detectors in a local stereoscopic mechanism (Maske et al., 1984). To assign a depth value to a particular feature, the two receptive fields of a binocularly activated cell must respond to one and the same object feature. This can be done only if the organizations of the two receptive fields are identical, or nearly so. In our experiments, we selected a series of simple cells that had monocular responses from each eye of sufficient amplitude to be able to examine each of their receptive field organizations in quantitative detail. By that time, we had developed a rear-projection system that was able to provide stimuli that were both lighter and darker than the background. Using Risley counter-rotating prism assemblies, the two receptive fields were widely separated on the projection screen so that the receptive field for one eye could be stimulated independently of the receptive field for the other eye.

The two receptive fields of a given cell were remarkably similar with respect to a range of different attributes. The number and spatial sequence of the subregions in response to the movement of light and dark bars were always the same, as were the interpeak separations. The direction selectivity for any given cell was nearly always the same, independent of stimulus contrast. Estimates of the horizontal and vertical position disparities of the response peaks provided a particularly stringent test for the degree of sim-

ilarity. Some significant differences, however, exist between the two receptive fields, namely, with respect to the overall ocular dominance and position disparity, preferred stimulus orientation and, rarely in Area 17, direction selectivity. Except for ocular dominance—the functional role of which remains a mystery—the remaining attribute differences have key roles in binocular vision.

As a disparity-encoding process for a given cell, the main feature used to determine the phase difference between the receptive fields for the two eyes is the overall shapes of the response peaks to light and dark bars (DeAngelis et al., 1995). Our rear projection methods made it difficult to achieve an exact balance between the contrasts of the light and dark bars, so there would have been some distortion in the overall shapes of the response peaks to the two kinds of bar. Hence, from our observations, we would have been unable to arrive at any conclusion regarding the role of phase differences in a disparity-encoding scheme. However, it should be noted that, even on a monocular basis, there can be different phase-sensitive responses to different stimuli with a 90 degree phase difference between the response to a bar and response to an edge (Kulikowski and Bishop, 1981).

In a paper on the ability of striate cells to discriminate orientation and position disparities (Nelson et al., 1977), we concluded that the binocular response is very sensitive to position disparity but relatively insensitive to fairly large orientation disparity changes. A quantitative analysis showed that simple striate cells are probably able to discriminate position disparities known from behavioral testing to be near the limit for the cat. When the two receptive fields of a simple cell are in spatial register (zero position disparity) the amplitude of the binocularly facilitated response to an optimal stimulus can be as much as two or three times the sum of the two separate monocular responses to the same stimulus. However, this binocular response can be considerably reduced by a position disparity as small as a 10-minute arc.

Retirement and General Activities

By the end of 1982, I reached the statutory retiring age of 65, and I had to give up my laboratory in the John Curtin School. For two years after my retirement, at the invitation of Richard Mark, I worked as a visiting fellow in the Australian National Univeristy's Research School of Biological Sciences and was able to get most of the backlog of our research material ready for publication. During this period, my wife and I spent some time in Dunedin, New Zealand. There, at the invitation of John Parr, I worked in the department of ophthalmology of the Otago Medical School. Then, for most of 1985 and 1986, my wife and I lived in Europe, where we had the pleasure of visiting colleagues who had worked with me in Canberra. I was able to take part in the work that Guy Orban and his colleagues were doing

in his laboratory in the medical school of Katholieke Universiteit, Leuven, Belgium. Then my wife and I moved to Zurich. There, in the department of neurology of the University Hospital Zurich, Esther Peterhans was doing experiments on the awake performing monkey. My involvement in these experiments enabled me to gain a much better appreciation of the considerable possibilities offered by experiments of this kind. Finally, my wife and I spent most of 1986 with Fergus Campbell in Cambridge, England, where I was the overseas visiting fellow at St. John's College. We much enjoyed living in an attached cottage in the grounds of the College and walking daily to town across the Bridge of Sighs.

In much earlier times, before my retirement, my wife and I had lived abroad for extended periods. In addition to the years in England immediately after the war, we spent 1963 in Cambridge, Massachusetts, where I worked in Pat Wall's biology department at MIT. The experiment I did with Arthur Taub at MIT was the first and only occasion that I deserted the visual system to work on the spinal cord. At the invitation of the Japan Society for the Promotion of Science, my wife and I twice visited Japan. In 1974, as a guest of Kitsuya Iwama, I joined the work in progress in the department of neurophysiology of the Osaka University Medical School. Then, some years later in 1982, at the invitation of Motohiko Murakami, my wife and I lived in Shinjuku in Tokyo to be handy to the department of physiology of the Keio University School of Medicine.

On our return from England at the end of 1986, we moved from Canberra to live at Avoca Beach, a small coastal resort halfway between Sydney and Newcastle. Soon after the move to Avoca, Jonathan Stone, now Challis Professor of Anatomy at Sydney University, kindly invited me to accept a research associateship in the department, and since then I have made regular visits to the university, mainly to work in the library.

I have become interested in the role of vertical disparities in the binocular process, particularly in relation to the size and depth constancies. Our earlier experiments in Sydney had shown that the receptive field position disparities are distributed as much in the vertical as in the horizontal direction and that many cortical cells are specifically sensitive to vertical retinal image disparities. Soon after these observations were first reported, they were subjected to criticism on the grounds that only horizontal retinal image disparities contribute to stereoscopic depth perception.

Recently, I have published papers making a strong case for the essential role that vertical disparities play in relation to both the size and depth constancies (Bishop, 1994). These papers have led me to conclude that random-dot stereograms, being confined to one plane and so without any real depth intervals, cannot serve as a model for the perception of depth in relation to real three-dimensional objects. These observations are of the nature of thought experiments, as I have had to rely to a large extent on the experimental results of others. Many of my conclusions are counter to

long-held beliefs, so it is not surprising that journal referees should subject them to searching criticism. This is as it should be, although the long delays occasioned by the refereeing process can be rather frustrating.

