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Controversies in otology
(The first Sir Morell Mackenzie Lecture)

by

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I have long considered it unfortunate that there has not been a 'Mackenzie Lecture'. I was very pleased to hear that this omission was to be corrected and extremely honoured at being invited to give the first Sir Morell Mackenzie Lecture. It is particularly gratifying that the contributions of Mackenzie are going to be remembered in this way, especially with the recent closure of the Golden Square Hospital which he founded.

Sir Morell Mackenzie

Let me begin by referring briefly to Sir Morell Mackenzie's achievements. He has been called the Father of British Laryngology and certainly deserves the title. He was the first English laryngologist. In 1865, without a lot of encouragement, he founded the first hospital in the world for diseases of the throat. When Mackenzie first raised the idea of a specialist throat hospital, Sir James Paget suggested that a hospital for diseases of the great toe would be equally appropriate! With Norris Wolfenden he founded *The Journal of Laryngology*. He was the author of the first standard textbook on diseases of the throat and he was the first protagonist of a scientific study of diseases of the throat among English speaking doctors.

As an otologist, I found the reading of Mackenzie's *'Manual of Diseases of the Throat and Nose'* quite fascinating.

I was particularly impressed with this illustration (Fig. 1) from his book although I suppose it really demonstrates my own laryngological ignorance. I can still recall the first time that I came across a cuffed tube in 1963, when I started my ENT career. At that time I was most impressed by this recent advance in medicine — how wrong I was.

Undoubtedly, Mackenzie (Fig. 2) was a great man but he had two misfortunes. The first one plagues most of us; by and large we are spared the second.

His first misfortune was the desire to become famous. Scott Stevenson (1946), in

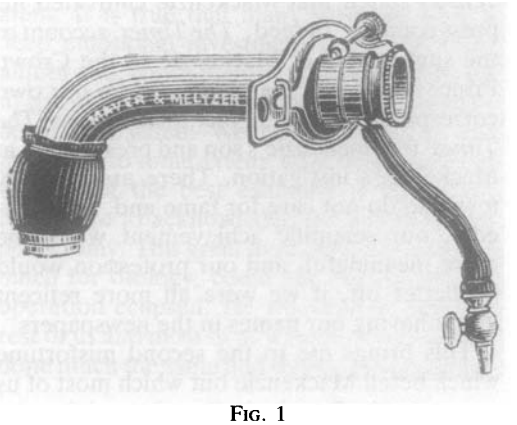


FIG. 1
Cuffed tracheostomy tube.
Reproduced from 'A Manual of Diseases of the Throat and Nose', Ed. Morell Mackenzie, J & A Churchill, London, 1880.

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FIG. 2
Sir Morell Mackenzie (1837–92).
Reproduced from 'A History of Oto-Laryngology', Eds. R. S. Stevenson and D. Guthrie, Livingstone, Edinburgh, 1949.

his book on Morell Mackenzie, refers to what he called 'Mackenzie's harmless liking for seeing his name in the newspapers'. Scott Stevenson stated that Mackenzie cultivated his press contacts. Indeed, *'The Times'* account of the summoning of Mackenzie to the Crown Prince which had the by-line 'From our own correspondent' was, in fact, provided to *'The Times'* by Mackenzie's son and presumably at Mackenzie's instigation. There are a happy few who do not care for fame and, undoubtedly, our scientific achievement would be more meaningful, and our profession would be better off, if we were all more reticent about having our names in the newspapers.

This brings me to the second misfortune which befell Mackenzie but which most of us are spared. Paradoxically, the misfortune which is worse than wanting fame, is achieving it. Undoubtedly Mackenzie must have enjoyed considerable elation on receiving the cable from Berlin and the message from Queen Victoria, but once the German physicians got to work on his reputation and his famous patient died, the publicity turned sour and it is unlikely that Mackenzie ever again had any sustained peace of mind.

