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Pierre Buser began his research with neurophysiological studies of the optic tectum and then carried out pioneering studies on the electrophysiology of corticofugal projections and visuomotor integration.

He introduced the term fictive locomotion to describe the rhythmic discharges related to walking that can be observed in the absence of feedback from the limbs. He also conducted studies on thalamocortical electrobiological rhythms accompanying attentive states in behaving cats and monkeys.

Pierre Buser

I was born in Strasbourg on August 19, 1921. My parents both belonged to the population that had been migrating for centuries along the Rhine valley, from the source of the river in the Swiss Alps, up to Karlsruhe in Germany, passing by Zürich, Basel, Freiburg im/Breisgau, Mulhouse, Colmar, and finally reaching Strasbourg. Therefore, my origins are difficult to determine. All I knew, from my earliest days was that Switzerland was a kind of distant 'Heimat.' I very rapidly became bilingual, understanding, and later speaking, both an approximate form of French and an approximate form of Alsatian, a German dialect. Throughout my life, I have oscillated between two distinct cultures, and despite my sharp 'Frenchification' I have not forgotten my origins during my years of Parisian life. I married a 'true' French lady, spoke French, and later learned another language, common to all scientists all over the world. I am speaking of course of the approximate English, thanks to which we manage to understand one another.

In my early years, Strasbourg was still recovering from the World War I and from German occupation since 1870. I can remember that most of our school teachers came from outside Alsace and spoke only French, and we were (much to my bitterness, I must confess) dissuaded from speaking Alsatian at school. Seventy years later, at a time when Europe is growing and developing, this seems a somewhat obsolete approach.

My parents were very simple. My father sold small electric supplies (e.g., bulbs and brushes for electric motors). Life became difficult between 1929 and 1933 and I remember the efforts my parents made to ensure that I could continue going to school and to complete my examinations (the French baccalaureate). At the time, this examination had two parts: in the first year, Latin, Greek, French, math, and physics. The next year, a choice was given between mathematics and philosophy. I selected special mathematics, which attracted me very much. I passed the examinations with only very moderate success. I never regretted having learned some Greek and Latin. Even now, I occasionally open my Greek-French lexicon (bought in 1935!) to help me get to grips with some etymologies.

Just after obtaining these qualifications in 1939, war broke out. I was on holiday on the French west coast. My parents were obliged to leave Strasbourg, which was completely evacuated by order of the French

military authorities. They stayed with friends in the Vosges mountains, where I joined them, hoping to be accepted for a preparatory course for entry into the Ecole Normale Supérieure. I was accepted and spent 1 year at Tournon, a small town on the Rhone, which I discovered with an immense pleasure but where I saw, in 1940, the first Nazi troops invading France. After another year in Paris, I had one of my first real successes, one which would determine my career. I was lucky enough to be accepted by the Ecole Normale Supérieure. I stayed there, through the dreadful years of the Nazi occupation, graduating at the Sorbonne in physics and biology. When I entered the Ecole Normale Supérieure, I had to make a choice between becoming a physicist or a biologist. Much to the director's horror (he was an excellent physicist), I selected biology.

My first choice, however, was not neuroscience. I joined a group at the Pasteur laboratory of the Curie Institute, where studies on experimental cancer were being carried out. I spent 1 year there analyzing the effects of *per os* administration of methylcholanthrene, which is highly carcinogenic, on mouse digestive tract. My results were hopelessly negative (whereas my control subcutaneous injections of course induced severe sarcomas). Thereafter, I decided to change my field of interest. Before that, however, the war was in its latter stage. I volunteered for the army and was sent to North Africa, ready to fight in Indochina thereafter. Then the Hiroshima bomb was dropped, everything stopped, and I went back to civilian life.

My Early Years in Neuroscience

It took me some time to find a laboratory at which I could finally do what I had hoped, namely, work on the central nervous system. After some trials and errors, I discovered that the ideal place was not the Sorbonne, nor the College de France, but a tiny old cottage at the western limit of Paris, the Institut Marey. Alfred Fessard, who was not yet a professor, was the director of this institute and worked with his wife Denise (Albe-Fessard) and a technician. He welcomed me warmly. Shortly afterwards, Jacques Paillard and Ladislav Tauc arrived. We began to have many visitors. One of them was Yves Laporte; he was just back from a stay in New York, working with Rafael Lorente de Nò and later with David Lloyd, and was appointed professor at Toulouse. He often came to the institute and I remember our interesting discussions; we soon became close friends.

It also took me some time to establish a program for my doctoral work. I started recording electrocorticograms (ECoG) from the behaving rabbit (I think that this was probably one of the first studies in this line). However, I soon realized that my program (understanding the ECoG) was far too ambitious, and that I had to find a simpler model. Alfred Fessard suggested that I should consider the mesencephalic optic tectum of lower

vertebrates (from fishes to birds), a structure that is relatively simple because optic fibers arrive directly from the retina, run on its dorsal surface, and then turn at a right angle and have synapses with neurons with mostly radial dendritic extensions. In various fishes, particularly the catfish (my favorite species), this arrangement provided fairly good topological conditions for recording from the surface and, using fine isolated needle electrodes, at various depths. I was thus able to identify the topology of a dipole, oriented perpendicularly to the surface of the tectum. The very typical polarity reversal that I observed, from the dorsal to the ventral surface of the tectum, appeared as one of the most conspicuous models of a pure dipole. Many years later, and even now, while recording the ECoG in animals or in humans, I contemplated these much more complicated dipole-like activities, searching for their generators. I often think of these early data and wonder whether real progress has been made toward understanding the neurophysiological bases of the electroencephalograph (EEG). Another problem arose from this analysis of the tectum when I tried to understand why its response to single shock stimulation of the optic nerve mainly consisted of a slow wave (lasting approximately 20 milliseconds). I managed to demonstrate that this wave was due to slow conduction in dendrites. I had some strong evidence, and this was the conclusion that I developed in my D.Sci. defense. However, even now, I believe that I did not obtain definite proof and I know of no one who has advanced further in this analysis. I remember Rafael Lorente de Nò, to whom I explained my difficulties, very severely telling me in his bass voice, 'my young friend, your structure is too complex.' Of course, he was referring to the extreme complexity that Cajal, and his brother Pedro Ramòn, had identified in the neuronal arrangement of the fine tectum circuitry. He was certainly right.

