during one five minute contact a GP asked about the psychological sequelae of diabetes mellitus. During the discussion he revealed that a diabetic patient of his had recently committed suicide. Subsequently it became clear that the GP had gained a good deal from the contact, particularly reassurance about his own practice, beyond the information ostensibly sought.

As community care extends within psychiatry, both GPs and psychiatrists have an increasingly important role coordinating the resources of psychiatry and primary care. Before starting the study we had established that few contacts were taking place between other mental health staff and the primary care team. We were also surprised to find relatively few contacts between the psychiatrists and non medical primary care staff. While increased liaison by non medical staff may be desirable, it appears that the doctors were acting as the filters and communicators between complex multidisiciplinary teams. The central importance of communicating, who is doing what for whom when, is suggested by the most commonly discussed category being "clarifying the role of staff" followed by "conveying information".

This combination of liaison with routine clinical work in general practice is part of what has been called the "comprehensive collaborative model" of liaison psychiatry in general practice (Tyrer *et al*, 1990). It enables ordinary out-patient clinics to provide more patients with the benefit of specialist psychiatric advice (Creed & Marks, 1989), as well as perhaps enhancing the care of those receiving direct psychiatric care. There is also evidence that patients seen in a primary care setting are more likely to receive all their psychiatric care in this setting and have significantly less need for in-patient services (Tyrer *et al*, 1990). It seems reasonable to conclude that the brief liaisons we have described are both cost effective and useful to both general practitioners and psychiatrists and that this "hybrid vigour" could be extended elsewhere without much difficulty.

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Psychiatric Bulletin (1990), **14**, 594–596

Observations on the management of depot neuroleptic therapy

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For the past three years I have been involved in a study of the effects of depot neuroleptic therapy on the social functioning of chronic schizophrenics with Dr D. A. W. Johnson. Having had to screen the bulk of the depot patients of two Area Health Authorities (South and Central Manchester) in the search for possible study entrants, and then monitor a large group of patients for two years who were under the clinical control of a range of Consultant Units, I have been in a good position to make observations about depot management. These are generalisations and may not apply equally to other psychiatric services, but I suspect they will have some relevance.

The main management failure I have seen concerns stable uncomplaining out-patients who are too often left on the same high depot dose that they were discharged on following an acute episode many years previously. Perceived as presenting little management problem, such patients are commonly passed to the most junior doctors who may see them only once or twice in a six-month placement. Faced with a patient who is well controlled and not complaining of any side effects, why risk the wrath of the patient's relatives and your consultant with a boat-rocking dose reduction? So the patient continues to run an unnecessarily high risk of tardive dyskinesia, potentially life shortening obesity, and possible reduction in social functioning. The not inconsiderable cost of unnecessary depot medication must also be considered.

Since it is impossible to learn good depot management simply by experience in six-month placements, the answer has to lie partly in teaching, and partly in better supervision of junior doctors in this area. To be effective, the latter needs to be what I term "active" rather than "passive". Passive supervision says, "Come and talk to me about any problems with your out-patients" – but the group under discussion do not present any – whereas active supervision says, "We will have regular meetings to discuss the management of all your out-patients".

Turning to teaching, I have often been asked by new doctors (presumably because I am often to be seen around the Depot Clinic) how one decides when it is appropriate to reduce or stop depot therapy. What they are usually hoping for is a simple formula: reduce to a certain dose after so many months, stop completely after 'X' years, etc. Unfortunately, the more I try to manage depot therapy, the more I find that each case has to be assessed individually using the criteria set out below. I do not regard reduction or cessation of depot therapy as discrete events during depot treatment, but rather as part of a continuous risk/benefit analysis for each individual. Stopping therapy should be just the final stage if a carefully managed process of reduction has not revealed a need for continuing depot therapy.

So how does one decide if a dose reduction is in order? Obviously by the patient's mental state initially; generally the presence of active psychosis contraindicates reduction. The exceptions are patients who have very long-standing phenomena that have never responded even to very high neuroleptic doses, and are no longer distressed by them. In such cases quality of life may sometimes be improved by dose reduction without exacerbation of symptoms, but management by an experienced clinician with an excellent long-term knowledge of the patient is required. Assuming that the patient is free of psychotic symptoms, what other factors are entered into the risk/benefit analysis? Conventional neuroleptic side effects and tardive dyskinesia are unlikely to be forgotten when considering the risks of therapy, but the role of neuroleptics in inducing weight gain which can lead to reduced social involvement or non-compliance can be easily overlooked, as can relationship threatening impairment of sexual function.

The duration of a stable mental state must be a major consideration. Although disagreeing with rigidly applied formulas, I would not normally reduce a depot dose until the patient had been free of psychotic symptoms for at least six months – unless forced to by severe side effects — and not stop an injection completely until at least five years from the last acute episode. There is evidence (e.g. Johnson 1976, 1981, 1984), that relapse rates are increased if depot therapy is terminated earlier than this, and although unproven it may be justifiable to project this trend beyond five years. Remember that we are weighing up risk against benefit for each patient, rather than trying to minimise the risk of relapse at any cost.