Mackenzie did not practise otology and consequently the otological aspect of my title is not appropriate. However, Mackenzie was a scientific laryngologist and I am recommending for otology the principles which Mackenzie practised in his management of the Crown Prince.

The illness of the Crown Prince

I wish to underline four principles in scientific otological practice, all of which were important to Mackenzie and before considering the ear I wish to look at Mackenzie's management of the Crown Prince in the light of the four principles.

First is the *pathology*. Mackenzie had considerable experience and skill in removing tissue from the larynx in the conscious patient holding a laryngeal mirror in one hand and forceps in the other. It is doubtful if any of the physicians who had been caring for the Crown Prince had this ability. Consequently, not only did they not try to obtain biopsy material, they scoffed at the very idea of it. However, Mackenzie was adamant that the histology must be known and, although he removed tissue on three occasions, at no time did the great pathologist Virchow see any malignant cells. But Mackenzie was right. Before embarking on any treatment, we must try to establish the pathology.

Secondly, Mackenzie paid attention to the *prognosis* or the natural history of the untreated disease. One hundred years later there are many who still do not ask themselves the question, 'What happens if we do nothing?'. In his book on Frederick the Noble, Mackenzie puts forward the claim that, by keeping the surgeons away from the Crown Prince, he probably prolonged his life.

This brings us to the third principle which is the *proficiency* of any treatment that is proposed. The German surgeons were proposing

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an external operation and in his book Mackenzie looked at the three options and their success rates. Up to that time the laryngofissure had been reported in 22 cases with 2 cures—a 10 per cent success rate. Partial laryngectomy had been successful in one cure out of 35 cases—3 per cent success. Total laryngectomy had been successful in curing 8 out of 138 cases—6 per cent success.

Mackenzie points out that there may well have been many failures which had not been reported but stated that it is unlikely that there had been any unreported successes. Little has changed! He stated that these are the best possibilities. They may have been much worse. He also showed his scientific approach, well ahead of his time, in that he commented on the absence of untreated cases or controls.

Finally he looked at the *price* paid by the patient. I think it is important that we do the same, thinking not only of the financial cost to the patient and the National Health Service but the risks and potential complications which any procedure or treatment carries.

In laryngo-fissure, in his survey of the published results, Mackenzie reported an immediate mortality of over 25 per cent, delayed mortality in a further 10 per cent, and recurrence and, as he presumed, acceleration of the disease in the remaining 55 per cent.

Partial laryngectomy was no better with an immediate mortality of just under 25 per cent, delayed in a further 20 per cent, and recurrence with acceleration of the disease in those who were not cured.

Total laryngectomy was even worse with an immediate mortality of 35 per cent. In addition, if the patient survived the operation his lot was usually pitiable.

Otology

Mackenzie attempted to do for diseases of the throat what Wilde and Toynbee attempted for the ear—set it on a sound scientific basis. This brings me back to my title.

I suppose that the most obvious comment to make about controversy is that it is unlikely that any form of treatment is free from it.

However, so long as we do not lose our heads and take up untenable positions, controversy is not necessarily a bad thing.

Mackenzie, Wilde and Toynbee all laboured, in the words of Wilde, 'To divest this branch of medicine of that shroud of quackery with which it has become encompassed'.

Well, how far have we come?

The fourth principle referred to above can be a difficult exercise. We are often guided by the published results of others and herein lies a problem. Although there is very little deliberate dishonesty there is often inaccuracy, for example, due to imprecise case selection or a so-called 10-year follow-up meaning that one patient in the series has been followed-up for 10 years. However, three specific problems can be identified.

The first of these is one of those problems which one attributes to politicians. While *they* are changing their minds on something, they resolutely insist that they believe firmly what they have always believed and then, without signalling, suddenly, make a U-turn. This is not acceptable in medicine. If one is having second thoughts about a particular treatment surely one ought to say so sooner rather than later.