Apart from my university activities, I have served on the main national and international committees concerned with research in the physiological sciences. From 1959 to 1966, I was a member of the Research Advisory Committee of the Australian NH&MRC. This committee is responsible for making recommendations regarding all government research grants in the area of the health sciences. Much later, from 1972 to 1976, I also served on the Australian Research Grants Committee (now the Australian Research Council), which recommends government research grants in areas other than medicine. In the international sphere, from 1968 to 1977, I was a member of the Council of the International Union of Physiological Sciences, and later I also was a member of the Governing Council of the International Brain Research Organization (IBRO). In 1960, I was one of the founders of the Australian Physiological and Pharmacological Society, organized its first scientific meeting, and served as its first treasurer. In 1967, I became a fellow of the Australian Academy of Science and, 10 years later, a fellow of the Royal Society of London. The Australian Honours List for 1986 made me an Officer of the Order of Australia, and the Commonwealth Government jointly awarded Horace Barlow, Vernon Mountcastle, and me the 1993 Australia Prize. I was pleased to be made an Honorary Doctor of Medicine by my old university as well as an Honorary Life Member of its faculty of medicine.

Though I have had a fortunate life, my one great sadness is that, with my exacting work schedule, I saw so little of my wife, Hilare, and my family. We have three children. Our elder daughter, Phillippa, married a cardiac surgeon, Douglas Baird, and they have four children, now all grown up. Our second daughter, Clare, is a senior member of the staff of the Department of Immigration in Canberra. Over a period of 15 years, she served abroad in posts as diverse as Hanoi and New York. Our son, Roderick, graduated in medicine at Sydney University and is a specialist in Emergency Medicine. He is married to Margaret Wallen, a pediatric occupational therapist, and they have one daughter.

Only by providing me with a stable home life and taking full responsibility for its management did my wife enable me to lead the kind of life that my work demanded. More than that, she also made the department very much a family affair, meeting overseas visitors and their families on their arrival and being generally concerned for their welfare, particularly during the process of settling into a new environment. We welcomed visitors to the department in our home, and once or twice during the year, but always at Christmas time, my wife entertained the whole department at our home. The many visitors to the department remember Hilare with warm affection.

Selected Publications

Bishop PO, Burke W, Davis R. The interpretation of the extracellular response of single lateral geniculate cells. *J Physiol (Lond)* 1962;162:451–472.

Bishop PO, Jeremy D, Lance JW. The optic nerve. Properties of a central tract. *J Physiol (Lond)* 1953;121:415–432.

Bishop PO, Pettigrew JD. Neural mechanisms of binocular vision. *Vision Res* 1986;26:1587–1600.

Bishop PO. Size constancy, depth constancy and vertical disparities: A further quantitative interpretation. *Biol Cybern* 1994;71:37–47.

Henry GH, Bishop PO, Coombs JS. Inhibitory and sub-liminal excitatory receptive fields of simple units in cat striate cortex. *Vision Res* 1969;9:1289–1296.

Joshua DE, Bishop PO. Binocular single vision and depth discrimination. Receptive field disparities for central and peripheral vision and binocular interaction on peripheral single units in cat striate cortex. *Exp Brain Res* 1970;10:389–416.

Kulikowski JJ, Bishop PO. Linear analysis of the responses of simple cells in the cat visual cortex. *Exp Brain Res* 1981;44:386–400.

Maske R, Yamane S, Bishop PO. Binocular simple cells for local stereopsis: Comparison of receptive field organizations for the two eyes. *Vision Res* 1984;24:1921–1929.

Nelson JI, Kato H, Bishop PO. The discrimination of orientation and position disparities by binocularly-activated neurons in cat striate cortex. *J Neurophysiol* 1977;40:260–284.

Pettigrew JD, Nikara T, Bishop PO. Binocular interaction on single units in cat striate cortex: Simultaneous stimulation by single moving slit with receptive fields in correspondance. *Exp Brain Res* 1968;6:391–410.

Sanderson KJ, Bishop PO, Darian-Smith I. The properties of the binocular receptive fields of lateral geniculate neurons. *Exp Brain Res* 1971;13:178–207.

Vakkur GJ, Bishop PO. The schematic eye in the cat. *Vision Res* 1963;3:357–381.

Other Publications Cited

Barlow HB, Blakemore C, Pettigrew JD. The neural mechanisms of binocular depth discrimination. *J Physiol (Lond)* 1967;193:327–342.

Dawson GD. Cerebral responses to electrical stimulation of peripheral nerve in man. *J Neurol Neurosurg Psychiat* 1947;10:137–140.

DeAngelis GC, Ohzawa I, Freeman RD. Neuronal mechanisms underlying stereopsis: How do simple cells in the visual cortex encode binocular disparity? *Perception* 1995;24:3–31.

Gabor D. Theory of communication. *J IEE (Lond)* 1946;93:429–457.

Hubel DH, Wiesel TN. Receptive fields of single neurones in the cat's striate cortex. *J Physiol (Lond)* 1959;148:574–591.

Lettvin JY, Maturana HR, McCulloch WS, Pitts WH. What the frog's eye tells the frog's brain. *Proc IRE* 1959;47:1940–1951.

Marčelja S. Mathematical description of the response of simple cortical cells. *J Opt Soc Am* 1980;70:1297–1300.

Martin GR. Schematic eye models in vertebrates. In: Ottoson D, ed. *Progress in sensory physiology*, Vol. 4. Berlin: Springer-Verlag, 1983;43–81.