SPECIAL LECTURE

Some reflections on approaching retirement

J Payan

My first reflection is a melancholy one, that recruitment to our specialty is poor and that when numbers fall so does quality. This has made me think about organisation, recruitment and training, the usefulness of our contribution, and some aspects of our practice. I hope it will not be without interest if I start by retracing some of the steps which have led to my standing here today.

Clinical electromyography (EMG) is a relatively young subject: its first international conference took place in Padua in 1961, the year I became a member of the Royal College of Physicians. The following year unabashed hedonism drew me to Stanford, California as a resident in neurology. I chose Stanford because of its location and neurology in the expectation, on the whole fulfilled, that my clinical training would enable me to survive in a technically advanced medical environment.

My time was divided between the Palo Alto-Stanford Medical Centre and the Palo Alto Veteran's Administration Hospital. One weekend we were taken to San Francisco for a seminar on EMG at which the principal speakers were Fritz Buchthal from Copenhagen, Herbert Jasper from Montreal and Ed Lambert from the Mayo Clinic. I remember almost nothing of the occasion except an overwhelming desire to sleep. Our own struggles with patients in a wire cage had a Heath Robinson quality which made it difficult to take the subject seriously.

I had been encouraged to find that here the practice of neurology was seen as a legitimate and attainable ambition, not the preserve of an intellectual élite. Now came a further revelation. Having decided on a ward round that a patient needed EMG we would advance into the cage, led by the associate professor of neurology, who would supervise the investigation. The professor himself was a renowned expert on epilepsy who took an intensely practical interest in the EEG. Not only he but any member of the staff could give a competent opinion on an EEG record. Thus the neurologists were themselves functioning clinical neurophysiologists, a matter to which I shall return.

The pleasure of new work, the generosity of colleagues, the beauty of the surroundings, and the all-pervading excitement and optimism of the Kennedy era were overwhelming, but having no intention of pursuing either neurology or neurophysiology I came home to England.

The early 1960s were heady years in London. At 30 anything seemed possible and there was ample time for exploration. I found myself drawn to the example of Dr John (later Lord) Hunt, who after an academic start at St Bartholomew's Hospital in the 1930s went on to become a flourishing and respected general practitioner in an elegant quarter of London. As an assistant in several West End practices I found an exciting life replete with extraordinary characters and situations, but was still too unsure of what I wanted to accept a permanent position.

Turning again to neurology I was reminded that it was still in London a specialty requiring, even in the presence of exceptional ability and determination (of which I had neither), influential support and a succession of the "right" jobs from the moment of qualification even to secure a training post. This was less true of the increasingly attractive Newcastle of Henry Miller and of John Walton, whose offer of a research post I accepted in the hope that I might one day become his registrar.

It was a false step. Happy in my cottage on Hadrian's Wall I dreaded each day of uncongenial work in a department I certainly did not adorn. My wonderfully tolerant mentor, Alan McComas, now Chairman of Neurosciences at McMaster, sent me to Oxford to talk to Geoffrey Rushworth, the distinguished physiologist and electromyographer. He honoured me with dinner at the high table of Jesus College and firmly advised me to "learn EMG properly" (I had been studying H reflexes in Newcastle).

Under the gaze of surely the oddest couple who ever hung together on the wall of a dining room, Harold Wilson and Lawrence of Arabia, I heard the words which determined my path. Lambert at the Mayo Clinic did not reply to my letters, his usual practice he later told me, but Buchthal replied, also characteristically, by return, from Copenhagen, inviting me to "come for a week and see what's going on or for a year and work on a project".

To obtain money for this venture I went, newly married, to Aden for six months as a well paid medical officer at the BP Refinery Hospital. My ward full of Arab infants with dehydration was far from the H reflex. I took a course in colloquial Arabic (which has not gone entirely unused since) and before setting off for Copenhagen visited the Hadhramaut.

This Aden digression, undertaken for impeccable reasons, was perhaps a last trans-

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parent attempt to avoid commitment and introduce the possibility of yet another change of course.

