Medico-political matters could occupy much space here, as they occupied much time at the Federal Council and are occupying much time and energy within the Association and its Federal and State Secretariats. They are in two main areas—health planning and medical fees—and Federal Council gave its careful attention to both. Undoubtedly the most important exercise here is to keep the two matters in a right perspective. The fee issue is prominent and has some disturbing aspects, but it is the lesser issue of the two. It certainly suits the Minister for Social Security, Mr Bill Hayden, and his Government to keep fees in the spotlight, for not only does this leave relatively in the shadows the Labor Party’s policy in relation to health services and the report of the Health Insurance Planning Committee, but also the controversy can be manipulated in an effort to discredit the medical profession and so the A.M.A.’s criticism of the Government’s health plans. It is of the greatest importance that these facts and the implications of the Government’s policy be kept clearly before the Australian public.

It has been said before in these columns, and we stand by it, that in a conflict between the Government and the medical profession over the Labor Party’s health policy the winner (whichever it may be) may well experience a Pyrrhic victory. In principle, then, the desirable course is one of discussion, mutual understanding and acceptance of reasonable compromise. The tragedy of the emerging situation, however, is that the Government is leaving no real gate open for such a course. Its idea of discussion seems to be: “You talk; I listen and say no.” Its idea of compromise seems to be: “You compromise on my terms.” Its idea of mutual understanding seems to be: “Heads I win; tails you lose.”

One of the oddities of the present situation seems to be that, as has been said in another context, the two parties in conflict more often than not shoot past rather than at one another. This is because they have their own differing targets. The Government’s arrows on health are coming, paradoxically, not from the Department of Health, but from the Department of Social Security, having been shaped by a team of economists and political theorists. What emerges is a system of economic aid, with an unpleasant potential for regimentation, aimed at a target of budgetary accommodation. The medical profession on its side shapes its arrows in the form of personal medical service having as its target the highest standard of healthcare for the community. And it asserts—rightly, we believe—that this cannot be achieved on the cheap. We find it difficult to see anything but a continuing impasse unless Mr Hayden stops searching the pages of Roget’s Thesaurus for scarring epithets to describe doctors and recognizes that the A.M.A. speaks for a profession, the vast majority of whose members really do care that people and their health are properly looked after.

COMMENTS

ENURESIS

Young and Morgan, writing from England on “Conditioning Techniques and Enuresis” (page 329 of this issue of the Journal) state that it is common for general practitioners to reassure the parents of enuretic children that the child will grow out of the condition and not to recommend the use of the only really effective treatment—the enuresis alarm, or “pad-and-bell”. This appears to be equally true in Australia, not only of the general practitioner, but also of the pediatrician.

It is difficult to understand why this is so. Young and Morgan point out that in the psychoanalytic view enuresis represents an expression of some underlying emotional disorder; if it is treated in isolation, substitute symptoms may emerge. They also point out that there is no evidence of such symptom substitution occurring; on the contrary, there is considerable evidence demonstrating marked improvement in the general adjustment of enuretic children treated with the pad-and-bell. It could be that Australian general practitioners and pediatricians are unaware of these findings and retain the prejudice induced by the analytic theory. If this is so it is extremely disappointing. Much of the original work dealing with this and many other aspects of the pad-and-bell treatment have been carried out by Australian workers. Apart from those quoted by Young and Morgan, Bostock, Dibden, and Place have made important contributions, and the pad designed by Coote for treating this condition is so satisfactory as to be much in demand overseas.

However, pediatric psychiatry in Australia is taught almost exclusively by the analytically-orientated, a group not primarily interested in the findings of experimental clinical research. It could well be that the results of the Australian workers have passed unnoticed by them and hence have not been transmitted to medical students or pediatricians; or their validity has not been accepted: for example, Katz writing in this Journal reported the pad-and-bell treatment as no more effective than drug treatments, which were no more effective than placebos.

1 Coote M. A., Behav. Res. Ther., 1965, 2: 233.
2 Katz, L. Med. J. Aust., 1972, 1: 127.
Of course the analytic influence on pediatric psychiatry is not limited to Australia. Molony\(^1\) has pointed out that most of the popular textbooks of pediatrics written in the United Kingdom or the United States stress psychodynamic factors as causing enuresis, and either do not discuss treatment, or indicate that it should be purely psychological. The consistent demonstration that enuretic children are no more disturbed psychologically than controls and that purely psychological treatment is apparently no more effective than placebo therapy and vastly inferior to the pad-and-bell method\(^2\) seems to have been disregarded.