During my thesis work, I also wanted to study other methods and concepts, and I was especially interested in structural studies on the brain. I therefore decided to spend time in one of the most outstanding laboratories in human architectonic studies headed by Oskar Vogt and his wife Cecile Vogt, who had already been his coworker for more than half a century. I stayed at this laboratory at Neustadt im/Schwartzwald, in the middle of the German Black Forest, for several months and it was a very fruitful experience. It is there that I had my first training in human neuroanatomy, which I used much later in my collaboration with Jean Talairach. I remember with some emotion listening to Oskar Vogt telling stories about his stay in Paris before World War I, with Dejerine at la Salpêtrière, while his future wife, Cécile, was working with Pierre Marie. The Vogt family had therefore been caught in the middle of the ancient fights between Dejerine's localizationism and Pierre Marie's antilocalizationism regarding aphasia. It was there, at the Hirnforschungs Institut in Neustadt, that I met Rolf Hassler and Jerszy Olszewski, both very

distinguished neuroanatomists but almost hidden by masses of histological slides of human brain piled up everywhere in their laboratories.

Back in Paris, after obtaining my D.Sci. on the optic tectum and its dipoles, I decided to cross the Atlantic, discover the New World, and, most important, get a new perspective on the exploration of the central nervous system.

Horace Magoun and the Reticular Formation at UCLA

Thanks to some friends, in particular Dr. Robert Livingston, I had the good fortune to be accepted at Dr. Magoun's laboratory at UCLA. The laboratory was not then at Westwood. The planned Brain Research Institute was only in the early stages of construction. We were housed in prefab buildings at the V. A. Hospital at Long Beach, California. Everybody there was busy with the ascending reticular activating system, accumulating a fabulous set of findings and theories on the control of the state of vigilance by a rather mysterious structure that had been previously described as the reticular formation by the anatomist Ramón y Cajal. A conceptual system was thus elaborated, according to which all vigilance states were controlled by pathways ascending from the mesencephalic and pontine brain stem. This raised another specific problem—that of the descending actions from the neocortex back to the reticular formation—and Dr. Magoun asked me to explore this in monkeys. The general atmosphere at Long Beach was extremely warm. Daily meetings with people such as Jack French, Don Lindsley, Ross Adey, Mike Verzeano, John Green, Eve King (later Mrs. Killam), and, not very far away on the UCLA campus, Ted Bullock were a marvelous surprise. Together with Jose Segundo, we were able to establish that in our macaques, a local electrical stimulation of a neocortical area elicited generalized arousal through its descending effects on the reticular formation. After my return to Paris from Los Angeles, Jose continued this investigation with Robert Naquet, who had just arrived. Everything was new to me: a new class of experiments and a very elaborate system of thinking. Dr. Magoun directed the laboratory with his own particular sense of humor ('Pierre,' he once said to me, 'don't worry, if you don't find what you expected, somebody else will find it!').

The climax of this first New World experience occurred when I was lucky enough (thanks to one of its organizers, Herbert Jasper) to be invited to attend the symposium organized at Ste. Marguerite (Quebec) as a satellite to the 1953 International Physiological Congress (Montreal). At this laurentian meeting, the title of which was Brain Mechanisms and Consciousness, I discovered many of the key players in the new push given to studies on the mammalian brain: from histology, Walle Nauta and Jerzy Olszewski; from physiology, Horace Magoun, Giuseppe Moruzzi, Herbert Jasper, and Mary Brazier; from psychology, Donald Hebb and Karl

Lashley; from pathology, Richard Jung and Wilder Penfield; and so many others. Alfred Fessard delivered a very elegant paper on his theoretical views on consciousness. I remember this meeting as one of the most illuminating events in my scientific life. My feeling after leaving Ste. Marguerite was that brain research should be pursued along the lines followed by the Los Angeles group. In retrospect, I think that this initial push kept dominating many of my choices, even though I was determined to follow up my own ideas and the programs that I wanted to initiate.

Back to Paris at the Institute Marey (1954–1961)

After returning to Paris, I became a full professor in 1955. Initially, my teaching duties (a high load indeed) were split into two: courses in basic biology for medical students, whose first year was spent in our School of Sciences, and courses in experimental psychology for students of the Institute of Psychology of Paris University. The basic biology teaching program was of no interest to me. I think that I did my job reasonably well, probably as a result of an innate gift for teaching, but it gave me no pleasure. On the other hand, teaching physiological psychology (neurobiological bases of behavior) required a considerable number of background readings. What I learned in order to prepare my seminars became an appreciable source of knowledge that enabled me to make new choices, given that I had decided to stop working on lower vertebrates as soon as possible.

