mode of transmission nor the extent of genetic determination is by any means clear.[5 6] In one of a collection of papers on current concepts of schizophrenia published in the *American Journal of Psychiatry*, Matthysse and Kidd[7] have calculated that neither a single major locus nor a multifactorial model can account adequately for the rates of schizophrenia found in practice in the relatives of schizophrenic patients. Both models predict genetic heterogeneity among schizophrenics, which means that biological factors might not be operating in all cases. On the other hand, the most promising work on environmental transmission has not been adequately confirmed on repetition possibly because of differences in the diagnostic composition of the series of "schizophrenic" patients.[8 9]

The value of the phenothiazines in treating the central symptoms of schizophrenia and preventing their recurrence is undoubted. The injected forms have some advantages because of more certain absorption and because some patients who are willing to accept injections would not bother to take tablets. The possibility that there may be undesirable consequences from very long-term medication cannot, however, yet be eliminated. This advance in pharmacotherapy has stimulated research interest. For example, abatement of schizophrenic symptoms by phenothiazines and butyrophenones is associated with blockade of dopamine receptors in the brain, while provocation of similar symptoms by amphetamines seems to result from an increased synaptic activity of dopamine or noradrenaline, or both. Snyder, who first wove the threads of this dopamine hypothesis together, has remained cautious in his appraisal,[10] since there is no direct evidence that dopamine has anything to do with schizophrenia, but he can justifiably claim that the indirect evidence is impressive. It is too soon to talk about the anatomy of schizophrenia, but the work of Slater, Beard, and Glithero,[11] who described typical "schizophrenic" symptoms in patients with temporal lobe foci, and more recently of Gruzellier,[12] who found a lateralised dysfunction in schizophrenic patients, when combined with our increasing knowledge of the distribution of the dopamine system in the brain indicates some potentially fruitful lines for investigation. Several other biochemical hypotheses are well worth pursuing.[13]

Meanwhile, it remains true that schizophrenia becomes a chronically handicapping condition in at least one-quarter of cases in spite of pharmacological treatments. As with other such disabilities, this does not mean that nothing can be done to help. Progress has also been made recently in understanding the nature of the environmental factors that can help maintain an optimum balance on the schizophrenic tight-rope.[14] There are dangers on each side. The tendency of the schizophrenic patient to withdraw may well be protective in part, but in conditions of social poverty it can go too far.[15] On the other hand, the patient seems to be vulnerable to social pressures that most people take in their stride.[16] Those who live in a family where there are unrealistic expectations, a ready tendency to criticise, or too much emotional involvement are particularly at risk of relapse.[17] Under these circumstances medication is most useful, but the provision of alternative residential or daytime environments may also be therapeutic.[18]

The family environment benefits the patient in a substantial proportion of cases—a fact that requires emphasis. The myth of the "schizophrenogenic" mother dies hard, and the fact that the relatives are often acting as unpaid and untrained primary care workers is often forgotten. In our present unfortunate conditions they are caught between a hospital system that is running down and a community alternative that has not yet built up to anything like adequate levels.[19] The recent White Paper recognised that the burden on relatives is sometimes intolerable.[20] We may expect further advances in scientific knowledge, but we should also take care to apply to the full the knowledge we already have.

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Nuclear-powered pacemakers

As the applications of implantable artificial cardiac pacemakers[1] have become increasingly numerous and complex[2] advances in technology have reduced wire and electronic faults to negligible proportions.[3] Nevertheless, the power source continues to be an important limitation to their usefulness. Ideally a single implantation would last the rest of the patient's life, but the mercury-zinc cells used in conventional pacemakers seldom last more than two years[4] and are apt to fail suddenly.[5] Replacement is a relatively minor procedure; but it is not a pleasant outlook for the elderly, who do not relish hospital admission, or the young, who may have 20 or more replacements to look forward to. Furthermore, the procedure is costly in terms both of pacemaker units and of theatre time and personnel.

Since nuclear power is the most potent and long-lasting energy source known, not surprisingly considerable efforts have been made to develop reliable nuclear-powered units. Different isotopes and principles have been employed,[6] but the technological problems have not proved unduly difficult, and since 1970 about 1400 units have been implanted in patients. A recent review of the published cases[6] shows that power failure has not occurred and that the component failure rate has been no greater than with conventional pacemakers. More important, there have been no reports of cancer or

leukaemia which might have been induced by local radiation from implanted units, though the exposure time (2000 patient-years) is still relatively short. Recovery of units from patients after death has been very high, allaying fears[7] of widespread dissemination of plutonium-238. Nevertheless, nuclear units are still not recommended for the very elderly or for infants, and it seems reasonable to suggest that a safe chemical power source with a life expectancy of about 20 years would be preferable to a nuclear power source with a half-life of 87 years and unknown dangers. This possibility may have been realised with the new solid lithium-iodide battery,[8] which has been giving promising results in clinical trials and may eventually prove to have the best cost/efficiency ratio.