The second is the natural reluctance to publish a paper retracting an earlier one, or similarly to state that a technique has been abandoned and why.

The third problem is emotional involvement. It is true that many surgeons have a deep emotional investment in certain techniques and, consequently, they find it difficult to acknowledge that their technique may have been superseded. We have all seen it and probably, to some extent, all of us are guilty of it. One of the best examples is Lempert's reluctance to acknowledge the merits of stapedectomy. This great man, who had done so much for otology, could not bear to see his operation eclipsed. He was human, like the rest of us and most of us would probably have done much the same had we been in his shoes.

Cochlear otosclerosis

I wish first to say something about cochlear otosclerosis, not only because of the condition

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itself but also because of the type of situation which it represents. There are other otological conditions, eg, autoimmune deafness and some aspects of perilymph fistulae, which fall into the same category. Essentially what happens is that a disease entity is built around something which, of itself, is very dubious. However, if this dubious foundation is overlooked, patterns are established which result in self-perpetuation of a myth. Now, first of all, let me say what is understood by cochlear otosclerosis. It is otosclerosis causing sensorineural deafness without any associated conductive loss. This condition has been, and is being, widely diagnosed.

Almost twenty years ago Shambaugh (1966) laid down five specific diagnostic criteria. Cochlear otosclerosis may be said to occur when bilateral symmetrical sensorineural hearing loss is present with stapes fixation on only one side. Secondly, it may be present when there is progressive sensorineural deafness in patients with a family history of stapedial otosclerosis. A Schwartze's sign may suggest the diagnosis in someone with a pure sensorineural hearing loss. Fourthly, progressive sensorineural hearing loss coming on insidiously in early or mid-adult life may lead to the diagnosis. Finally, progressive loss with certain audiometric configurations are suggestive.

Later, polytomography of the labyrinth was called in to help diagnose the condition. It was said that this condition could be diagnosed radiologically, despite the fact that there is a wide range of normal bone density of polytomes of the labyrinth and despite the fact that otosclerosis may result in either an apparent increase or decrease in the density of labyrinthine bone. In a paper in 1970, Britton and Linthicum said, 'the subtle changes from normal in otosclerosis are perhaps more difficult to interpret than any other area of diagnostic X-ray'. But despite this, in 1972, Linthicum reported a 66 per cent incidence of radiologic evidence of otosclerosis in over 100 patients with sensorineural hearing losses greater than 40 dB. So now all that is needed to make this diagnosis is bilateral sensorineural deafness and an abnormal polytome—even though there is doubt about what constitutes a normal polytome.

In 1984, Balle and Linthicum stated that anyone with a progressive sensorineural hearing loss and a family history of otosclerosis should be suspected of having cochlear otosclerosis. And he again referred to the value of polytomes in making the diagnosis. He went on to outline the management of this condition and described how fluoride, calcium and vitamin D may help prevent further progression of the loss.

And so a disease entity, and its treatment, has been established. The treatment will be considered to be successful if the hearing loss does not progress. Consequently, anyone with a sensorineural hearing loss from any condition may now have this diagnosis since there is, in any event, a wide range of normal variation of bone density in the labyrinth. Not only that, if there is no further progression of the hearing loss, as is the case with many patients with sensorineural deafness, then the treatment can be considered to be successful. So what we have now is a common cause of sensorinerual deafness which is amenable to treatment.

But does the condition exist? When Schuknecht and Kirchner (1974) went to their temporal bone collection and looked for it they found in a collection of 734 ears, only 32 showing histological otosclerosis, i.e., otosclerosis without stapedial fixation. In these 32 ears only three involved the cochlear endosteum, which according to the proponents of this disease is the important factor. In two of these three ears there was only mild hearing loss which, in any event, could be attributed to the atrophic changes which are frequently observed in ageing individuals without otosclerosis. In the third case the atrophic changes were identical to those changes present in the other ear of the same individual which did not show an otosclerotic focus. Obviously this does not prove that the condition does not exist but it certainly shows that if it does, it is rare.