My own plans were temporarily delayed by an unexpected (happy) event, namely, the advent of a new technology, glass micropipettes, which made intracellular explorations possible. Denise Albe-Fessard persuaded me to collaborate with her in intracellular explorations in the cat somatomotor cortex. Her skills in electronics (building up ‘cathode followers,’ special amplifiers to pick up bioelectrical activities with high-impedance electrodes, which were not commercially available at that time) and mine in stereotaxy that I had learned at UCLA were of course key factors favoring success. We were indeed successful. I have never been able to determine whether we were the first to carry out intracellular studies of the cortical pyramidal neurons. I suspect that our late friend Charles Phillips probably preceded us by some months, following of course John Eccles’ success with spinal motoneurons. We were indeed very proud to penetrate cortical neurons, to measure their membrane potential, to watch EPSPs and IPSPs, and, above all, to characterize the short latency of responses to somaesthetic stimulation. We thus found that cortical (presumably pyramidal) cells made a major contribution to the early phase of the classical biphasic evoked potential, a new and unexpected finding. Of course, when I now contemplate the huge number of studies performed since, with much more elaborate techniques, in the field of intracellular recording

from cortical neurons, I realize that our tools were at that time far from perfect.

The time had now arrived to start my own programs. (Note that I use the plural to designate my work from this time forward.) This was probably one of my major mistakes. Very quickly, several young collaborators asked to work with me. Instead of wisely saying no to some and yes to a select few, I welcomed them all. Perhaps this was because my ego was flattered, but it was probably also because I was full of ideas for future experiments that I evidently could not perform by myself.

Visual Pathways in Pigeons: Picking Up from Where I Left Off

The tectum experiments had led me to study pigeons and I did not want to abandon them completely. One of my new students, Arlette Rougeul, was interested in studying the visual pathways in this species for her M.D. thesis. I suggested that she complete the work I had initiated on the tectum and extend electrophysiological exploration to other brain structures. I worked with her and we made three discoveries. First, we showed the possible existence of uncrossed optic fibers in the optic chiasma by detecting the presence of short-latency visual responses on both tecta after occlusion of one eye. These results conflicted with well-established data and we were strongly criticized by histologists who could not confirm the presence of uncrossed fibers in the optic chiasma. It is only fairly recently that, much to our satisfaction, it has been clearly established by new marking techniques that uncrossed fibers are indeed present. Second, we accumulated evidence that the telencephalon also receives visual messages. This result was later widely confirmed. Finally, we observed that the cerebellum was also activated by visual stimuli. We used the newly acquired intracellular recording method and, much to our surprise, we discovered that whereas some intracellular responses of Purkinje cells resembled those of neocortical pyramidal neurons (as previously observed with D. Albe-Fessard), others were totally different, appearing to be long-lasting depolarizations on which were superimposed several small spikes. Unfortunately, we did not go further; however, when, shortly afterwards, Granit and Phillips described 'complex spikes' of the Purkinje cells, we realized that what we had recorded were in fact such complex spikes.

The Multimodal Associative Areas in Cat Cortex

Shortly afterwards, I initiated my first topographical studies on cat neocortex. My aim was to explore the associative cortical areas. At that time, very fine explorations had already been achieved, initiated by E. Adrian and extensively carried out by C. Woolsey and many others since,

showing precise topographical correspondences between the sensory periphery and its cortical representations, but these were restricted to the primary receiving areas. I had difficulty accepting the idea that areas such as the suprasylvian gyrus in cat were sensorily silent, receiving no incoming information. I suspected that the depth of anesthesia was probably a factor limiting the extension of some putative projections to the associative areas. Together with P. Borenstein, a psychiatrist who was keen to spend time performing animal experiments, I explored cats that were under very light anesthesia and paralyzed with a curare-like substance. We decided to concentrate on visual and auditory global responses (evoked potentials, now often called field potentials) as indices of afferent sensory messages. Averaging devices were not available, and we simply superimposed several successive sweeps. This led us to the following conclusions: (i) Several foci in the associative suprasylvian area display visual and auditory, long latency, and small amplitude evoked responses; (ii) these responses disappear when the animal is highly aroused with low-voltage, fast-running electrocortical activity; (iii) they are completely masked when the animal is in slow sleep with delta activity and spindles. In other words, there is a state of optimal vigilance, apparently favoring the diffusion of visual and auditory information to the associative areas; and (iv) these associative responses are not observed under deep barbiturate anesthesia but are highly amplified by another narcotic substance, chloralose, through a mechanism that remains mysterious even today. Of course, in chloralose-treated preparation, the associative potentials show no amplitude variations with vigilance, but their topographical distribution over the cortex is much easier to determine.

Another interesting observation was the existence of associative-like potentials in the primary area for the other modality, i.e., visual EPs in the auditory areas and auditory EPs in the visual area. Given what we now know about cross-modal activations from recent PET and fMRI imaging, these data were indeed relevant.

A second question that arose was whether the primary motor cortex was a multisensory area. Based on the data we obtained at this time with Michel Imbert, we concluded 'yes.' We again worked on lightly anesthetized, curarized animals and animals under deep chloralose narcosis. We refined our investigation, performing a single-unit extracellular study. Our results were fairly clear. We showed that a large number of cells (presumably pyramidal neurons) in the cat motor cortex reacted to the three principal stimulus modalities—visual, acoustic, and, of course (but this was not a new finding), somatic.

By combining these results, we developed a (provisional, very schematic, and somewhat naive) view of the cat neocortex, with specific sensory areas (also receiving messages from other modalities), associative multimodal areas, and the primary motor cortex, which was also a multimodal

structure. Looking back over the period from the time of these experiments to the present day, some of our (very approximate) results have been confirmed using much more sophisticated electrophysiological or imagery methods.