I should like to think that this autobiographical fragment might encourage others uncertain of their way. Compromise in the choice of one's life work is the source of much unhappiness. When sitting on selection committees I do not find it difficult to understand and sympathise with those whose careers have been unorthodox or incoherent.

Arriving in Copenhagen in late 1966, just as the great monograph on the recording of sensory potentials was emerging, I sat at Buchthal's feet (and not infrequently felt his metaphorical boot) for nearly two and a half years. He taught me many things, but above all that EMG is a practical craft which has to be learnt by apprenticeship. He made us work on paid volunteers until confident of our technique, then under supervision on selected patients, discussing with him, and occasionally with Poul or Anneliese Rosenfalck, every single measurement and its significance. Buchthal never ceased to emphasise that the report, which is our only product, is a castle built on sand unless we can be sure that what we record is real and reproducible and can support the clinical and pathological inferences we draw.

Towards the end of this time I wrote to the professor of neurology at Oxford to ask whether I might become a registrar in his department and eventually practise both neurology and electromyography. He replied that this had not been found to be a successful combination and that I should choose one or the other.

In due course I found my way to Queen Square and spent five years there as registrar and senior registrar. By then unusually well trained, on paper at least, my opinion was in retrospect hardly worth having. Only experience provides whatever usefulness one has. I should like therefore to say something of what I have learned through actually practising EMG, what has emerged from and survives the daily battering in the clinic. This will not be a "state of the art" lecture, more a ramble along familiar byways.

The use of EMG

EMG can be invaluable, valuable or valueless. I have found it an interesting exercise to try and think of situations in which even an experienced neurologist should be reluctant to proceed without asking for such contribution as we can make. Foremost among these are disorders characterised by weakness without sensory loss (which we should remember also occurs in non-neurological illness, painful movement, joint disorder, tendon rupture, disuse and lack of effort).

At the beginning of life there is the floppy infant. EMG is indispensable in deciding between true weakness due to neuromuscular disease and hypotonicity from causes in other systems or other parts of the nervous system. When appointed to the Hospital for Sick

Children in 1978 I had almost no idea how to answer this question in the brief time usually available and with minimum distress to patient and parent. The literature was supremely unhelpful. I eventually found that it could be done in 10 minutes in three steps, described in my chapter in Edward Brett's book *Paediatric neurology* (Churchill Livingstone).

The clinical distinction between lower motor neuron involvement in motor neuron disease, degenerative motor root disease, multiple motor neuropathy with conduction block and even chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) can be very uncertain, and the difficulty is not confined to nerve disorder: I have more than once found polymyositis in patients given a confident diagnosis of motor neuron disease at a reputable institution. Rheumatologists, naturally, are apt to make the reverse error.

Benign fasciculation causes much alarm and despondency, particularly among the medically aware. A senior registrar in psychiatry regarded my investigation as superfluous and was already putting his affairs in order. I have known only one exception to Buchthal's dictum that if a patient goes to the doctor because of fasciculation alone it is always benign.

The commonest difficulty, in my experience, lies in differentiating motor neuron disease from chronic degenerative root disease occurring at both cervical and lumbosacral levels. It is sometimes impossible to be sure, although a clue resides in the fact that in such patients EMG abnormality tends to be greater in relation to disability than when motor neuron disease is present. A disproportionate reduction in the density of the interference pattern in relation to strength is a subtle clue to upper motor neuron involvement.

It was an early surprise to me that patients with patchy weakness and wasting from polio many years before should show striking abnormality in strong muscles. The premature decline and death of anterior horn cells, resulting in the "post-polio syndrome", is not the only possible explanation of new weakness in a patient long used to his disability. Here, as in other conditions such as multiple sclerosis, syringomyelia and old stroke, we can be instrumental in identifying a new peripheral lesion as the cause of symptoms too readily ascribed to the underlying disorder.