In 1978, Applebaum and Shambaugh reported three temporal bone studies in patients where otosclerotic involvement of the cochlea had been diagnosed by polytomography. In one of these the diagnosis was of pure cochlear otosclerosis and in the other

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two the patients also had a conductive deafness and, clinically, the diagnosis was clear. However, in the case diagnosed by X-ray as having cochlear otosclerosis, temporal bone sections failed to reveal any otosclerosis whatsoever. In the two cases of clinical otosclerosis, the otosclerotic lesion did not involve the cochlea and, worse still, in one of these patients the contralateral ear did indeed have otosclerosis involving the cochlea but this had not been seen on the polytome.

Once again, these findings do not prove that the condition does not exist. They may simply mean that one radiologist had misread his polytomes. However, the weight of evidence is such that, whatever else one may say about cochlear otosclerosis, it is extremely rare.

I have been unable to find in the literature any report of polytomographic evidence of cochlear otosclerosis subsequently being confirmed by histological section.

Consequently, in this condition we can go no further than the pathology. If we cannot even be sure that it exists we cannot talk in any meaningful way about the prognosis or the proficiency of treatment. It is unlikely that the fluoride which these patients are ingesting does them any harm and, psychologically, it may even do them some good. However, they would be better off without the radiation in the polytomes and the clinicians involved would be better not to be deceiving themselves that this is a common condition.

This is a remarkable situation. I have searched for a parallel in the history of medicine but could not find anything which had the same three self-propagating qualities as are evidenced in cochlear otosclerosis. First, we have a not uncommon symptom. Secondly, we have a rather non-specific investigation. Thirdly, we have the really big self-propagating factor where, having got it wrong once, we are more likely to get it wrong in the future.

Consequently, I have had to go into the realms of fantasy to get an analogy.

Septic dragon bite

I want you to consider skin lesions with a range of appearances—puncture wounds, thermal injuries and secondary infection. Let us consider that someone diagnoses these as infected dragon bites, laying down at least two criteria. There must be an infected skin lesion and, in addition, the ESR must be raised. There is a third feature of the septic dragon bite and that is, it tends to occur in dragon-infested areas. Consequently the more one diagnoses it the more likely one is to continue to diagnose it. Indeed, there is no longer any need to diagnose impetigo because each week for the past few weeks one has had a similar case and, clearly, one must be living in a dragon-infested area and therefore the most likely diagnosis is septic dragon bite.

If one writes papers on it and includes reports of cases from the next region, one will show that dragons are also present in the next region and the local chap really ought to stir himself, brush up his diagnostic acumen and start recognizing the condition.

Treatment need not be any different from the treatment of any other skin infection but that does not matter because most will respond in the same way as any other skin infection. If one throws in a little bit of mumbo-jumbo, they will still respond equally well and one's reputation will rise.

The terrible thing about this condition is that anyone who does not believe that dragons exist finds it increasingly difficult to refute the diagnosis. One of the diagnostic criteria is that it occurs in dragon-infested areas and it soon becomes likely that most areas are dragon-infested.

Just how does the sceptic set about showing that this is a non-condition? If cases are occurring only sporadically it becomes well nigh impossible for him to disprove the existence of this disease. However, if the condition really is widespread then surely it ought not to be difficult to find someone who has actually seen a dragon biting a person. Indeed, the dragon does not need to be seen biting but merely seen!

For the propagation of a non-condition all that is required is any disease entity, any non-specific test and the energy to propagate the idea. In otology we have seen it in the various aetiologies and treatments of Menière's disease and sudden deafness, and more specifically, in the propagation of ideas about

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perilymph leaks and autoimmune deafness. We also have seen arteries being elevated from nerve roots, and facial nerves decompressed or probed for facial paralysis or hemifacial spasm. We have even seen internal auditory meatuses being decompressed.