Cortical Transient, Top-Down Permissive Controls on Subcortical Structures

During my time at UCLA in Dr. Magoun's laboratory, I was faced for the first time with descending actions from the monkey neocortex down to the activating reticular system. After exploring the cortical associative areas, I decided to return to the problem of descending corticofugal actions, this time in cats. The chloralose preparation, which was very stable, provided good conditions for exploring, as systematically as possible, the effect on subcortical structures of altering the pattern of functioning of a given cortical area. In the Los Angeles experiments on monkeys, we had used focal electrical stimulation of the cortex. In this series, we decided to generate the reverse effect, namely, transient abolition of the activity of a given cortical area by local cooling, which would presumably suppress all descending corticofugal messages. Taking the amplitude of the evoked potential of the treated area as an index, we followed changes in the amplitude of evoked potentials to sensory stimuli recorded from 'nonspecific' subcortical areas, such as the mesencephalic reticular core, and the nonspecific thalamic nuclei, center median, and the intralaminar nuclei (centralis lateralis or parafascicularis), which were already known to be multisensory, displaying responses to various external stimuli. We also checked the sensory responses in the corresponding specific thalamic nuclei (lateral geniculate, medial geniculate, and ventralis posterior), knowing the numerical importance of the descending connections from specific sensory areas back to their corresponding thalamic nucleus. Our results were unexpected: (i) If a given sensory area were blocked, the sensory nonspecific thalamic or reticular response to that same modality was completely abolished as long as the sensory cortex was depressed and its network presumably not working; (ii) contrastingly, nonspecific responses to other modes of sensory stimulations remained unchanged; and (iii) surprisingly, the sensory responses in the corresponding specific thalamic nucleus were not affected. From this inspection of gross evoked potentials, we concluded that the cortex exerts an instantaneous permissive control (the nature of which remains to be discovered) over the processing of sensory messages for the same mode of stimulation in the nonspecific structures.

The other (apparently paradoxical) finding was that, after surgical decortication followed by a period sufficiently long to allow recovery (e.g., a few hours), nonspecific projections were again present, showing

that the observed subcortical depression was only temporary and that the original sensory messages were actually due to an ascending action (as already demonstrated by several other groups) and not simply to a descending volley from the cortex. These messages, which would otherwise reach the nonspecific structures in the absence of the cortex, seemed to be under transient permissive facilitatory cortical control if the cortex were present.

We were naturally concerned that all our data on cortical descending control had been obtained using animals under deep chloralose anesthesia. We therefore undertook a series of investigations on curarized, slightly anesthetized animals. We recorded single units from the mesencephalic reticular core, controlled their reactivity to light and sound, cooled the visual cortex, and observed exactly the same selective disappearance of the reticular responses to visual stimuli, with the acoustic responses unaffected.

I have two final remarks to end this section. First, the facilitatory action from the neocortex down to the nonspecific core conflicts entirely with what one would have expected from the common belief that the neocortex inhibits a variety of deep structures. Second, recent, more refined studies have demonstrated what we could not observe in our recordings, even with single units—namely, that one sensory area at least (the visual area) strongly modulates responses in its specific thalamic relay (the lateral geniculate nucleus). Evidently, to demonstrate this descending corticothalamic influence required much more sophisticated patterns of visual stimulation than the ones we used.

Events inside and outside the Laboratory

In the meantime (1961), I had left the Institut Marey and moved into the brand new buildings of our Faculté des Sciences. There, I had more space and could establish several laboratories. I was surrounded by a very active team composed of Philippe Ascher, Michel Imbert, Jan Bruner (a former research associate of Jerzy Konorski at Warsaw), Nelly Zilber, and Arlette Rougeul, whom I married in 1962. Other collaborators included Dora Gerschenfeld, Cesira Batini (who moved from G. Morruzzi's lab to mine), Mario Wiesendanger from Zürich, Horacio Encabo from Buenos Aires, and Jacques St. Laurent from Canada. Michel Lamarche also joined our group.

Our real problems began in 1968 with student strikes and riots. Our laboratory was not greatly affected, although work was interrupted for about 6 months. Things were never the same. The government produced new legislation, completely reorganizing the educational system. The Faculté des Sciences became two separate universities called Paris 6 and Paris 7. These two universities occupied the same campus, with no real borderline between the two. Paris 6 (my university) became Université

Pierre et Marie Curie, and Paris 7 became Université Denis Diderot. After so many years, one can only regret this division. Most of the neuroscientists became part of Paris 6, including, over the years, Yves Galifret, professor of psychophysiology, who set up his own laboratory; Denise Albe-Fessard, who joined the university after the Institut Marey closed; Marie-Jo Besson, a neurochemist; and Jacques Taxi, a cytologist, successor to René Cousteaux, an internationally known specialist of the motor endplate. Our labs were mainly supported by the CNRS (Centre National de la Recherche Scientifique), with less money allocated by the university. The next major change was the decision by the CNRS in 1985 to create the 'Institut des Neurosciences' at Université Pierre et Marie Curie. This institute still exists. I became its first director general, and it had four separate departments—those of D. Albe-Fessard, who soon retired and was replaced by Michel Imbert, Galifret, Jacques Taxi, and my own, the running of which was taken over by my wife Arlette. I was replaced by André Calas, a specialist in *in situ* hybridization, when I retired at age 70 in 1991 (a late retirement because I have three children).

