rescue, the probability that the person would be found, his accessibility to rescue, and the amount of time that elapsed before rescue. Although both factors often vary together, so that persons who make highly lethal suicide attempts also arrange low rescue potentials, the ratio serves to remind the clinician that an elevation of either factor may indicate the seriousness of the suicide attempt.

One difficulty, as the article by Reich and Kelly elsewhere in this issue of the *Journal* illustrates, is the incidence of false-positive indicators of suicide potential when one attempts to predict suicide before the attempt. Although physicians daily witness profound despair and tragedy in their patients, suicide attempts are an unusual event, and successful suicide is rarer still. The problem is akin to the dilemma of predicting violent behavior in general; even in the face of previous self-directed or other-directed violence (the best single predictive factor), one can state with assurance only that certain persons are in a high-risk group; attempts at predicting individual behavior usually only succeed in cases of extreme behavioral disorder. Reich and Kelly report 17 suicide attempts during a seven-year period, with no deaths, during which 70,000 patients were admitted to the hospital. Similarly, Litman, in a survey of Los Angeles County Hospitals with a daily census of over 20,000, found only 12 suicides during a two-year period.[11]

Thus, although the clinician must be alert to the potential for self-destructiveness in his patients, an actual suicidal act will often occur as sudden and unexpected behavior. The determinants of human behavior are complex and continually defy any simplistic guidelines. The task of the clinician is to allow himself to respond to painful feeling states in his patients, particularly those of bitterness and desperation, which so often raise the suicidal impulse. Psychiatrists learn to allow such responsivity in themselves through their training,[12] but studies such as that of Reich and Kelly serve as a reminder that the primary physician needs also to be vigilant for the intensities of affect that can propel patients towards destruction by their own hand.

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SERUM FERRITIN FAILS TO INDICATE HEMOCHROMATOSIS — NOTHING GOLD CAN STAY

FOR several years we have enjoyed a comfortable certainty that serum ferritin would provide, in healthy people, an accurate reflection of the body's stores of iron.[1,2] Although the test is exasperatingly difficult to perform, once established it seemed able to sort out people with iron deficiency (even without anemia) and those with hemochromatosis (even before the iron deposits had ruined their liver, pancreas and testicles). Now that reflection has been tarnished. Wands et al., at the Massachusetts General Hospital (page 302), have demonstrated normal ferritin levels in iron-loaded people afflicted with preclinical familial hemochromatosis. There is no doubting their data. The diagnoses were established by liver biopsy, and the ferritin levels were measured in triplicate in Brisbane and confirmed in duplicate in Seattle. (It is a tough procedure, when Boston must go to Brisbane.) Already, 4 to 15 g of iron have been removed from seven of their patients without exhausting the stores of iron.

How does it happen that the depots can be overfilled with iron, stored therein as ferritin, while the serum ferritin remains normal? Wands et al. suggest several possibilities. The title of their paper "precirrhotic hemochromatosis" implies, perhaps, that the serum ferritin levels may not increase until damage has been done; they mention high ferritin values in hepatitis, "a finding that probably reflects, at least in part, either impaired uptake or the release of ferritin from damaged liver cells." They also mention the reticuloendothelial system as the source of serum ferritin, a possibility discussed by Jacobs and Worwood.[2] It may be important that most patients with hemochromatosis do not store iron in the spleen and bone marrow.[3] Furthermore, iron-loaded macrophages in hemochromatosis contain only tiny grains of iron, and they do not accumulate in the intestinal villi, whereas in transfusion siderosis the macrophages contain multiple large accretions of iron, and they pack themselves into the tips of every intestinal villus.[4,5] These interesting aberrations have suggested to some of us that the sessile macrophages in the spleen and the wandering macrophages in the tissues are incapable, in hemochromatosis, of properly synthesizing ferritin. If this hypothesis is true where does the excess serum ferritin come from in some patients with precirrhotic hemochromatosis and all patients (so far) with advanced, destructive hemochromatosis? One easy answer is that the abnormal serum ferritin may be of epithelial, not reticuloendothelial, origin.