Clearly it would not be possible to deal with each of these this afternoon. Last year I had the honour of speaking at the Section of Otology on controversies in otologic surgery. Many of you were present at that time and I shall spare you repetition of what I said then and avoid the subject of surgery.

Idiopathic sudden sensorineural deafness

I want to spend the remainder of my time thinking about one other condition, idiopathic sudden sensorineural deafness, and then to make a few positive suggestions.

There must be few conditions in medicine where the management varies from physician to physician more than here. At one extreme there are the therapeutic nihilists who resolutely and stoically say to their patients that there is no treatment that will influence the condition and that they must simply await the outcome. At the other extreme are those physicians who sometimes jeopardise their patients' lives with blunderbuss therapy in the hope that at least one of the drugs will do some good.

There are many problems in this condition. The first is that idiopathic sudden deafness is, in fact, idiopathic and we don't know the pathology. Obviously there will be cases that are idiopathic when first seen but two or three days later, when the vesicles of herpes zoster appear or when a CT scan shows a cerebellopontine angle tumour, they are no longer idiopathic. However, most will remain idiopathic with the possibility that the deafness has been caused by a viral infection, a vascular lesion, a membrane rupture, an immune reaction involving the inner ear, the spread of toxins from a distant infection, malingering or hysteria.

Schuknecht *et al.* (1973) from the study of their postmortem material have suggested that viral infections are the most likely aetiological factor. However, pathological studies have tended to be limited to those patients where there was little recovery. Indeed, it is unlikely that more than a sprinkling of cases with good recovery will ever find their way into a postmortem series and, consequently, it is probable that there are other factors in the idiopathic group.

The second problem in establishing a rationale of treatment in this condition is the prognosis. There is a high incidence of spontaneous recovery. All those who advocate treatment make a special point of emphasising the importance of starting this treatment early. However, it is well known that the statistics are distorted by the inclusion of those who are going to recover without treatment or the exclusion of those who are going to fail to recover despite treatment. For example, the reported results of treatment of sudden sensorineural deafness in doctors are better than in any other group in the community. Presumably this is simply because they have such easy access to otologists that all cases find their way into the series very readily, including those that would have recovered spontaneously within 48 hours, and therefore, otherwise would not have been seen.

What are the prognostic indicators? What does happen if no treatment is given? Without treatment, over 60 per cent of all these patients will make a good recovery. When the hearing loss is only mild the majority recover, but when the degree of hearing loss is profound only about one-quarter recover.

Ascending and flat audiometric curves carry, on the whole, a much better prognosis than descending curves. This is especially so at the upper end of the scale and a falling curve between 4000 and 8000 Hz is a particularly bad prognostic indicator.

In managing these patients attention must be paid to the psychological aspects. We must be careful that we do not engender in our patients resentment or bitterness which can poison the remainder of their lives. We are especially at risk of doing this when we appear to fail to take any action to deal with their problems. Effective psychological management is particularly important when the residual handicap proves to be severe. We must beware of appearing to wash our hands

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of our patients, especially if we are not actively prescribing any medication or when our efforts have failed.

Assessing the proficiency of the treatment brings problems when we consider the forceful publicity given by some otologists for treatments for which there is little logical basis and which lacks a controlled trial. Innumerable drugs have been credited, at some time or another, with having cured idiopathic sudden deafness.

I think the confusion is best summed up in these quotations: 'Steroid therapy is potentially harmful in viral infections and there is no evidence that it is beneficial' (Nadol and Wilson, 1980).

'Steroids had a statistically significant effect on recovery of hearing in patients with moderate hearing loss. The nature of the hearing loss and its susceptibility to improvement with steroid therapy lend support to the hypothesis that viral cochleitis is the primary cause of idiopathic sudden sensorineural hearing loss' (Wilson *et al.* 1980).