Until about 1979, I taught neuroscience at the undergraduate level, year after year telling my students what happened in axons and in the spinal cord, describing in great detail all of the Sherringtonian laws of reflex activities, and talking much less about the thalamus and the neocortex because of lack of time. In 1979, I was officially appointed to organize (to reorganize is perhaps a better way to put it) courses for graduate students. In planning this new higher level teaching program, I decided to achieve an old ambition. As a young assistant professor, beginning my research in Alfred Fessard's laboratory, I had discovered with much sorrow that there were three independent schools of thought and experimentation in neurosciences in France, at best ignoring each other and at worst fighting among themselves: neurophysiologists working at the schools of sciences, neurophysiologists with a medical tradition at the schools of medicine, and the experimental psychologists and psychophysicologists. I was profoundly shocked by this situation. Almost 30 years later, while organizing graduate teaching, I firmly decided to have all three subsets of neuroscientists (in the broader sense) involved in my training program. I invited cellular neurobiologists, system neurophysiologists, some Ph.D.'s, some M.D.'s, basic scientists or clinicians, and psychophysicologists and, recently, neuropsychologists. I am proud that I began a process that has since become more widely adopted in the teaching of neurosciences. My only regret is that new trends are beginning to develop, with a tendency to return to more specialized teaching, oriented toward neural reductionism, or else ignoring elementary levels and focusing on cognitive sciences.

Now I return to research after this short interlude of teaching and administration.

Mechanisms of Visuomotor Performance: From Acute Models to Studies with Behaving Animals

After demonstrating a permissive control by the visual cortex of a variety of subcortical nuclei, we analyzed the effect of reversible cooling of the visual cortex on input–output operations at the level of the motor cortex. First, with Philippe Ascher, we took the pyramidal tract discharges as a baseline control. We knew from the work of Adrian and Moruzzi that in chloralose preparations all types of brief sensory stimulation can determine a short, phasic discharge in the pyramidal tract. The effect on these discharges of reversible cooling of the visual cortex showed that pyramidal responses to light were selectively abolished, whereas responses to sound and to somatic stimuli remained unaffected. Moreover, we demonstrated through a variety of lesion experiments that this permissive control by the visual cortex involved a complex loop including subcortical nuclei projecting into the motor cortex. This view is clearly not consistent with current ideas because most interarea cortical connections are now considered to be corticocortical. Later, with Mario Wiesendanger, we confirmed these data, this time by recording the visual responses of single pyramidal cells identified by antidromic stimulation of the pyramidal tract. Those were our last experiments under chloralose; from then on, this anesthetic was banished forever from our laboratory.

I very much wanted to confirm our conclusions on the visuomotor loop with animals performing tasks. At that time, my future wife, Arlette Rougeul, had already developed methods to train cats to press a lever in response to a given signal to get a food reward (an instrumental conditioning protocol). The signal was a series of visual flashes or of tone bursts. For this particular purpose, the animals were overtrained to press the bar to either light or sound, indifferently. A cooling device was implanted on their visual cortex.

If our acute data were correct, the animal should, during cooling of the visual cortex, cease to respond to the visual stimuli but continue to press the bar in response to the tone bursts. This is indeed what happened, confirming our hypothesis of permissive transient control of the motor cortex (presumably responsible for the pressing movement) by the visual cortex. Again, if our hypothesis about this transient control were correct, cats permanently lacking their visual cortex (bilaterally surgically removed) should, after recovery, be able to press the bar in response to a visual stimulus. We thus confirmed *en passant* a very old finding, the persistence of residual vision in the absence of visual cortical areas, just as we confirmed our hypothesis on the permissive transient cortical control. It was as if there were a permanent effect of the visual cortex on the traffic of messages on their way to the motor area. However, in the chronic absence of the sensory area, a certain extent of

recovery may take place, allowing these messages to reach the motor cortex again.

Further Studies on Visuomotor Mechanisms in the Cat

Later, in the 1980s, I undertook a further, more analytical investigation of the mechanism(s) involved in visual guidance of a tracking movement in cats. With Michèle Fabre-Thorpe, a graduate student who has since become a senior researcher at Toulouse, I tried to identify the structures that are required for the performance of a visually guided movement. After some trial and error, we built an experimental setup consisting of a light spot moving at random in front of the animal, under a translucent screen (in fact, the spot was a small bulb fixed to the pen carrier of an X-Y plotter): The animal was rewarded if it touched the spot with its forepaw, the dependent variables being movement time and accuracy. This model proved to be extremely useful in analyzing the importance of a variety of structures in visuomotor skills. We thus identified deficits after lesions of the anterior suprasylvian cortex (areas 5 and 7), of structures belonging to the extrageniculate subcortical system (colliculus and pulvinar), and the possibility of relearning rapidly after major bilateral ablation of the visual areas. One result was quite unexpected. It concerned bilateral elimination of the nucleus ventralis lateralis (VL), one of the most important thalamic nuclei thought to participate in the activation of the motor cortex. Much to our surprise, ablation of this nucleus did not significantly affect the animal's performance, provided that it had been overtrained beforehand. On the other hand, we tried to train some VL-lesioned naive cats to perform this task. We knew from experience the average training time required to reach criterion. It soon became clear that animals lacking their VL thalamic nuclei were completely unable to learn the task (despite appearing clinically normal otherwise). We concluded from these experiments that at least one subcortical structure (the VL thalamic nucleus) is indispensable for acquisition of the visuomotor skill, but it is no longer essential once the task has been well learned. This was (to me) one more example of a transient, time- and state-dependent function. Similar observations were performed in the monkey at about the same time by Vernon Brooks with A. D. Miller (see Brooks' interesting remarks on this point in this volume).