Another possible factor is that the majority of general practitioners have had no experience with this treatment method and are reluctant to make the necessary efforts to familiarize themselves with it. Medical students complete their pediatric training in both Sydney medical schools without ever seeing the pad-and-bell equipment, let alone having its use demonstrated. Whatever the reason, the result is that most enuretic children are denied an effective treatment; so for several years they cannot stay overnight with friends or in camps and are frequently ridiculed by their siblings, if not subjected to harsher measures. Meanwhile, their mothers have the additional burden of excess washing. This is not good medicine.

Doctors who are using the pad-and-bell method may be surprised to read Young and Morgan's conclusion that with the pad-and-bell method of treatment, initial arrest of enuresis may be expected in a median period of 17 weeks. Most workers here find that initial arrest to the same criterion (14 consecutive dry nights) occurs much earlier. This could be due to the use of a louder bell. This variable does not seem to have been examined in a controlled study. Worthy of note is Young and Morgan's report that the frequency of relapse is markedly reduced if, after reaching the criterion, the child is required to drink up to two pints of fluid in the last hour before retiring. Most workers have encouraged their subjects to drink increased amounts of fluid in the later stages of treatment, but possibly the more rigorous method of Young and Morgan is more effective. Even though the condition frequently remits with further treatment, relapse is very disappointing to all involved. Any method which may reduce this is worthy of study.

PREVENTION OF TETANUS

Although tetanus is a readily preventable disease it still causes a number of unnecessary deaths each year in this country. Active immunization has been shown conclusively to protect against this terrifying disease,\(^3\) yet two Australian surveys have shown that almost two out of every three adults have no active protection against tetanus. In a paper surveying tetanus immunity in Victoria, Trinca and Fraser reported that in 1967 only 829 of 2,403 adults had completed an active tetanus immunization course.\(^4\) This information was obtained from questionnaires returned by general practitioners who participated in a survey conducted by the Victoria Faculty of the Australian College of General Practitioners. More recently, Chapman and Davey have measured tetanus antibody levels in serum samples obtained from the Busselton survey in Western Australia in 1969. In a paper published in this issue (page 319) they report their finding that 839 of 1,400 adults had no detectable antibody to tetanus toxoid.

Natural immunity to tetanus does not occur in humans. Artificial immunization is, therefore, the only means of conferring immunity against this unpleasant disease. Immunity depends on the presence of sufficient circulating antitoxin to neutralize any tetanus toxin that is elaborated by the causative organism, *Clostridium tetani*, while multiplying in a site that favours its growth, e.g., in a tetanus-prone wound. Two methods of artificial immunization are available, namely active and passive immunization.

Active immunization is achieved by giving a course of tetanus toxoid-containing injections, the immunity conferred being solid and long-lasting, but taking several weeks to develop.\(^5\) Active immunity is maintained by giving regular booster injections throughout the individual's life. If a fully immunized patient sustains a tetanus-prone wound, a booster dose of toxoid should be given if more than two years have elapsed since the last dose of toxoid. The need to achieve active immunity of the whole population cannot be over-emphasized.

Passive immunization is indicated when a patient who is non-immune or partially immune sustains a tetanus-prone wound. The only way of achieving immediate protection in such patients is by the administration of preformed tetanus antibodies. Tetanus Immunoglobulin (Human)\(^6\) (TIG) should be used for this purpose. The hazards of giving heterologous antitoxins are well known, and, since the supply of TIG is assured, there is no longer a place for the use of equine or bovine antitoxins. In addition to TIG, a dose of adsorbed tetanus toxoid should be given simultaneously into a different site and, where appropriate, further doses of toxoid administered later to complete the active immunization course.

For routine prophylaxis a dose of 250 IU of TIG is given by intramuscular injection. The dose should be doubled if the wound is grossly contaminated, or if more than 48 hours have elapsed between wounding and the seeking of medical attention. Tetanus Immunoglobulin (Human, Intravenous)\(^7\) is also available in a 4,000 IU ampoule for the management of clinical tetanus. It should be noted that these doses correspond approximately to 1500 and 20,000 IU of heterologous antitoxin (equine or bovine), and that there is no need to give repeated doses of the human material because of its long life in the circulation. Experimental studies have shown that the material has a half-life of 3 to 4 weeks.

In summary, the long-term aim for tetanus prophylaxis must be active immunization for everyone. Until this goal is achieved passive prophylaxis will still be required in some cases. Such patients should now be given Tetanus Immunoglobulin (Human); there is no longer a place for the use of equine or bovine tetanus antitoxin.

