His tonsils were greatly enlarged, partly obstructing the upper airway; cervical nodes were also enlarged. After adenotonsillectomy he dramatically improved; his snoring stopped, night sleep was peaceful, and he became lively and active during the day and did not need his afternoon nap. Within two months both his height and weight had increased. He gained 1.5 kg, crossing from below the third centile to between the third and 10th centiles (fig 2).

Discussion

The disappearance of noisy breathing, snoring, and daytime somnolence and the dramatic increase in weight and height after operation show the degree to which overall wellbeing is affected by partial and intermittent upper airways obstruction, leaving no doubt about the wisdom of adenotonsillectomy, an uncommon operation in this age group. Despite the severity of the symptoms seen in these cases, however, and the frequency with which both failure to thrive and upper respiratory infections are found in young children tonsillar enlargement is rarely diagnosed as a cause of poor

weight gain or growth in Britain,³ although it is more commonly noted in the United States.⁴ Lack of clinical awareness and a reluctance to subject young children to tonsillectomy could account for this apparent difference and partly explain the late diagnosis in case 2.

These two cases show clearly how enlargement of the tonsils may cause failure to thrive; this is rapidly reversed after adenotonsillectomy.

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Portraits from Memory

19—Dr John Smith (1892-1976)

JAMES HOWIE

To himself John Smith was, and remained, just plain John Smith. To the many who profited from his incredible industry and

penetrating insights into the problems of microbiology and infectious diseases he was a centre of excellence in himself, though all his work was done in the modest laboratory of a small fever hospital. In 1919, when he gained his DPH, Professor Matthew Hay of Aberdeen University had him appointed as assistant medical officer of health with special duties in connection with the laboratory diagnosis of infectious diseases—part time laboratory work—with the rest of

his time being devoted to clinical tuberculosis. The DPH course of those days included much good practical bacteriology, but Smith literally taught himself all the extras that his work required him to master. The laboratory was opened in 1920 and soon afterwards John Smith was made its director but he never gave up his interest in clinical problems and kept a close watch on all new admissions to the hospital. It was not long before the usefulness of his service spread outwards and attracted visits not only from general practitioners in the city and

county of Aberdeen but also from consultants at the Aberdeen Royal Infirmary, which, until 1938, was not served by the university department of bacteriology.

Plain but not obvious

In addition to teaching himself bacteriology, John Smith gave great thought to developing a really useful postal service with many exceptional features to overcome the inherent disadvantages of any such system. In the event Smith provided bacteriological services for the city of Aberdeen and for the counties of Banff, Kincardine, Nairn, Elgin and Moray, and—from 1934—also for the county of Aberdeen. His laboratory was never without visitors seeking his advice as well as an account and interpretation of his laboratory findings. In 1938, when the university department of bacteriology at last became responsible for the Royal Infirmary, I made a point of visiting John Smith specifically for advice on the technical problems arising from the laboratory diagnosis of leptospirosis. At that time he had recently diagnosed it as a problem among the fish workers at Aberdeen. John Smith and Stanley Davidson together worked out how the infection was acquired and spread, and how to prevent it. Thanks entirely to Davidson's importunity a joint paper was published, despite Smith's protestations that it was "all plain and must be obvious to everyone."

My efforts at setting up reliable laboratory diagnostic methods for leptospirosis ran into so many technical problems that I had no hesitation in seeking John Smith's help. He was delighted to reveal all the necessary tricks of the trade to a junior member of the rival establishment and indeed was so helpful and friendly that I never hesitated to seek his help with other problems. The help was always freely given. He had met and solved all problems with the leptospirae, and took it for granted that the answers must surely be obvious to me. They were not. At that time a live culture was used as

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antigen. But to grow it we needed rabbit serum in a mixed saline medium. But, as Smith pointed out to me, the rabbit serum would be inhibitory unless we got a right and a young rabbit; and, in due course, as it grew, its serum also would become inhibitory and we'd need to find another suitable rabbit. The pH of the medium was thought to be critical. So it was, but not so critical as the technicians trying to get it just right might suppose. They would be liable to go on adding more and more of the two buffer solutions trying to get the pH exactly right. As a result the excessive salinity of the medium itself would often inhibit growth. It was all very obvious once it was pointed out; but not until then.

During our discussion I mentioned to Smith that I had never managed to culture leptospires from the urine. The reason was obvious to Smith. There might well be leptospires in the urine but there was also antibody; and on dilution the antibody to leptospires became lytic, did it not? Of course. None of this was published anywhere, and I said to Smith that he would be doing a service if he made all these points plain in a paper.

"Not at all," he said, "it's all too obvious. You make a paper of it if you like!"

I did not like, and later developments have made it unnecessary to use live cultures for diagnostic antigen. But that was Smith all over—what he had worked out and what had become obvious to him must be plain to all. He did excellent work on brucellosis and on infantile gastroenteritis. In 1942 I sent him serum from a patient in Nigeria thinking that it might provide the first evidence that leptospirosis was actually present in that country—as it was then supposed not to be. The test was positive; but Smith thought it so obvious that leptospirosis must be present in Nigeria that he left that discovery to be later reported by others.