You may say, 'So what? We already know that different people think different things, and that the same person thinks different things at different times'. I agree. The problem is that both these statements were made in the same year, in publications in which there was a common author. Furthermore, he was a well known physician from an internationally renowned department. Let me say in his defence that, in both papers, there were one or more other authors and pressures may have been applied to agree to what was written. I ought also to say that one of these papers was the best review of idiopathic sudden deafness that I have come across.

Unfortunately, so far, no drug has been shown in a controlled trial to provide an unequivocally improved prognosis in idiopathic sudden sensorineural hearing loss.

When we are assessing the price to be paid by the patient we have got to look at the problems caused by the condition, i.e., the handicap. The handicap in idiopathic sudden deafness varies from patient to patient. If there is no handicap, and not likely to be any, treatment is rarely indicated. I shall emphasise the obvious. A mild degree of unilateral hearing loss in a professional musician is not comparable to a similar amount of hearing loss in a 'tone deaf' bank manager. Similarly, a severe degree of hearing loss in one ear with good hearing in the other is not comparable to a severe sudden hearing loss in one ear where the other has been totally deaf for years from some unrelated cause. I even venture to suggest that there would be few therapeutic nihilists who would refrain from all treatment for themselves if, with a longstanding total deafness in one ear, they were suddenly stricken by severe deafness in the other. I feel that their reactions would be similar to those of the famous Eighteenth and Nineteenth Century atheists who suddenly became Christians on their deathbeds.

So the sacrifice required from the patient in any proposed plan of treatment ought to relate to his handicap. And what of the price? What will the treatment do to his general health? What will it do to his general welfare, bearing in mind that admission to hospital means time off work which may affect his earnings, career prospects and possibly even his continued employment?

And what are the risks? Sadly, all effective drugs appear to have major side-effects and complications. Even ineffective drugs are the same. Zaytoun *et al.* (1983) reported an unfortunate patient who was given low molecular weight dextran which led to renal failure and death. The risks must never be forgotten in considering the prescription of any drug, especially when there is some doubt about the indication. So then, how do we approach the management of idiopathic sudden deafness?

First, if there is any history of physical stress or exertion as a possible aetiological factor, bed rest is a logical treatment. In the absence of such a history there must be some doubt about the wisdom of recommending this.

I have not set aside the time to enter into a discussion on perilymph fistulae but obviously we must have this in our differential diagnosis.

Secondly, efforts to achieve vasodilatation are logical only if one accepts the possibility of vasospasm or vascular occlusion as an aetiological factor. The administration of carbon dioxide and oxygen is accepted as being without significant risk and is known to have a

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vasodilating effect. There are also many drugs which have been advocated because of their vasodilating properties.

Thirdly, there is the question of steroids. Obviously if there is the likelihood of a membrane rupture one would hesitate to prescribe steroids. However, in the absence of this, and in the absence of any general contraindication to steroids, the evidence is such that these are worthy of consideration. Controlled trials have shown differing results in those cases where steroids have been administered. Hence the man who faces two ways at once.

However, there is no doubt that in certain types of sudden deafness, for example congenital syphilitic labyrinthitis, or deafness associated with overt autoimmune disorders, a dramatic improvement in the hearing often results from the administration of steroids. There are many who believe that there is a distinct clinical entity of autoimmune deafness. Unfortunately there is a lack of unanimity on what constitutes this condition, but if it exists, one would expect improvement from steroids.

Fourthly, one must consider the administration of intravenous preparations such as low molecular weight dextran. Given sensibly, the risks of such a regimen are minimal. Nonetheless, there have been complications, including death, and I am not aware of any controlled trials which show a clear benefit from low molecular weight dextran.

