policy, though there was little agreement on the extent to which the anaesthetist should be involved in the overall management of these units. While economy and practicality suggest that intensive care units should be sited near similar areas such as postoperative recovery rooms, separation between the groups of patients seems necessary if cross-infection and other undesirable results are to be avoided. Within the field of respiratory intensive care infants present special problems. The need for humidification, the difficulty of maintaining long-continued artificial ventilation, and the vexed question of tracheal intubation versus tracheostomy in infants were debated.

A little removed from the clinical sphere but of great potential importance for the future, much attention was devoted to the pharmacokinetics of anaesthetic drugs and to the applications of the computer to anaesthetic work. The broad mechanisms by which inhaled anaesthetics are distributed to tissues of different sorts is now well understood, and the importance of the solubility of the anaesthetic in blood in determining the speed of both induction and recovery is clear. The influence of ventilation on the uptake of inhaled anaesthetics is now well known. Blood volume is also an important factor. Thus in hypovolaemia arterial tensions are higher, but elimination is quicker, than in normovolaemia. Newer studies show, for example, the influence of depression of the cardiac output in apparently potentiating inhaled anaesthetics by tending to raise the arterial tension of the anaesthetic. The rate of uptake of volatile anaesthetics in infants is not necessarily the same as that for adults, nitrous oxide, for example, being taken up much more rapidly. The difference appears to be due to the relatively large cardiac output in the newborn. Certain halogenated anaesthetics formerly regarded as highly stable do in fact undergo biochemical transformation. Thus labelled bromine is released from halothane and accumulates in the body to be excreted in the urine.

Computers offer many possibilities to the anaesthetist. Apart from the handling of clinical records, at least one application which will be welcomed is the immediate transformation of data from monitors into physiological quanta. The matching of the data against a pre-set programme may well lead to the era of computer-assisted anaesthesia, and perhaps even to computer-controlled anaesthesia. Computers will also help in the study of pharmacokinetics and in the visual demonstration, for teaching purposes, of complex physiological systems. An unusual symposium in a congress of this sort was on communication in anaesthesia. This was sponsored by the Council for International Organizations of Medical Sciences and discussed the barriers which exist between specialists of different sorts within medicine, between the anaesthetist and the scientist, between teacher and student, and, perhaps most important of all, the barrier of language between specialists of different countries. Already these barriers are falling as anaesthetists emerge from their hitherto rather restricted technical sphere, for their interest is expanding rapidly both from the bedside to the basic science laboratory and from the operating-theatre into almost every other branch of medicine. A serious attempt to break down the barrier of language is the construction of a vocabulary of anaesthetic technical terms giving their precise definitions and their equivalents in at least one or two of the major world languages. This is already far advanced and will do much to eliminate many of the embarrassments both of translation of manuscripts and of communication at congresses.

[1] Intensive Care, B.M.A. Planning Unit Report No. 1, 1967. British Medical Association.

The congress showed that, though the changes in day-to-day work of the clinical anaesthetist have not been dramatic since the last congress four years ago, the understanding of his work and its influence on the patient has progressed greatly. Research has been intense on many fronts. Its influence on clinical use is sure to become apparent soon—much of it before the next congress due to be held in 1972 in Tokyo.

Tinea Pedis

Large numbers of any population of shoe wearers have abnormalities of the skin of their toe clefts, but most of them have no recognized pathogen there. In some the skin will show under Wood's light the pink fluorescence of erythrasma, a few may carry candida, and occasionally a bacterial infection appears to be responsible for the skin changes. The term tinea pedis, or athlete's foot, should be strictly reserved for those infected with a dermatophyte fungus.

The prevalence of this condition in the general population is unknown, and a survey of a true representative sample would present considerable difficulties. But much is known of the epidemiology of tinea pedis in different communities, especially from the work of Mary P. English and her associates in Bristol. The latest of her series of investigations—a study of tinea pedis in patients attending a chiropody clinic—is reported with Mr. J. Turvey in this issue of the B.M.J. at page 228. They found that 23% of 161 men and 4% of 98 women were infected, two-thirds of the cases being due to Trichophyton mentagrophytes var. interdigitale (=T. interdigitale). From this and earlier[1] studies it seems likely that the difference between the sexes is due largely to differences of exposure to infection and partly to differences in footwear. Experimental evidence confirms that an inherent difference in susceptibility is improbable.[2] We know that communities of boarding-school boys, long-stay hospital patients, and coal miners if they use pit-head baths all have high levels of infections—up to 60%.[3] [4] Boys at day schools are more likely to have tinea pedis if they are frequent swimmers at public baths. Dermatophyte fungi have been isolated from the floors of swimming-baths and shower-rooms[5] and from socks even after laundering.[3] The routes of transmission are in little doubt, and in this context the family bathroom should not be forgotten.

Though this condition is often trivial and may be self-healing, it is right to try to reduce the level of infection by preventive methods. Exclusion of infected swimmers from public baths is completely impracticable. Foot-baths are useless (or worse), but frequent hosing down of walk ways and shower-room floors is easy and of proved value. The floors of shower-baths are most heavily infected immediately after use.[5] It would seem logical, therefore, to incorporate in them a separate horizontal jet, set low to sweep the floor automatically after each person has passed. It is unrealistic to expect the general public to modify their washing habits or

[1] English, M. P., Brit. med. J., 1961, 1, 1086.
[2] Rosenthal, S. A., and Baer, R. L., J. invest. Derm., 1966, 47, 568.
[3] English, M. P., Wethered. R. R., and Duncan, E. H. L., Brit. med. J., 1967, 3, 136.
[4] Gentles, J. C., and Holmes, J. G., Brit. J. industr. Med., 1957, 14, 22.
[5] Gentles. J. C., Brit. med. J., 1957, 1, 746.

hose down their own bathroom floors, and many with real or supposed athlete's foot will continue self-medication with proprietary fungicides.