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Postoperative pain

Postoperative pain has not aroused as much concern in the lay public as the control of pain in patients with terminal cancer; nor in many instances have doctors and nurses been as assiduous in its management as they might be. There are several reasons for this. Relative to terminal cancer, postoperative pain is often short-lived though equally or more severe; many patients quickly forget their pain, or, having recovered from their operation, feel it ungracious to complain of earlier discomfort. Surgeons may be more concerned with the technical aspects of the operation and the eventual outcome; too readily they may delegate the responsibility for control of postoperative pain to their juniors, who in turn lean upon the nursing staff, who do not have the authority to prescribe controlled (DDA) analgesics. Both doctors and nurses are afraid of inducing addiction and, like many patients, have too complacently accepted pain as an inevitable consequence of surgery. In some instances potent analgesics are withheld because the patient's blood pressure is low, neither doctor nor nurse realising that it is low as a consequence of pain.

In addition to discomfort and misery postoperative pain is apt to bring a train of avoidable complications that may jeopardise the patient's recovery. Immobility induced by fear that any movement may cause pain increases the liability to deep vein thrombosis, bed-sores, hypostatic pneumonia, muscle wasting, and urinary retention and constipation; it may substantially retard convalescence.

The key to successful control and management of postoperative pain is imaginative anticipation of the patient's needs. As to patients with terminal cancer, analgesics of adequate potency and in adequate dosage should be given *before* the discomfort is so intolerable that the sufferer counts the minutes before he can no longer desist from pushing the bell-button for relief, which may be some long time in coming —particularly at night. Pain is always worse and more intolerable in the small hours, when nursing staff are most depleted and the difficulties of having the dose of a DDA drug checked are greater. It is for the doctor to decide whether the patient's insomnia is due to pain or to anxiety and strange surroundings. The distinction is important, but often it is helpful in establishing peace of mind and confidence to give an analgesic last thing at night and first thing in the morning.

In anticipation of severe pain an intramuscular (or rarely an intravenous) injection of diamorphine, morphine, pethidine, or pentazocine is required. For effectiveness and lack of side effects diamorphine takes pride of place. The fear of addiction in these circumstances has been exaggerated to the detriment of patients. Diamorphine is less prone to induce vomiting than morphine, and is preferred, particularly after abdominal surgery. The respiratory depressant effect of morphine has been overstressed, as any physician knows when watching the respiratory excursions increase after relief of severe pleural pain in lobar pneumonia. Pethidine is a less effective analgesic, as is pentazocine, which in some patients may have the additional disadvantage of causing frightening nightmares or even hallucinations. Patients in Britain should count themselves fortunate that (in contrast to some other countries) diamorphine is available for the relief of pain after surgery or myocardial infarction, an emergency in which it causes less haemodynamic disturbances than morphine. Postoperative pain is most appreciable nowadays after abdominal or thoracic surgery. Analgesia must not only be adequate but should be given to synchronise with physiotherapy, so that breathing exercises and coughing are effective in keeping the respiratory tract free of mucus.

The transition from a parenterally to an orally administered analgesic may be difficult to bridge, but by the time the patient is taking fluids by mouth the pain is usually less intense. Fluids should be warm to prevent the upper gastrointestinal tract going into spasm. Soluble aspirin, paracetamol alone, or the more potent combination of paracetamol and dextropropoxyphene (Distalgesic) may suffice if given every 4-6 hours. Codeine alone or in combination with aspirin or paracetamol may be effective, but dihydrocodeine, parenterally or orally, is often more potent. There is a real need for a more powerful oral analgesic, particularly for patients averse to injections. To a large extent this hiatus is filled by an effervescent preparation of soluble aspirin 500 mg and papaveretum 10 mg, tablets which are exempt from DDA control because of their size and were originally formulated and produced in the pharmacy at Westminster Hospital. So widespread throughout Britain has their demand become, that they are now commercially available. The usual dose is 1-2 tablets every 4-6 hours, but—as with all analgesics—the dosage must be related to the age and the weight of the patient, and to personal characteristics, some individuals being less sensitive to pain than others.

Any drug is valueless if it remains in its ampoule or bottle. Doctors need to anticipate each patient's analgesic needs in the light of his personality and the surgical trauma and then write up a variety of appropriate medications and leave clear instructions with the nursing staff how these agents are to be used—emphasising the importance of forestalling the evolution of unacceptable pain. Before operation the patient should be told that these arrangements have been made, so winning his confidence. Realism is added if the patient is told that he must report at once any serious discomfort, which may require for its relief more potent analgesia—or the simple but all important nursing skills that are still taught and available in Britain.