At all ages the distinction between normality and a mild myopathy can be difficult, electromyographically as well as clinically. Here the critical inspection of the motor unit potential afforded by low frequency attenuation is of particular value. An exhausting example of the genre is what I think of as "the middle aged aching woman", an everyday problem to which too little attention has been paid: the unhappy sufferer not infrequently despairs of orthodox medicine. Perhaps the best the electromyographer can do is to consider polymyalgia rheumatica, in which careful searching often discloses a mild myopathic abnormality, and help to exclude polymyositis.

The condition popularly referred to as "ME" is another such problem. Occasionally,

especially in older children and adolescents, I find a mild stable increase in fibre density which, though quite insufficient to explain physical collapse, results in smiles, even tears of gratitude that "at least something has been found". What the finding signifies I have no idea. Others have reported increased jitter in this condition.

Discordance between EMG and histological findings is a subject of considerable interest in the paediatric field. At first unsettling, it can be turned to profitable discussion.

The value of EMG in disorders of neuromuscular transmission needs no emphasis. I have more than once picked up myasthenia gravis by chance when the amplitude of an evoked muscle action potential has fallen during routine motor conduction study. The condition has to be rather obvious for hand muscles to show a decrement, and Erb's point stimulation is painful. Unless the Lambert-Eaton syndrome is being considered I usually proceed directly to sampling the extensor digitorum communis, using low frequency attenuation.

A protracted search for increased jitter in this muscle is rarely necessary if myasthenia is present, and it is pleasing to show unequivocal abnormality when the physician has adduced a negative antibody test as evidence against the diagnosis. A remarkable case was that of a tired female anaesthetist treating herself with anticholinesterase inhibitors for myasthenia she did not have. Increased jitter disappeared when the drug was withdrawn.

EMG is invaluable in the study of nerve injury when it detects axon survival in the presence of total paralysis, or regeneration after neuronotmesis and suture or graft. By revealing the proportions of neurapraxia and axonotmesis EMG provides the only reliable prognosis after acute nerve compression or traction. In injuries to the brachial plexus a great deal can be discovered by simple means, and it is worth emphasising the usefulness in all nerve injuries of simply stimulating a nerve or plexus and seeing what moves.

Determination of the pathological type of a polyneuropathy, quantification and the serial assessment of natural course or response to treatment are highly important parts of our work. The same considerations apply to individual neuropathies, when only meticulous attention to technique and critical interpretation are good enough. The grossest errors are occasionally perpetrated: we have all seen ulnar nerve transposition for intrinsic muscle hand wasting due to motor neuron disease.

There are many conditions in which EMG is not invaluable to the good clinician but useful in confirming the diagnosis and establishing a baseline. I turn now to general matters.

Patients

Without patients we should have no living, so even at this mundane level we should treat them better than we often do. It is my conviction that EMG should be a consultation, not just a procedure, and for this reason I prefer

not to delegate any part of the investigation to a technician. We can become better than some neurologists at diagnosing peripheral disorders even without EMG, so many do we see. We are well placed to give advice which others have forgotten to give, such as to a diabetic patient with peripheral neuropathy not to sit with legs crossed or lean on bent elbows.

It is important to give patients time to say what they have to about their complaints before starting the investigation. This is sometimes a new experience which immediately predisposes the patient in your favour, improving cooperation and making the investigation easier.

The investigation is unavoidably painful to some degree however skilful we are, and we should lean over backwards to compensate for this by warmth of welcome, by making sure that he or she (or the parents if it is an infant) understands the purpose of it all, and by explanation and reassurance in advance of each step. A patient's tolerance can be likened to a credit account at the bank: each painful stimulus is a cheque drawn on the account and when the balance is zero cooperation effectively ceases. Some have large accounts to start with, others small, but the tolerance of all is finite and must be skilfully used.

In the *BMJ* of 22 May 1993 there appeared the cautionary tale of a consultant physician who had undergone EMG for recent acute general weakness. "The electromyogram was the worst hour of my life because the operators muttered among themselves and would not talk to me. A rude and clumsy intervention by the head of the department did not help much". Some days later: "I had to return and submit to two hours of single fibre electromyography, at the end of which I was told that I certainly did have motor neuron disease and that I should go away and enjoy myself while I could". Eventually a colleague tried him on pyridostigmine with a "magical result": he had myasthenia gravis. Even if this account is less than fair to those concerned, that any patient should be left with such feelings is a matter for reproach and a reminder to us all.