For those who do seek medical advice the diagnosis should ideally be confirmed by microscopy of scrapings cleared with potassium hydroxide solution and the species identified by culture. The patient with scaly feet, however, commands little priority in a busy surgery, and the nearest medical mycologist may be fifty miles away, so clinical criteria will often have to be relied upon. Erythema, fissuring, a scaly spreading edge extending beyond the toe cleft, and the presence of vesicles are more likely to indicate tinea pedis than are maceration and peeling strictly confined to the web space. The differential diagnosis includes psoriasis, especially the pustular form, shoe dermatitis or eczema, and pompholyx.

Therapy will vary with the type of case. Acute inflammatory tinea pedis is always best treated symptomatically with saline soaks, oily calamine lotion, and rest. After a few days a dilute steroid cream will often help, but should not be continued for more than a week or two. Then if necessary an antifungal application may be used, as in the ordinary case. There is probably little to choose between zinc undecenoate ointment *B.P.*, benzoic acid compound ointment *B.P.C.* (Whitfield's ointment), and 1% tolnaftate cream. Topical applications usually fail in the treatment of extensive, dry, chronic cases caused by *Trichophyton rubrum,* and griseofulvin by mouth, 500 mg. of the fine-particle preparation daily in divided doses after meals, may be necessary for several weeks. For reasons ill understood even this treatment often fails to eradicate the fungus from the toe clefts, and it is unwise to employ it for this purpose. The patient should be encouraged to wear light, open shoes whenever possible, but he who advises cotton socks these days will do well to find out first where they can be found.

Genetic Code Revealed

The Nobel prize for medicine has been divided this year between three American scientists. They are Dr. Marshall Nirenberg, of the National Institutes of Health, Bethesda, Maryland ; Dr. Har Gobind Khorana, of the Enzyme Institute, Madison, Wisconsin ; and Dr. Robert Holley, of Cornell University and now of the Salk Institute, La Jolla, California. Their contributions to the elucidation of the genetic code have won them the prize.

The central dogma of molecular biology has it that information can flow from deoxyribosenucleic acid (D.N.A.), the genetic material, to protein, the stuff of life, but not in the reverse direction. The information contained in the sequence of nucleotide subunits in D.N.A. is first transcribed into a corresponding sequence in ribosenucleic acid (R.N.A.), the "messenger" R.N.A. This is then decoded and the information acted on by the protein-synthetic machinery of the cell. Many distinguished men have applied their minds to the question of the general nature of the code. The award has been given to the experimentalists who cracked it. The subunits in the messenger, it was known, were read in threes, the triplet code-word corresponding to one amino-acid. Since only twenty amino-acids are generally found in proteins, at minimum only twenty codons, as they were called, might have been expected. Protein synthesis was also known to require adaptor molecules, remarkably predicted by Crick, which are small R.N.A. molecules, "transfer" R.N.A.s. The amino-acids are attached to them and they are presumed to recognize the codon sequence in the messenger. But, though the sequence in several proteins was discovered, the nucleotide sequence of no R.N.A. or D.N.A. molecule was known and the prospect looked distant.

How then could the code be elucidated ? At this point Nirenberg entered the arena. He and his collaborator from Germany, Matthaei, had been hard at work perfecting a system for protein synthesis which would operate in vitro. They found incredibly, and by all accounts unintentionally, that a synthetic R.N.A. analogue, polyuridylic acid (containing the single base uracil) would act as a messenger and that the protein analogue polyphenylalanine was formed. The first codon had been elucidated. UUU coded for phenylalanine. That was in 1961. A feverish burst of publication from several laboratories followed, in which a variety of other synthetic R.N.A.s containing the other bases, A, C, and G for short, were used. More and more codons were revealed, though some were of doubtful validity. Khorana and his colleagues in the meantime had been putting enormous effort into developing the chemical synthesis of polynucleotides. Showing a rare judgement in combining chemical and enzymatic methods, they produced polymers with defined nucleotide sequences. When used as messengers they produced polypeptides of composition and sequence exactly as predicted from Nirenberg's codon assignments. Nirenberg at this time also devised a rapid method for codon assignment—the triplet binding assay. By this means most of the 64 chemically or enzymatically synthesized triplets were studied. Out of all this, and in a remarkably short time, came the important conclusions that the code was degenerate—up to five codons could correspond to the same amino-acid—and moreover the codons originally assigned for the cell-free system from *Escherichia coli* were almost certainly uniform throughout nature. The code was universal.

But what of the molecular basis of codon recognition ? How does a transfer R.N.A. recognize its corresponding codon in the messenger ? Holley and his collaborators had been working doggedly on the separation of the individual transfer R.N.A.s from the mixture, which presumably contained 60-odd different species. The pure alanine-specific R.N.A. was isolated from yeast. Using largely enzymatic degradation, they succeeded in elucidating the complete sequence of the 77 constituent nucleotides. Very soon afterwards the sequences in two serine-specific R.N.A.s were elucidated by Zachau and collaborators in Germany, and since then the number has steadily mounted. As a result, the nature of the interaction of the messenger with transfer R.N.A., the fundamental act of information transfer, is becoming clear.

What have we as a result of this activity ? At first sight something quite trivial—a genetic dictionary with only 64 words: AAA = lysine, AAC = asparagine, and so on, including the two punctuation marks for "begin" and "end" protein synthesis. There is something sublime in the simplicity of this little book, which must have been written and fixed for all time at a very early stage in evolution.

The application of the new knowledge must sooner or later find an outlet in medicine. But to the question, What can medical science expect to gain from these developments ? the short answer is, "At present, nothing." True, our under-