A Short Episode with Locomotion: Creating a New Term, 'Fictive Locomotion'

Why did I suddenly switch to locomotor pattern generation in mammals? Probably because I had, among my many other interests, an interest in understanding the central programming of efferent activities. The concept

that walking could simply be the result of a chain of reflexes was quite unacceptable to me. I wanted to explore whether the spinal cord, with or without certain supraspinal structures (including perhaps some parts of the reticular formation), can organize locomotor movements in the absence of any feedback information from the moving limbs. We studied, with Claude Perret, Didier Orsal, Jean-Marie Cabelguen, Guy and Denise Viala, and, for a time, Alain Berthoz, the behavior of unanesthetized spinal or mesencephalic animal preparations (the latter comprising spinal cord and brain stem) immobilized with a curarizing drug blocking all phasic messages from receptors, tactile or proprioceptive, that would normally be produced by limb movement. The results were as expected: Recording from peripheral nerves, we observed rhythmic discharges in perfect alternation in the nerves to flexors and to extensors. In the rabbit, the left and right sides were symmetrically active, mimicking the synchronous jumping pattern usually observed in this species, whereas cat preparations displayed alternating patterns (left extension with right flexion and so forth). We created a new term, first in French, which very quickly and rather amazingly became accepted elsewhere—fictive locomotion. My coworkers in this adventure of fictive locomotion were all very competent, each in his or her own field, so my personal contribution to these studies on locomotor programs was only very temporary. It seems to me that I helped to launch these studies but quickly lost contact with the multiple and complex details that were elaborated by them (and which are still being generated). This was particularly true because they all left the laboratory in the late 1980s to become professors in other universities and to set up their own laboratories.

Last Studies on Acute Cat Preparations: Investigating Corticocortical Callosal Actions

One major question remained unanswered after my previous studies on corticofugal permissive influences: Does the cortex always act as a reflex network, sending back a volley (via its descending long axons) after receiving an afferent message, or are there more subtle conditions for this reflex type of functioning? This led me to select another model, in which efferent activity could be more easily followed: the commissural connection of a given cortical area to the symmetrical contralateral one via the corpus callosum. So started my last adventure with acute cat preparations. One of my students in the late 1970s (Chantal Milleret) initiated this study. She chose to work on the primary visual cortex and its callosal interconnections in adult cats. After sagittal section of the optic chiasma and the covering of one eye, the ipsilateral visual area is deprived of its visual afferences, except the callosal ones originating from the contralateral area. We were struck (as others had been before us) by the reliability and

precision of timing of this transhemispheric activation when the contralateral area is visually stimulated. We then investigated changes in the size and other characteristics of the 'transcallosal receptive fields,' with time after chiasma transection and eye occlusion. Our principal finding was that size increased up to about five times the control surface, indicating that the efferent volley originating from the other side activated a larger number of cortical columns, and that a so-called plastic change had occurred during the time after chiasma transection (plasticity in adults has now become a well-known process, but it was relatively new when we made our first observations in the early 1980s). This enlargement of the fields is progressive and slow, and it is complete about 30–45 days after chiasma section and eye occlusion. In contrast, when the eye is uncovered at the time of the final exploration, it takes only about 1 hour for the field to return to its normal size. This amazing difference in time courses probably rules out structural changes at cortical synapses and instead suggests a process based on changes in neurochemical receptors. Currently, we continue to explore the mechanisms involved in these plastic changes affecting the cortical map of the transcallosal visual field. Since I retired, this new series of experiments has been carried out at Alain Berthoz's laboratory at the College de France, with Chantal having become a member of this laboratory.

Working with Arlette Rougeul, My Wife, and Her Group : The Long Story of Electrocortical Rhythms in Behaving Cats and Monkeys

Very soon after returning from Los Angeles, I was very keen that a group in my laboratory should work on behaving animals with implanted electrodes in a state that would now be qualified as 'conscious' (a term that was almost prohibited at that time). This was rather new in 1955 and most of my coworkers were reluctant, preferring more comfortable acute explorations. Therefore, it took me some time to organize the 'chronic cat lab.' Arlette Rougeul, abandoning her pigeons, finally accepted the challenge. Thus, we started a close collaboration that has lasted for 45 years and continues.

Of course, our technical approaches were at first rather unsophisticated. Unit recordings were not considered possible and we therefore concentrated on a variety of programs that could be carried out with gross electrode recordings from the cortex or deep structures.

The first approach closely paralleled the set of acute explorations that I was performing at that time: Arlette and I made systematic recordings of evoked potentials from a variety of neocortical areas. Basically, we superimposed oscilloscopic tracings in the peristimulus period. Our data closely resembled that for acute preparations: (i) Sensory multimodal projections

were present in associative areas, especially the suprasylvian cortex, expressed as long-lasting, low-amplitude evoked potentials; (ii) these EPs had their optimal development in states of 'quiet waking'; (iii) they almost completely disappeared in states of high alertness with full ECoG desynchronization; (iv) they were masked during slow sleep with extensive delta activity and spindles; and (v) cross-modal EPs also existed in a given primary receiving area for the stimulation of other sensory systems.