Referrals

I have no solution to the problem which besets us all, that of the variable quality of referral. The demand for EMG is inversely proportional to neurological understanding, and many patients need never have been referred if a good history had been taken and a competent physical examination performed. I have a few simple rules.

(1) The only patients who can be referred directly by the general practitioner are those with suspected carpal tunnel syndrome. This saves the patient a good deal of time and discomfort.

(2) The traditional referral form is almost always unhelpful. I ask to receive a copy of the letter the referring doctor is sending to the general practitioner.

(3) When the referral form or letter displays greater than usual confusion I ask for appoint-

ments to be made for the patient to see a consultant neurologist colleague interested in peripheral disorders first, then me later on the same morning if he considers EMG indicated.

(4) I politely return pointless referrals, notably for pain without physical signs (for example, compressive radiculopathies, backache and "sports injuries") but also for conditions such as meralgia paraesthetica and Morton's metatarsalgia. One may of course take the view—it is a matter of time and inclination—that referrals should be accepted irrespective of nature and source so as to prevent occasional gross diagnostic error and unnecessary surgery.

(5) I try to discuss with the referring doctor all non-neurological inpatient referrals and all requests, whatever their source, for a repeat investigation during the same admission. A doctor helpless or reluctant to admit the possibility of error may believe that what you have not found the first time you are bound to find the second.

(6) When I call for an inpatient I not infrequently find that he or she is eating, sleeping, in the bath, having another investigation, has gone home or is simply lost. I indicate to the houseman that if the patient does not come when called he will not be seen that day.

(7) I ask to receive the inpatient summary when an EMG has been performed. (It is one thing to ask...)

(8) I keep the referral form or letter and a written copy of the report in a docket for ever. Patients seen by me 20 years earlier, used to their outpatient notes being lost, are surprised when the first report is in my hand within a minute of asking for it. I explain that this is possible only because I do not use a computer.

(9) Whatever the source of the referral I want to be posed a question, asked to solve a problem, not told to investigate certain nerves and muscles. This tendency must be nipped in the bud immediately.

Normal values

The importance of normal values was put in a nutshell by Buchthal: "The diagnostic value of findings from motor unit potentials in neuromuscular disease depends upon the probability of obtaining the same findings in normal muscle."

Nevertheless, having done my share of establishing normal values when working on the ulnar nerve in Copenhagen, I have found that slavish adherence to a fixed system can sometimes stand in the way of intelligent and clinically productive use of results. An important part of teaching, often neglected, is concerned with the proper use of normal values. The subject can be effectively discussed only by reference to individual cases, but perhaps one can give two pieces of advice:

(1) Try to think in terms of what you expect to record in a given patient in relation to age, build and occupation.

(2) Ask yourself whether a seeming abnormality is: (a) relevant to symptoms and (b) sufficient to explain them.

Unless we are the initiators of a new technique few of us, I suspect, actually compile a comprehensive set of normal values over the whole range of our activities. Instead we use what seem to be established normal values as a template, gradually modifying them according to our experience.

"Criteria"

Instigators of research are prone to lay down criteria for diagnosis. At first it seems an irreproachable idea: to ensure agreement by imposing order on the unruly and disorganised behaviour of colleagues. Only later, in practice, does the crystalline purity of the concept begin to cloud over.

I have survived two attempts to make me obey sets of criteria. The first was in relation to CIDP, the second to motor neuron disease (emanating, appropriately, from El Escorial). They presupposed that we operate in a clinical vacuum, as if any patient might have any condition, whereas in everyday life it is unusual for there to be more than two principal possibilities. These can often be immediately distinguished by a single characteristic abnormality regardless of whether or not other criteria are being satisfied.

One danger is of missing patients who have the disease but not the criteria; another is of defining a disease by means of the criteria laid down. It is not many years since I was told that if sural potentials were small the diagnosis could not be motor neuron disease.