Fifthly, I wish to look to the future and mention the possibility of help from acyclovir, a recently introduced antiviral agent. In a small personal series of facial paralysis and sensorineural deafness secondary to herpes zoster oticus, I have been impressed by the results of the administration of this drug (Hall and Kerr, 1985). I have not done, and do not intend to do, a controlled trial in all cases of idiopathic sudden deafness. Nonetheless, there is the possibility that some of these cases are due to herpes zoster oticus without obvious vesiculation and a case could be made for such a trial.

I make no apology for referring again to the psychological aspects of this problem. Many of these patients feel that their lives are crumbling around them, especially when their sudden deafness occcurs in an only hearing ear. It is imperative that we provide appropriate psychological support either personally or through counsellors and hearing therapists.

In the light of our present state of knowledge and practice, the management of idiopathic sudden sensorineural deafness is far from scientific. It is an art which should be practised with conviction and high motivation. We may not influence the disease process but we can influence the patient's attitude to his resulting handicap.

One shudders to think of the cost to the NHS of much of the treatment of sudden deafness without any good evidence that it helps.

Conclusions

In otology we have controversies in the management of so many conditions: cochlear otosclerosis, sudden deafness, Menière's disease, autoimmune deafness, perilymph fistula, facial paralysis and hemifacial spasm. Let me put a provocative question: Do tinnitus clinics increase the overall suffering in the community from tinnitus or do they decrease it? Paradoxically, it seems likely that by increasing the numbers of children who are taught to swim, we shall increase the number of drownings in our community. Is there a parallel with tinnitus?

I could go on. The question of cholesteatoma has not yet been resolved; many people are taking a new look at our management of glue ears and otosclerosis.

Mr Chairman, I am very much aware that I have asked more questions this afternoon than I have answered. I will, therefore, attempt to end on a positive note and make three positive suggestions as to the way forward.

First, is the subject of national co-operative prospective clinical research. The Otorhinolaryngological Research Society has, in my view, raised the standard of research in our specialty in this country. The importance of controls and statistical analyses are well ingrained, often painfully, into the minds of our younger members. However, in

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the management of those conditions where the natural history frequently results in spontaneous recovery, large numbers and tight controls are required. Both are difficult to achieve. Multicentric trials are useless if strict criteria are not laid down and rigidly adhered to. This requires not only high motivation from all engaged in the trial but also a leader who is both energetic and somewhat obsessional. Such people are around and we ought to encourage them.

Secondly, I wish to raise the question of medical audit. The autonomy of the consultant is one of the strengths of our health service but we shall lose this if we abuse it. In Northern Ireland in 1983, and again in 1985, we had otolaryngological audit meetings and we plan to continue with these every two years. Each meeting lasts one day, and, throughout the province, all ENT units are closed for that day. So far, we have had at each meeting over 90 per cent of all those practising otolaryngology, from senior house officers to consultants. Of those who did not attend, all but one were out of the country at the time of the meeting. These were organized through our Postgraduate Council and were seen as much as matters of postgraduate education as of audit. They were run by otolaryngologists for otolaryngologists. We looked only at the management of common conditions and, by and large, we were honest with ourselves and with each other. In any event, our junior staff were there and they knew when we were being dishonest. These were very instructive, and often humbling occasions, and I put it to you that each region, or better still, each subregion ought to have such meetings.

We did not solve many controversies in otology but we have set a course for ourselves whereby we shall look at the rationale of all that we are doing. The object is not to standardize our treatments but to ensure that they are rational. Sadly, the pressures under which we work make it difficult to assess statistically the results of what we are doing. However, it is a very worthwhile exercise to be called upon to justify to our immediate colleagues just how we are using the time, money and other resources of the National Health Service.

The truth of course is that we do not practise as a national group, nor do we practise as a regional or even a sub-regional group. We practise as individuals. My third suggestion is simply a reiteration for our personal practice of the principles practised by Mackenzie. In our management of patients we must think of the pathology of the condition, the prognosis if it is untreated, the proficiency of any treatment which we prescribe and the price paid by the patient and the community at large.

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