Arlette then decided to train cats to press a lever to obtain food (as described previously). The animals were implanted with electrodes at several cortical sites and their running, spontaneous ECoG was recorded before and during each trial. Nothing unexpected resulted from this first experimental series, except for the fact that the stimuli that we used, positive 'go' stimuli and negative, differential, or 'no go' stimuli, were brief flashes or brief tones repeated at a frequency of 2 per second. In response to the positive stimuli, the ECoG suddenly displayed clear desynchronization at a given latency after their onset, just before lever pressing. In response to the negative stimuli, the animal very rapidly developed slow patterns, suggestive of a kind of drowsy state. Interestingly, the latency of onset of these drowsiness patterns was similar to that of desynchronization. This suggested that 'refraining from moving' was an active process, with a precise time of onset after the no go stimulus, similar to the time of 'decision to move' after the go stimulus. Incidentally, the presence of a drowsy state and accompanying slow ECoG patterns in the no-go situation fit quite well the Pavlovian hypotheses about 'internal inhibition' elicited by negative stimuli, a concept long since forgotten but widely accepted at the time. We were pleased to observe this correlation in well-defined conditions.

My wife's leading idea then became to try to observe spontaneous changes in the running ECoG in behavioral conditions as close to normal as possible. She considered that the bar-pressing situation was artificial. Therefore, we launched a new program, which we have been carrying out for more than 20 years, with many collaborators over the years (J. J. Bouyer, M. F. Montaron, M. Chatilah, L. Dedet, etc.) on cats implanted with multiple arrays of closely arranged cortical electrodes, allowing a very systematic topographical exploration of the cortex. These animals were placed in two different situations in which they displayed a behavior suggesting attention.

The first was fairly classical: We placed the cat in front of a mouse protected by a transparent perspex box. A 'good' cat would watch, motionless, the visible potential prey for several minutes. We expected to record simultaneously a low-voltage fast ECoG activity. We observed instead (to our great surprise) long-lasting sequences of well-developed rhythms around 35–40 Hz. This was at the time when we could afford to buy our first computer (it was a huge PDP!). We processed the records with the

brand new automatic fast Fourier transform algorithm and obtained what are now termed waterfall displays showing that rhythms at a very constant frequency occur during the period of sustained focused attention. These rhythms were restricted to two foci of limited extent—the motor cortex (Brodmann areas 4 and 6 a) and the posterior parietal area 5. To pay tribute to the EEGers who first described them in the human motor cortex (Jasper and Penfield), we called them ‘beta.’

In the second situation, the cat had to wait for a mouse placed behind an opaque wall. It could hear it, smell it, and possibly even see its nose popping out of a small hole made in the wall, but it never caught it. A ‘good watcher’ would usually remain motionless in front of the hole for several minutes (or sometimes much longer). In these conditions of waiting for a prey, typical electrocortical rhythms also occurred in successive trains, but their frequency and location differed from those of the beta rhythms: They were very precisely situated in the cortical somatic area SI and their frequency was very close to 14 Hz. Again to pay tribute to the discoverers of such rhythms in humans (H. Jasper, H. Gastaut, and G. Chatrian), we called them ‘mu rhythms.’ We concluded that beta and mu rhythms are determined by the kind of attentive state: beta activity in a classical situation of focused attention on a given item and mu in a situation of conditional expectancy of an event to occur (a Bayesian situation, as some might now say).

In 1977, a symposium was held at the Senanque Abbey, in the French Provence, titled *Cerebral Correlates of Conscious Experience*. Paul Dell, a French neurophysiologist, had taken the first steps to organize this meeting, but he unfortunately died in 1976. The project was taken over by a ‘French triumvirate’ (Sir John Eccles’ term!)—Michel Jouviet, Robert Naquet, and myself—and the congress was a success, with Sir John, Vernon Mountcastle, Benjamin Libet, Giovanni Berlucchi, Rolf Hassler, Janos Szentagothai, Brenda Milner, Hans Kuypers, and many others (Dr. Karl Popper was also invited, but unfortunately he could not attend). On this occasion, Arlette and I delivered a paper summarizing our current views on electrocortical rhythms and short-term fluctuations of selective attention. It gave rise to some difficult, though fascinating discussions because not everyone believed in using the running ECoG as a functional index. Finally, my wife and I were committed to the difficult task of editing the proceedings, which were published by Elsevier.

Much later (in fact, 15 years after our first description of the beta rhythms), ECoG activities in about the same frequency band were described, first in the rabbit olfactory bulb, by Freeman and somewhat later by Pöppel, Eckhorn, Engel, Gray, and Singer, mainly in the visual area. These authors agreed to call them ‘gamma’ (to distinguish them from all other described rhythms) and this began a long series of studies

and interesting hypotheses, the most fascinating being of course their involvement in the interneuronal 'binding' assumed to underlie perception. We were very pleased to see other groups finally interested in the functional meaning of the ECoG, for which we had fought for years. However, we became hesitant about the terminology that we should adopt: Should we change betas into gammas or not? We soon decided to keep our own because we realized that the gammas as they were described were essentially stimulus locked, whereas our rhythms were mainly state dependent.

Our story then continued. In brief, we finally (after many years!) performed thalamic single-unit explorations in the conscious but painlessly fixated animal placed in a situation to develop one of these two types of rhythms. We thus explored several thalamic nuclei, especially the ventroposterior (VP) nucleus, the probable thalamic participant in mu rhythms, and the nucleus posterior (PO), the probable focus for the parietal beta rhythms. We also investigated the nucleus reticularis and found no neuron that accompanied any such waking rhythms, but we confirmed that this nucleus is involved in sleep spindles. In other words, we have been unable to confirm the current popular contention that the 'attentional spotlight' involves the nucleus reticularis. We tend to consider that it is as if several distinct thalamocortical channels in the waking animal may independently or in correlation become rhythmic at a given moment, depending on the requirements of planning of perception, attention, or action. These channels can also be modulated by noradrenaline and dopamine (as we showed in a long series of neuropharmacological studies not described here). We still need to find explanations for the concomitance of mu and beta rhythms and motionless attentive states: Thus far, we have developed no plausible functional hypothesis at the neuronal level similar to that proposed for binding in perception for the gamma rhythms.