A less serious example is that of the carpal tunnel syndrome. Once a certain median nerve sensory latency has been exceeded the diagnosis is held to be established, and much time and ingenuity has been devoted to the detection of smaller and smaller degrees of sensory delay in the belief that this makes the diagnosis more and more accurate. Yet symptoms may have nothing to do with nerve compression and sensory delay is not infrequently found in symptomless subjects.

Equipment

I am devoted to the Medelec MS6, the equivalent in long and reliable service of the Morris Minor car and the Dakota aircraft. Modern refinements such as calculation of jitter or potential area are not available, but I believe that nothing essential to diagnosis is lacking if there is a trigger and delay line and the amplifier is capable of low frequency attenuation.

This is not to decry modern technical advances, only to warn that improvements in diagnostic effectiveness do not invariably follow in their train. It can be instructive, and depressing, to compare sensory potentials recorded 30 years ago by Gilliatt and Buchthal with those sometimes presented for discussion today. An expert photographer can produce exhibition pictures with a Box Brownie.

Surface recording electrodes and ground electrodes are made for me by Reginald George, a retired Guy's physicist. As to needle electrodes, although I am not aware of having

done harm with the autoclaveable variety (or even those we used to boil in the DISA “kettle”) I welcome and now use exclusively the disposable sort made by Medelec. Whenever possible I use the small diameter “facial” size as being kinder.

Ahead of us lie the so-called “expert systems”. Some features, such as reminders of anatomy and the recall of rare combinations of findings and references to the literature seem to me admirable, but if I am to be the patient I shall want my data presented to an expert system by an expert, not by a neophyte relying on it to make good his deficiencies.

Research

To be involved in research is seen *ipso facto* as meritorious, although not by all: Jowett, the famous 19th century Master of Balliol regarded research as “a mere excuse for idleness”. You will indignantly repudiate this but one sees what he means. Research is not invariably preceded by hard or creative thinking. It may repeat unnecessarily what has been done before or may concern itself with matters so trifling that the researchers themselves run the risk of death from boredom. Its educational value may be negligible if inadequately supervised. It not infrequently results in what Kingsley Amis called “pseudo-solutions to non-problems”. If you doubt this, read through the abstracts of any large international meeting.

As far as EMG is concerned, good technical research now emanates from very few centres worldwide, so complex and dependent on expensive equipment has it become and so familiar with disciplines such as biophysics and computer programming do its participants have to be—as well as competent electromyographers. The subject received its early impetus from work in Copenhagen, Stockholm, Queen Square, and the Mayo Clinic but now Erik Stålberg in Uppsala leads the forward technological thrust.

It was after an inspiring month with him in 1973 that I lighted, more or less by chance, on the technique of low frequency attenuation of motor unit potentials picked up with conventional needle electrodes. I have found it invaluable ever since and occasionally meet people who agree.

A physician such as myself trained to use basic equipment to complement clinical examination can still do useful research, but it needs experience to know what questions still await—and need—answers. Were I starting my career again my first priority would be to devise a foolproof method of follow up, so that I did not depend on others’ good nature or good memory to find out what befell those I had examined.

An area of research which is having a direct impact on EMG is that of molecular genetics. As knowledge of chromosomal abnormality in specific disorders advances, the formerly crucial importance of EMG in such conditions as dystrophia myotonica, Duchenne dystrophy,

and HMSN type I diminishes and will perhaps one day disappear.

Audit

It is politically correct to pay lip service to this current expensive fad. Its object, to make good practice common practice, is unachievable in EMG in the present climate for reasons which are not difficult to discern and which I shall shortly come to. The efficient organisation of our time is another matter, and has been gravely affected by new managerial activity. We shall not go far wrong, even in the face of this vexatious onslaught, if we try to act according to an instruction to staff I once spied in a French hotel: “Always remember that clients are the point of our work; they do not interrupt it”.

Training and the future

I have already said something of the rewarding experience of studying under someone brought up in the rigorous tradition of Germanic academic discipline. The contrast on returning to England in 1969 was alarming. Would-be electromyographers groped their way through investigations, apprehensive of drawing attention to what they did not understand and teaching each other in a truly apostolic succession of errors.