A hint not taken

Smith's reputation was not merely a local one. His work with Kinloch and Stephen on the method of transmission of streptococcal

puerperal infection was so convincing, thanks to Smith's refined typing methods, that it gained acknowledgment from the Colebrooks and the Medical Research Council. An interesting addendum to the wider recognition of Smith's contribution to streptococcus epidemiology was conveyed to me when I became medical director of the Public Health Laboratory Service (PHLS) in 1963.

In 1945, when the PHLS Reference Laboratories were being set up, it was suggested that it would be good to have some of them outside Colindale in north London. The streptococcus reference laboratory, it was thought, might usefully go to Scotland. There were many good reasons for this, one of them being the hope that those responsible in Scotland might see the offer as a step towards making sure that epidemiological studies of infection were not hindered by the border, which microbes had a habit of ignoring.

The proposition was tactfully put. In recognition of "the distinguished contribution of Scottish bacteriologists to streptococcal epidemiology" it would seem to be appropriate to locate the national streptococcus reference laboratory in Scotland. All the expenses would be met from the PHLS budget. Two questions were asked: Was the proposal acceptable to Scotland? And where would the laboratory be most appropriately located? The hoped for and obvious answer to the second question was John Smith's laboratory in Aberdeen City Hospital. But the English PHLS proposers had enough tact and common sense not to suggest that answer. The answer given by Scotland's advisers to the first question was that the proposal was welcome. But the answer to the second question was that four streptococcus reference laboratories would be required in Scotland—one in each of the then four university centres. So the PHLS streptococcus reference laboratory simply went to Colindale.

I was often tempted to tell John Smith about this abortive proposition, of which he would not have heard. But I never did. I knew that his comment would have been: "Just so; just so. That answer was surely obvious, wasn't it?" He knew more right answers than he ever let on about. In the end, he was given an honorary LLD by Aberdeen University; and other honours were also laid on his head, to his pleasure and to his great and genuine surprise.

What type and dose of aspirin is advised for the treatment of transient cerebral ischaemia?

The optimal dose of aspirin when used as an antiplatelet agent remains unclear. The *British National Formulary* states that a suitable dose is 75 mg daily,¹ others suggest that 1-25 mg daily may be optimal,² whereas 300 mg is common. It is, however, generally agreed that a minimum effective dose is used to keep side effects to a minimum but more important that higher doses might offset the desired inhibition of thromboxane A₂ synthesis by an inhibitory effect on vascular prostacyclin synthesis.² Prostacyclin itself inhibits platelet aggregation. The dissolution and bioavailability of enteric coated aspirin are claimed to have improved since the introduction of the cellulose coating.³ Compared with uncoated tablets, at equivalent doses, coated tablets have been shown to achieve similar peak plasma concentrations,⁴ provided that they are taken on an empty stomach.⁵ By this comparison and assuming that the previous views are correct then theoretically a 150 mg enteric coated tablet should achieve therapeutic plasma concentrations for use in transient cerebral ischaemia at steady state.^{3,4} The practical problem is that, to my knowledge, a 150 mg tablet is not available and halving an enteric coated tablet cannot be recommended. The alternative, if aspirin is to be used, is a soluble uncoated tablet, which if taken with food may help to reduce the gastrointestinal side effects. A 75 mg soluble tablet is available on prescription and is in common use for the purpose, but, like the enteric coated tablet, it has no licence for this indication.—JOHN HAMPTON, pharmacist, London.

Can infections caused by legionellas other than *L pneumophila* be diagnosed using *L pneumophila* as antigen? How common or rare are infections caused by other legionellas?

The serological diagnosis of infections caused by legionellas other than *L pneumophila* using *L pneumophila* as the antigen would require, firstly, the presence of one or more antigens common to species with the family *Legionellaceae*; secondly, that these react in the serological test used; thirdly, that the antibody response in legionellosis be directed at least in part against the common antigens; and, finally, that these antigens be specific to the *Legionellaceae*. Although members of the family do share some antigens, using Western blotting techniques we have been unable to show antibodies to such components in serum from many patients with legionellosis. We believe, therefore, that there is no evidence to support the view that infections caused by legionellas other than *L pneumophila* can be reliably diagnosed by using *L pneumophila* as the antigen. In laboratories in England and Wales the serological diagnosis of legionellosis is most commonly established by using antigens prepared from strains of *L pneumophila* serogroup 1 in an indirect fluorescence antibody or a microagglutination test, or both. Whereas these tests have been shown to be both sensitive and specific¹ there are no well evaluated serological tests for diagnosing legionellosis caused by legionellas other than *L pneumophila* serogroup 1, and hence such cases can be reliably diagnosed only by direct demonstration or in isolation of the causative organism from respiratory tract specimens or other clinical material. In the years 1979-86 the Communicable Disease Surveillance Centre, Colindale, received reports of 100 cases of legionnaires' disease proved by culture. Of these, only 13 were due to strains of serogroups other than *L pneumophila* serogroup 1 and only one to a species other than *L pneumophila*. Although most cases are caused by *L pneumophila* serogroup 1, it is not possible at present to assess reliably the clinical importance of other serogroups and species.—A G TAYLOR, head of Public Health Laboratory Service Legionella Reference Unit, London.

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