Essential information is the patient's response to previous reductions or cessations of depot therapy. If conscientious attempts in the past to reduce or stop depot therapy have led to repeated relapses, it would be pointless to try again unless there has been some major change in the patient's social circumstances, or many years have elapsed.

Social circumstances need careful consideration when deciding whether or not to reduce depot dose. Are the patients under stress from a difficult housing or financial situation, jobs they cannot cope with, or critical and over-involved relatives? If such problems are acute and not chronic, wait for a more auspicious time before trying a reduction. Look at the characteristics of previous relapses for further guidance. Did they start gradually with easily identifiable prodromal symptoms, or abruptly with no warning? In such acute episodes, were the patients dangerous to themselves or others, and what consequences would a repetition at this time have for the patient's social or vocational life? Patients whose function in normal employment are unimpaired by neuroleptic therapy and support a family have more to lose by an episode of illness than those who live with and are supported by their parents.

Safe dose reduction relies on early detection of deterioration, so that relapse can be prevented by re-increasing the dose. Thus, when assessing the risk of reduction, one must consider how much insight patients have, and whether they are aware of the beginnings of a relapse and can be relied upon to tell you, even though they know this will lead to their medication being re-increased. Similarly, have they relatives or close friends who will notice early signs of deterioration and inform the hospital? This is especially important if the doctor concerned is in

only a six-month appointment and does not know the patients well. Relatives should be involved whenever possible in the reduction or cessation process, partly for the preceding reasons, and partly to allay their anxieties about dose reduction. They and the patients need to be reassured that they can ring the hospital and be seen rapidly if any deterioration occurs. If appropriate I give the patients or relatives a supply of emergency oral medication.

Non-compliance is an issue to be considered, as it renders any form of management difficult. Patients sometimes stop or increase the spacing of their depots unilaterally because they suffer side effects, or fear long-term consequences, and feel that the doctor is unwilling even to consider reducing or stopping the treatment. In such circumstances, explaining the rationale of a cautious controlled reduction programme may prevent the patients stopping their depot abruptly, with the attendant high risk of relapse. If patients' non-compliance is due to carelessness or ambivalence however, I tell them that good compliance is a prerequisite for a programme of reduction and possible eventual discontinuation of depot therapy.

If, after consideration of these issues, a reduction seems indicated, how should this be achieved in practice? An essential principle is not to make changes too frequently. A new plasma level is obtained after three injection cycles in theory, but the relapse rate after stopping depots tends to increase sharply from three months onwards. It would seem prudent to use three months as a minimum change frequency if three injection cycles would be less than this. I do not generally decrease an injection by more than one third at any time which, combined with a three-month minimum interval between changes, means that it takes 12 to 24 months depending on initial dose to reduce an injection to zero. One might question whether such a long-term management strategy should be carried out by doctors in short-term posts. The answer should probably only be in the affirmative if the patient or relatives are very reliable, and there is good hand-over of information at the end of each six months. The management of less straightforward cases should be in the hands of a more senior psychiatrist.

With regard to the mechanics of actual dose reduction, if the patient is on supplementary oral neuroleptics, aim for monotherapy by reducing and eventually stopping these before altering the depot. Once this is achieved, the question arises whether to change the dose or the frequency of the depot. Half-life figures in drug literature, etc. are not of great value, as the effective half-life increases with the dose in mg per kg of body weight. If the patient is suffering ill effects at peak level times (around three days post injection) a dose reduction might be the first step, otherwise it is more convenient for the patient and depot administrator if the spacing is increased first. Longer intervals between injections makes compliance more likely, especially in employed patients, but four weeks should probably not be exceeded until the dose reduction process is quite advanced.

It will be noted that I have made no reference to absolute dose. This is because I believe the aim of treatment should be to have each individual patient on the minimum dose that will effectively maintain them, and this can vary widely.

The process of reduction may not, of course, continue serenely to eventual discontinuation of therapy. In many instances the re-emergence of symptoms will call for the dose to be returned to the level before the most recent reduction. This is in no way a failure of therapy. Rather it is now certain that the patient still benefits from the depot, and that the dosage is at the minimum compatible with this benefit. It must be remembered, however, that this optimum dose applies only to this patient, at this time, in a particular life situation, and could change either way in future.

In conclusion, I would contend that successful depot management requires a large amount of common sense, a basic knowledge of the pharmacology of these drugs, and an absence of unthinkingly applied formulas or ideologies. Management of the stable uncomplaining patient in particular could be improved by increased consultant supervision of junior staff, who should be taught as part of basic therapeutics how to weigh up the risks and benefits of therapy for each individual patient.

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https://doi.org/10.1192/pb.14.10.594 Published online by Cambridge University Press