The result is that there are still people practising who are largely self-taught or taught by those who were. For this and other reasons we are a strange, heterogeneous collection of people with far from uniform training and experience and a professional status which leaves a good deal to be desired.

Consider only that the amount of EMG work done by rheumatologists is increasing and that applicants for neurological posts are liable to be told that they will be expected to do EMG or EEG as part of their job—as if no training were required. As further evidence of this trend, which may have its origin in the amused condescension shown by neurologists when clinical neurophysiology consisted mainly of EEG, I am asked to show young research fellows with no EMG experience how to do nerve conduction studies so that they may investigate patients with CIDP, a notoriously demanding situation. The belief that a few lessons are enough to equip any normally intelligent person is oddly prevalent. It is mistaken.

Poor recruitment is the problem. If not enough people come forward for training there will be insufficient competition for consultant posts. I have sat on consultant selection committees which reluctantly appoint the one barely trained applicant because of pressure from a hospital administrator to provide someone to do the work. The high probability of becoming a consultant after a relatively short period of training is attractive to weak recruits unable or unwilling to enter more competitive fields. The status of the specialty falls and with it the prospect of attracting better entrants.

The ideal trainee has been a neurology registrar for two years, is attracted by scientific work and embarks on at least two years of full time training in clinical neurophysiology with a view to becoming a consultant in the subject. Will there ever be enough such people? If not, what is the answer?

Major centres require academic clinical neurophysiologists, but is there any reason in principle why others should not pursue their particular interest? Twenty years ago I had the privilege of electing to practise EMG alone. Others have felt the same about EEG. The

neurologists at Stanford had become expert in one branch of the subject and I suspect that their clinical practice was the better for it. We could do the same: a year of full time training in the chosen branch should suffice. Two such neurologists would in many hospitals make a full time clinical neurophysiologist unnecessary. It is even possible that this would lead to an increase in recruitment at the registrar level.

I suggest that our system is in danger of breaking down, and that we need to reconsider the whole question of the delivery of clinical neurophysiology services in this country.

NEUROLOGICAL STAMP

Rudolph Ludwig Carl Virchow (1821–1902)

The introduction of the compound microscope and the discovery of the cellular structure of tissues attracted Virchow to pathology. In *Die cellular-pathologie*, published in 1858, he formulated two propositions—namely, that every cell is derived from a pre-existing cell (*Omnis Cellula e Cellula*) and that cells are the “seat” of disease. In this book he destroyed the old fashioned “humoral” pathology. Virchow demonstrated that cells reproduce by division of the nucleus and cytoplasm.

His was the first description of leukaemia. He established the true nature of thrombosis and embolism and he was the first to recognise cerebral and pulmonary embolism and the nature of arterial plugs in malignant endocarditis. He attributed the condition to parasites. Other papers on neuropathology include studies on encephalomalacia resulting from thrombosis and embolism, cerebral haemorrhage, meningitis, congenital anomalies, and pachymeningitis haemorrhagica interna. He wrote on tumours of the spinal cord, gave one of the earliest descriptions of platybasia, discovered amyloid, myelin, neuroglia, and demonstrated the perivascular spaces of the nervous system.

Virchow had little time for the emerging germ theory of disease and he was also dissatisfied with the theory of evolution, which he tried to have banned from school curricula.

He became interested in archaeology and accompanied his friend Dr Heinrich Schliemann to Troy in 1859 and wrote an account of the discoveries there. In politics he was a member of the Reichstag from 1880 to 1893 and as a leading liberal was a bitter opponent of Bismarck who went so far as to challenge him to a duel in 1865. He had much to do with securing a good sewerage system and

water supply for Berlin. He was also widely known for founding, in 1847, the *Journal Archiv Für Pathologische Anatomie*, which he continued to edit for 50 years. He is shown here on a stamp issued by Germany (West Berlin) in 1952 (Stanley Gibbons B96, Scott GN89).

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