How is this observation "relevant to the pathogenesis of the abnormal iron accumulation in idiopathic hemochromatosis?" To comprehend this problem it is first necessary to understand the essence of hemochromatosis: a failure to control iron absorption. Iron balance, the exchange of iron with the environment, is controlled by modifying the amount absorbed. The intestine admits only enough iron to offset what is lost by blood loss and by the obligatory loss in desquamated epithelium. The mechanics of control are

which means that information about requirement must be generated somewhere, somehow, and transmitted to the gut. The information must be received by the gut and then translated into appropriate behavior, so that the villous epithelium absorbs iron when needed and refrains when it is not — a complicated system, involving many mechanisms. Failure of the system resulting in a failure of control would permit unneeded iron to enter the body. This is a useful definition of hemochromatosis.

The control might fail in several ways: wrong information, failure of transmission, inaccurate translation. Thus, we are brought to a conclusion that hemochromatosis is not one disease. It is a group of diseases. Or, like anemia, it is a sign of many disorders. Its association with many easily distinguished hereditary diseases supports this conclusion.[6] Recognition of this diversity should end the feckless efforts to explain hemochromatosis as a single disease with a single pattern of inheritance.[7]

One of several unresolved problems of the control of iron absorption is how information concerning iron requirement is carried to the intestine. It seems not to be anemia, oxygen, serum iron or the rate of hemolysis or erythropoiesis.[8] Greenman and Jacobs have examined the possibility that serum ferritin is the messenger, but injections of ferritin into rats did not impair the absorption of iron[9] (note, however, that the ferritin injected was of epithelial origin). Ferritins from different tissues display heterogeneity.[10] Perhaps reticuloendothelial ferritin is the messenger. Patients with hemochromatosis may be unable to synthesize ferritin in the reticuloendothelial system. The cloud of epithelial ferritin spilled from an iron-sodden liver into the serum may not be able to pass the word, "Hold, enough." A lack of reticuloendothelial ferritin could explain the failure of control for one kind of hemochromatosis. But what about hemochromatosis associated with thalassemia major, in which reticuloendothelial cells are filled with ferritin?[11]

What of the future of serum ferritin as a noninvasive test to find preclinical disease in relatives of patients? In the two families described by Wands et al. "serum ferritin values did not reflect mobilizable tissue iron stores." It means that we shall have to depend as before upon a diagnostic procedure properly called cumbersome: serum iron, the desferal iron-excretion test and, ultimately, liver biopsy. Of course, this cumbersome procedure may not be required in all families. In some the iron-loaded precirrhotic relatives may have high serum ferritin levels.

Finally, all iron buffs will be sad to learn that what might have been the perfect screening test for disorders of iron metabolism has not panned out.

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DEPROFESSIONALIZING THE PROFESSION

So many vocations are now identified — often self-identified, to be sure — as professions, that the designation has become all but meaningless. Traditionally and ideally, however, a member of a profession pursues a scholarly discipline in a way that inspires confidence and trust, and that prizes expert performance of a skill above financial gain. A profession thus suffers when suspicion and distrust replace its fiduciary image, and when commercialism supersedes other, less selfish motives. Unfortunately, such debasing trends appear to be affecting most of the country's professions, including that of medicine — "The Shame of the Professions," Max Lerner has recently called it.[1]

Few, interestingly enough, are happy about the deprofessionalization of medicine — neither the doctor nor his critic. The reasons for their discontent, however, differ. To the physician, the task of inspiring confidence and trust becomes impossible when the patient has been indoctrinated to disrespect medical authority and to see the doctor as an incompetent and arbitrary wielder of irrational decisions ("Talk Back to Your Doctor" exhorts a recent book[2]), a person to be sued. The critic is concerned not just by the size of medical fees but by the apparent unwillingness of the medical profession to make some charitable financial adjustments in the care of the poor or uninsured, by the unavailability of some doctors except during "business hours," and by the increasing threat and even use of doctors' strikes or other forms of what the British call industrial action.

Into this state of declining professionalism — desired by no one except a few doctrinaires who seek to eliminate any suggestion of medical elitism — federal bureaucracy has now intruded, and this intrusion must rank as sheer inanity to anyone who believes that superior medical care and the maintenance of some professional attributes go hand in hand. In brief, the Federal Trade Commission has initiated action that, if successful in hearings and in the courts, would remove the traditional ban against advertising by doctors. If the FTC has its way, and doctors suc-