often a prescription insurance requirement, the entire script will still cost them only that same $3?

The false economies of forced minimal script writing appall me. We all know that patients will have allergic or other unpredictable reactions to almost any medication. My usual (when unfettered) custom is to write for a one- or two-week supply, then have the patient phone me and, if appropriate, refill the prescription for the long term, satisfied that the patient is at least tolerating the medicine. But lately I hear, "Doc, my insurance covers all my prescriptions, but you have to give me enough for three months (or six months)." The implication is that it is to the patients' economic advantage to throw 100 or 200 pills down the toilet if they do not like them or if they perceive that they somehow do not help. Yet with the cost of prescriptions as high as it is, what is the physician to do? Economic self interest is certainly on the patient's side.

Insurance for prescriptions is a boon to us all. Some patients with chronic diseases save hundreds, even thousands, of dollars a year. Without such insurance many of them would have to forego medicines that we must prescribe and that they badly need. I realize that buying and selling in quantity saves money, but I cannot believe the economics that dictate that writing for a minimal three-month supply of medicine instead of a preliminary trial of one week or one month can account for the difference between a $3 prescription and a $100 prescription or that a patient must have my written prescription for a year's supply of medicine or be financially penalized. After all, these are powerful drugs we are authorizing, not stocking the freezer with a side of beef!

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National Health Insurance in America—The Issue of Copayment

To THE EDITOR: Dr Grumbach's well-written article in the August 1989 issue about the need for national health insurance[1] *almost* converted me to become a believer in it. The reason for the almost is that I have been in ardent opposition to national health insurance for the past 30 years because of the firsthand experience I gained in my own country, Hungary. In Hungary, national health insurance is bad for the patients and bad for the physicians. Of course, Dr Grumbach didn't propose to copy the Hungarian way of national health insurance; he suggests copying that of the Canadian system. In the same issue, the editorial, "A View From Canada," by Dr Banks indicated that the Canadian system also has many shortcomings and is not a solution either.[2]

I see the point, however. Now, as my grown-up children are joining the labor force, they are at the same time joining the 37 million uninsured in the job marketplace. I am concerned, not so much about the possible decline of my future income but about the health care of my children without insurance.

Now that communism has failed in every aspect of life, how can anyone recommend any form of national health insurance? A solution has to be found, though.

So, how can the national health insurance system work in the framework of free enterprise, be fair to everyone, and become even better than the Canadian experience? The key, I believe, is that patients shouldn't be relieved of total responsibility for their own health care, as Dr Banks pointed out. Human nature should be taken into account in order to prevent bankruptcy of the system. In order to avoid bankruptcy, the system should be set up in such a way as to avoid abuse. It appears to me that this can be done best with copayments.

I suggest making copayments on a sliding scale and excluding catastrophic illnesses so as to not hurt the poor.

I believe in these copayments because of what I have experienced in my own practice. All Medi-Cal patients are supposed to pay $1 per visit. Most physicians do not enforce this policy for one reason or another. I didn't either, but recently I started asking for $1 copayment, and I was amazed to see how the number of cases of recurring vaginitis and cystitis, vague abdominal and back pains, and so forth decreased. Based on this experience, I have started to believe in the concept of copayments, which help to decrease abuse of the system. With copayments, national health insurance may work.

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Alopecia Universalis in Autoimmune Polyglandular Syndrome Type I

To THE EDITOR: We read with special interest the report of Imam and co-workers in the September 1988 issue on the occurrence of alopecia universalis in a patient with autoimmune polyglandular syndrome type I.[1] During the past 16 years we have been following a very similar patient, a boy who presented at the age of 3 years with alopecia areata of the scalp. Signs of mucocutaneous candidiasis involving the oral mucosa and the fingernails developed the following year. Later, hypoparathyroidism (at age 5 years), uveitis (at age 7), and Addison's disease (at age 8) were diagnosed. The alopecia progressed from areata into universalis within four years, with loss of all body hair, including eyebrows and eyelashes. Pubertal development—such as testicular growth—was first observed at age 11. Plasma testosterone levels increased gradually to adult values, and basal plasma gonadotropin levels were normal. The patient's voice modulated normally, and he reported conscious ejaculations at age 14 years. Adult testicular volume was reached at 16 years, but no sexual hair appeared at any stage of puberty.

