ety can be performed; decision on the donating of eyes or other tissues that can restore lost functions to the living.

Whatever one may believe about a life after death, one can scarcely deny that the memory left in the thoughts of those still living provides a means of achieving some part of it.

As penned by an unknown author:

> We cannot know the ending of the path,
> Nor quite accept its regimented bliss,
> Devoutly planned for life's long aftermath,
> Nor hold to any certain thing, save this:
> They have not gone, nor can they dwell apart,
> Who still have place within some living heart.

INFECTIOUS DRUG RESISTANCE

SINCE the inception of antibiotic therapy, the dramatic healing power of antimicrobial agents has been threatened by the ever more insistent emergence of antibiotic-resistant bacteria. Some of the clinical[1] and biologic[2] problems posed by this phenomenon have been summarized recently. Until lately, the principal mechanism responsible for drug resistance was thought to be spontaneous mutation at a low rate to a particular drug resistance followed by selection of resistant cells in the presence of the drug.

Although threatening enough, this mechanism is slow and cumbersome in comparison with infectious multiple drug resistance, a newly discovered process that is intellectually fascinating and therapeutically frightening. First recognized in Japan in 1959, infectious multiple drug resistance is a process by which sets of genes determining resistance to several unrelated antibiotics are transferred together from resistant to sensitive strains by cell-to-cell contact.

These genes are not located on the bacterial chromosome, but on extrachromosomal genetic elements called R factors, which are composed of DNA but replicate autonomously. R factors contain a region called RTF (resistance-transfer factor) that determines infectivity and to which the separate drug-resistance genes are attached. R factors resemble viruses without coats, but they are also modified sex factors since they mediate their own transfer from cell to cell.

Transfer occurs not only within species of the enteric bacteria but also between groups as diverse as shigella, salmonella, klebsiella, vibrio, pasteurella, serratia and the ubiquitous *Escherichia coli*, which can serve as a reservoir. Indeed, *Esch. coli* is of key importance in facilitating transfer of R factors from one pathogen to another and from animals to humans.

An analogous situation exists in the staphylococci, where antibiotic therapy has been notoriously difficult. Genes for resistance to penicillin, erythromycin, tetracycline, chloramphenicol and kanamycin are carried on extrachromosomal particles called plasmids.[2] No RTF's have been found, but plasmids are transferred from cell to cell by phages. It may be the rapid emergence of drug resistance among the staphylococci is related to the dissemination of plasmids by phage transduction.

The first report of R factors outside Japan came from Great Britain in 1962,[3] and by 1965, extensive surveys of their distribution as well as studies of their fundamental properties had already been carried out there[4,5] as well as in Japan[6]; R factors had also been reported in other European countries and in Israel. However, the first clinical study of R factors in the United States, by Kabins and Cohen, appears elsewhere in this issue of the *Journal*, and similar studies are in progress in Boston and New York. These investigations stress the present widespread occurrence of enteric bacteria harboring R factors in this country. And they emphasize the threat to antibiotic therapy posed by these infectious agents as well as the need to monitor their spread.

Both Japanese and British studies have correlated the precipitous rise in frequency of R factors with the increasing use of antibiotics not only in clinical practice but also in the care and feeding of livestock. Antibiotics are now incorporated routinely in livestock feeds, providing a constant selection pressure on R factors that can be readily transferred to man. It appears that unless drastic measures are taken very soon, physicians may find themselves back in the preantibiotic Middle Ages in the treatment of infectious diseases.

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POST-TRANSFUSION PURPURA

A SIXTH case of post-transfusion purpura is described by Morrison and Mollison in this issue of the *Journal*. The infrequency with which this disorder occurs places it rather low among the various causes of postoperative thrombocytopenia, but some of its implications deserve consideration. Morrison and Mollison suggest that part of the antibody produced in response to the PI^{A1} platelet antigen crossreacts with a structurally similar antigen on autologous platelets, resulting in thrombocytopenia and absorption from the circulation of the cross-reacting antibody fraction and thereby rendering it