What next? During the 5 years before retirement, we stopped working on cats and carried out a study in macaques. We were lucky to be able to use the Psychological Testing System (kindly placed at our disposal by Dr. D. Rumbaugh). We used it to test focused visuomotor attention, accumulating as much data as possible on videotapes and ECoG record tapes to prepare for the time when we would no longer be actively working on animals but would still have computers at our disposal. This is the current situation. Currently, we are studying our monkeys' ECoGs with some new technology in signal processing based on time-frequency analysis (wavelet decomposition), and we look forward to obtaining a set of new data on the ECoG correlates of visuomotor operations. Our laboratory has been taken over by a very active group headed by Susan Sara, a specialist in mechanisms of memory and who kindly manages to provide us the best possible working facilities.

Working on Human Epilepsy at Ste. Anne Hospital: A 25-Year Collaboration with Jean Bancaud and Jean Talairach

I remember a day in the early 1960s when Jean Bancaud, a well-known French epileptologist and EEGist, invited me to attend a session for the exploration of a patient suffering drug-resistant focal epilepsy. The only available treatment at that time, and currently the only treatment in many cases, is to remove the epileptogenic focus by surgery. To guide the focal ablation, it was necessary to explore the patient with many indwelling electrodes, introduced in and around the area neurologically and electroencephalographically identified as the possible focus. The routine procedure was to try to reproduce the patient's seizure by stimulating one explored site (in the best cases) and to record from as many structures as possible (neocortical, limbic, or sometimes deeper structures such as thalamic nuclei or basal ganglia) to gain insight into the extension of the fit. Jean Talairach, our neurosurgeon, had already produced a very precise stereotaxic atlas based on specific coordinates. My weekly collaboration with the Bancaud and Talairach team began soon after this session and lasted for about 25 years. We explored one patient per week. I was lucky to have access to a variety of structures, to be able to record from them on oscilloscopic tracings, and to stimulate them (gently, to avoid inducing seizures). I could thus often contribute to the localization of the focus and took this incredible opportunity to analyze more closely a variety of intracerebral connections. Several structures were my favorites: relationships between hippocampus and amygdala (which behave differently in normal and temporal lobe epilepsy); corticocortical connections between structures on the midline, with emphasis on the transcortical callosal links between the symmetrical supplementary motor areas (SMAs); ipsilateral interconnections between SMA and the anterior cingulate gyrus; and connections between anterior and posterior cingulate. I collaborated on the second atlas, published in 1967. Recently, Talairach published with Tournoux two other atlases that have rapidly become well accepted by the neuroimaging community and are cited in a considerable number of recent PET and fMRI publications. After Talairach's retirement and Jean Bancaud's death, I continued to collaborate with the Ste. Anne group, first with Patrick Chauvel, who is now in Marseilles, and currently with Michel Lamarche. We are currently exploring various brain sites on the cortical midwall, in particular the cingulate gyrus, while our patients are asked to perform some (very simple) cognitive tasks.

Writing Books

One of my favorite tasks, aside from research, teaching, and administration, has been to write books for our students in neurosciences. I thank

Michel Imbert for his help. It is not that he wrote one-half of the book and I the other, but instead his main assistance was in the area of diplomacy. The acting director of our publishing company (Hermann Publishers) was a difficult man who was not interested in the scientific content of the books nor in their marketing, and Michel's influence was highly appreciable. We wrote six volumes (*General Neurophysiology*, 1975; *Sensory Physiology and Psychophysiology*, 1982; *Vision*, 1986; *Audition*, 1987; *Basic Neurobiological Mechanisms*, 1994; and *Autonomic Mechanisms*, 1996). Two of our books, that on vision and that on audition, were translated into English and published by MIT Press. When I look back, I think that writing these books was probably a mistake. Very few copies were sold, especially those written in French (French students appear to be very reluctant to buy textbooks!). In compensation, I recently wrote, this time alone, a book for a larger audience that described my neurophilosophical views on consciousness, the cognitive and affective unconscious, altered states of consciousness, and hypnosis, with a final chapter on meditation. It was published in French in 1998 by Odile Jacob and recently translated into Italian (McGraw-Hill Italia). It took me 5 years to write it, with periods of great pleasure and episodes of sorrow and tears.

Epilogue

Here ends the story of my journey through the neurosciences. Do I feel satisfied? Certainly not. I have too many feelings of not having achieved my goals, of having probably not been at the right place at the right time, and of having missed good opportunities to make my results more accessible to the international neuroscientific community. Publishing too often in French rather than in English is probably a contributing factor. Moreover, the 'publish or perish' principle was not as strong then as it is now.

I never, except perhaps at the very beginning, followed fashion, abandoning a line of research to start a new one in a popular new field that had just opened up. During my long life I have seen too many discoveries suddenly attract many researchers and give rise to meetings, discussions, proofs and counterproofs, and masses of publications, only to fall just as rapidly into oblivion or decline. I generally stayed away from these sudden novelties, always keeping faith with my own line of work and my own programs. I still do, for better or worse. Now, allow me to leave you, dear reader, to go back and analyze our ECoG monkey data plus some other EEG data gathered recently from human subjects playing with a joystick in a visuomotor task. Goodbye.

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