\(^1\)Trinca, J. C., Med. J. Aust., 1965, 2: 116.
\(^2\)Trinca, J. C., and Fraser, A. N., Med. J. Aust., 1968, 2: 300.
\(^3\)Trinca, J. C. (editor), CSL Medical Handbook, Commonwealth Serum Laboratories, Parkville, 1973: 39.
\(^4\)Commonwealth Serum Laboratories.
VIRUS INFECTION AND ASTHMA

It is a general clinical observation that in young asthmatics respiratory infections tend to precipitate exacerbations of their asthma, yet the pathogenesis of this association remains obscure, and even the epidemiological evidence is poorly documented. However, a group of American physicians have recently made use of a rather exceptional opportunity to study the question in some depth. This opportunity occurred in the management of two groups of young children, aged 1 to 5 years, who were kept in hospital for periods of 6 or 7 months "for intensive diagnostic study and treatment because of a history of severe episodes of recurrent reversible obstructive airway disease". All respiratory infections occurring during the periods of study were investigated by means of bacterial and virological cultures, and by serological examinations during both acute and convalescent phases of the infections.

The 32 children involved in the study had 102 identified separate viral respiratory infections during the periods of observation. In the first group (12 children), there were 70 acute wheezing attacks, of which 33% were associated with proved respiratory infection. In the second group (20 children), there were 69 similar attacks, of which 51% were associated with viral infections. There were several small epidemics. The first group had one due to respiratory syncytial virus and one to parainfluenza virus type 2; the second group, during one six-week period, had 18 cases of the second group, during one six-week period, had 18 cases of the second group, during one six-week period, had 18 cases of the second group, during one six-week period, had 18 cases of the second group, during one six-week period, had 18 cases of.

The association of wheezing attacks with respiratory virus infection was unmistakable, but not all viruses were equally involved. Respiratory syncytial virus is well known to be frequently associated with bronchiolitis in infants, and in all but one of the 25 infections identified in the present study it was associated with acute wheezing attacks. Coronavirus infections too were usually associated with wheezing attacks, while at the other extreme, none of 11 cases of influenza A infection were associated with wheezing.

McIntosh et alii note that studies attempting to explain the association of asthmatic attacks with respiratory infection on the basis of bacterial allergens have not in the past given any definite answers. They too made regular bacterial cultures from nasopharyngeal and throat swabs, taken in association both with respiratory infections and with wheezing attacks, and while they isolated the usual range of pathogenic bacteria, these showed exactly the same distribution in swabs taken during wheezing attacks as in those from patients who were not wheezing. They note that, in the management of asthmatic patients, "attempts to desensitize by means of bacterial vaccines have . . . been of questionable value".

McIntosh and his colleagues point out that their study was not designed to demonstrate a statistical relationship. Nevertheless, their evidence of an association between attacks of acute wheezing and infection by certain species of virus is strong. Two observations are particularly relevant: one is that certain children who were remarkably symptom-free between attacks "developed clearly defined wheezing coincident with upper respiratory disease and viral infection"; the other is the fact already noted that some virus infections were regularly associated with wheezing attacks, while with other viruses this association was observed seldom or not at all. However, they admit that it is not clear what the mechanism is. The latter part of their paper is devoted to a discussion of possible mechanisms, and in particular of the relationship to bronchiolitis and of the relevance of studies of the latter condition.

In conclusion they state:

Our data fail to define a mechanism for the airway obstruction observed. However, it is curious that respiratory syncytial virus, which has a particular propensity to cause bronchiolitis, and which has apparently been involved in vaccine-induced hypersensitivity, should have been so clearly associated with acute wheezing in these children. But it remains uncertain whether the pathogenetic process illustrated in this study is similar or identical to that involved in bronchiolitis during infancy or following killed vaccine. Some episodes of viral bronchiolitis probably represent early attacks of airway obstruction in children constitutionally predisposed (perhaps by their atopic nature) to develop asthma, and therefore do not differ significantly from the wheezing attacks described here.

In spite of our uncertainty about mechanisms, it appears clear from these studies that viral respiratory infections trigger a substantial proportion of wheezing attacks in young asthmatic children, and that prevention of such infections, through vaccines or chemotherapy, would significantly improve their clinical status.

This paper is clearly a major contribution, and will undoubtedly be widely quoted in future discussions of the subject. It illustrates clearly how much remains to be done in the study of many common conditions. Its authors note that they have been able to find "only a single previous report which attempts to link specific viral or mycoplasmal respiratory infections with exacerbations of wheezing in individuals with recurrent reversible obstructive airway disease".

1 McIntosh, K., Ellis, R. F., Hoffman, L. S., et alii, J. Pediat., 1973, 82: 578 (April).
2 Berkovitch, S., Millian, S. J., and Snyder, R. D., Ann. Allergy, 1970, 28: 43.