Because of cataract, both lenses were removed when the patient was 12. Primary hypothyroidism and vitiligo developed at age 18 years. An elevated titer of antiadrenal antibodies—but not of any other autoantibodies—was found, indicating a possible autoimmune pathogenesis of the disease. Like Imam and colleagues' patient, this patient had severe emotional problems related partly to his physical appearance and alopecia, and noncompliance with his medication regimen resulted in frequent episodes of hypocalcemia and, on one occasion, serious hypercalcemia due to vitamin D intoxication.

We think that our data on this patient support the assumption of Imam and associates that alopecia universalis might be a feature of autoimmune polyglandular syndrome type I and not just a coincidental association. Of particular interest

is that in our patient alopecia was the presenting sign of the syndrome preceding the endocrine abnormalities.

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Electroconvulsive Therapy Use During Pregnancy

To THE EDITOR: In the September 1989 issue, Guze and Guze very aptly discussed the concerns about the use of psychotropic medication during pregnancy,[1] and Kerns editorially expanded on the discussion of this dilemma, stressing the need to manage a patient with nonbiologic treatments.[2] This often is not successful in patients with severe psychiatric disorders, and some type of somatic therapy is needed to resolve a severe depression, catatonic state, or other psychotic condition. Elsewhere in the same issue, Gerner stated that electroconvulsive therapy (ECT) should be "included as an early intervention" in the treatment of intractable depression.[3] No discussion of ECT is present in either of the above discussions of psychiatric treatment during pregnancy.

Electroconvulsive therapy given during pregnancy has been shown to be both effective and safe for mother and fetus in many published case reports. Sobel concluded that ECT does not increase fetal mortality and morbidity; on follow-up, children born to mothers who had ECT developed normally.[4] Repke and Berger indicated that ECT may be the treatment of choice for the acutely depressed pregnant patient.[5]

A review by Gelenberg suggested that ECT may be helpful in the treatment of some cases of depression, mania, or psychosis during pregnancy.[6] Summarizing the current state of the literature, Abrams concluded, "ECT is not known to exert any such adverse fetal effects . . . and should be used in preference to drugs in pregnant women and during the nursing period."[7(p26)] He argued that excessive monitoring is not necessary.

Severe psychiatric illness affects not only the mother but the fetus, as well. Maternal disregard for health issues, poor food and liquid consumption, an altered biochemical environment of the mother, such as abnormal cortisol circadian rhythm, and attempted suicide could all adversely affect the developing fetus. Electroconvulsive therapy should be at least considered as a possible option for all patients during pregnancy and recommended when appropriate.

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Dialysis Disequilibrium Syndrome

To THE EDITOR: The article by Drs Harris and Townsend on the dialysis disequilibrium syndrome in the July 1989 issue[1] omits important information. If rapid dialysis in an acutely uremic patient is the major cause of this syndrome, the reader should know the size of the artificial kidney used, the blood flow, and the weight of the patient. The early literature on the subject always described it as probably due to the osmotic effect of rapid decline in blood urea and other osmotically active substances. The report by Drs Harris and Townsend[1] points out several clinical studies that do not support this explanation. Arieff and associates[2] described using mannitol and glycerol in the dialysis solutions in studying experimental cerebral edema in dogs. They were able to prevent brain edema in dogs with the use of glycerol and suggested that its addition to dialysate might be effective in preventing the dialysis disequilibrium syndrome.

For many years I have used a smaller kidney and/or a lower blood flow of 100 ml per minute for the first hour and then 150 ml per minute during the first dialysis; I have usually limited this dialysis to about two hours. I have not encountered dialysis disequilibrium for about the past 22 years by using these precautions.

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Drs Harris and Townsend Respond

To THE EDITOR: We wish to thank Dr Levin for his comments about the dialysis disequilibrium syndrome. Our patients undergoing dialysis for the first time are also put on a smaller kidney, and the blood flow is at a slower rate than that used for chronic dialysis patients. Although occasional patients have transient headaches, the patient discussed in our report was the only one to suffer such dire effects since the dialysis unit opened more than ten years ago. The flow rate used for those undergoing their first dialysis ranges from 200 to 250 ml per minute, in contrast to rapid dialysis done at 450 to 500 ml per minute. The patient described was dialyzed at a rate of 250 ml per minute on a CA90 dialyzer, which has a surface of 0.9 m^2 and is smaller than those used for chronic dialysis.

There is still controversy concerning all of the causes of the dialysis disequilibrium syndrome. We pointed out the various papers on the subject including the paper cited by Dr Levin written by Arieff and associates. Fortunately, this is a rare syndrome today. We presented the case simply to remind others that it can occur even under standard conditions of dialysis.

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