implications are obvious. The Royal Colleges' Recommendations recognize the problem and make a strong plea for extra resources for both the manpower and study-leave budgets.

The consequences of failing to achieve CME certification are not spelt out in the colleges' Recommendations, but can only be significant. It is likely that Trusts will require CME certification as a condition of service for career grade doctors, since the medico-legal implications of having uncertificated physicians on the staff are considerable. Uncertificated specialists are equally unlikely to find favour with the Royal Colleges and within their specialist societies.

How should rheumatology CME develop within the bounds allowed it by the Combined Royal Colleges' Recommendations [1]? The best way would be to encourage a consensus to develop rapidly within the career grade BSR membership by a programme of continuing research about which activities it most favours. Since CME is at the end of a continuum of learning which includes undergraduate and specialist training, this might lead to a 'core curriculum' along the lines of those developed for both undergraduate and specialist medical training in this country [4]. Certainly activities shown to be highly regarded by the BSR membership would carry considerable validity. The BSR Education Committee already scrutinizes and approves postgraduate courses run under the Society's aegis. Provided that concerns about possible conflict of interest can be addressed it would be a short step for the Education Committee to do the same in future using the Royal Colleges' CME Offices' criteria as the benchmark. If it takes on this role the BSR Education Committee should logically also perform the necessary research about what activities should be offered at BSR-run courses. Participative activities are likely to find much favour; it would be surprising if strategies to cope with the cascade of change in which British doctors now find themselves, were not also favoured.

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THE RHEUMATO-MICROBIOLOGICAL INTERFACE

MICROBIOLOGY is unique in pathological sciences in its rate of change, not so much in improvement of speed or accuracy of diagnosis of specific infections, but in the nature of the organism itself. Thus, those microbes associated with bone and joint diseases prevalent now may vary unpredictably in properties from those pathogens one or more decades ago. Even the names of organisms alter. Neisseria catarrhalis became Branhamella catarrhalis, only to be converted to Moraxella catarrhalis. Taxonomic changes are usually well founded as a result of improvements in the specificity and reliability of measurements of microbial components or products. For bacteria, these measurements have changed from gross colonial appearance and stains, to metabolic activity, to specific proteins, to DNA and now to RNA, particularly ribosomal analysis. The enthusiastic case report of a novel cause of septic arthritis in reality be due to a newly classified organism.

But micro-organisms do change, often dramatically, in properties and their effects over the years. Sometimes the exact reasons for such change are not understood. Thus rheumatic fever has become near extinct in the UK despite the ease of isolation of Streptococcus pyogenes from patients' throats. This trend has been attributed to the 'routine' use of antibiotics in sore throats, or spontaneous variation in the antigenic structure of the organism. These explanations are, however, speculative [1].

In other ways, bacteria do change rapidly in precise properties. Resistance to antibiotics comprises the most obvious means of microbial change. In recent years, resistance to penicillin in Streptococcus pneumoniae, to glycopeptides (e.g. vancomycin) in Enterococcus spp. and extended spectrum cephalosporins in Klebsiella spp. have been identified [2]. Changes in the properties of bacterium other than its sensitivity to antibiotics are also important, but may not be evident unless specifically sought. For rheumatologists, staphylococci remain the major cause of focal sepsis. The most pathogenic species, Staphylococcus aureus, was initially identified by its golden-coloured colonial appearance, then the ability of plasma to clump suspensions of it, and then through identification of extracellular products such as deoxyribonuclease or coagulase or the presence of the cell wall component, protein A. Typically, S. aureus is positive for all these factors, S. epidermidis is negative. But exceptions occur, not uncommonly. For example some golden staphylococci are coagulase-negative and S. epidermidis may, on occasion, produce DNase.

Indeed, some so-called methicillin-resistant isolates of S. aureus (MRSA) whilst being resistant to many antibiotics, possess little protein A or cell-bound
coagulase [3]. With these cultures, it is as if the bacterial cell in having to acquire antibiotic resistance genes to survive the hospital milieu, has lost the capacity to synthesize substances, including those associated with pathogenicity. Such cultures rarely appear to cause primary sepsis of bones and joints; rather their biological niche is predominantly that of colonizers of ulcers and other surface lesions, with only occasional pathogenic incursions to deep parts of the body. Yet these organisms are still named *S. aureus* with the implication of pathogenicity with which this species has been associated.

Similarly *S. epidermidis* may not always be the innocent commensal. When isolated in pure culture from lesions, with relevant clinical effects, it is highly likely to be pathogenic, often due to the production of a sticky glycopeptide secretion.

Thus, to distinguish whether a culture, such as an aspirate from a joint, yields pathogenic *S. aureus* or skin commensal *S. epidermidis* is not always easy. With other organisms, the uncertainties may be even greater because potential variables may rarely have been sought or characterized.

The crucial approach is that whenever an important, interesting or resistant bacterium is encountered, then an immediate and thorough dialogue between the rheumatologist and microbiologist should accurately delineate the significance of such an isolate through the latter performing further procedures other than those considered routine.

Similarly when active joint disease is thought to be associated with micro-organisms at a remote site, e.g. salmonella, campylobacter or clostridia in the gut, then such collaboration is also necessary. Such interface will not only provide the most accurate diagnosis and optimal management for the patient; but it will improve the understanding of the disease process, provide interest for both clinician and the laboratory-based worker, and reduce some of the spurious claims over the pathogenic roles of certain micro-organisms in joint disease.

It is regrettable that so many of the papers submitted to this journal that describe microbial events do not have any input by the local microbiologist. They are there to help!

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