When Should Entry into a Regular Haemodialysis Programme Occur?

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In this issue we publish an article by Bonomini and his colleagues which is iconoclastic. It casts doubt on the accepted notions for the degree of renal failure at which regular dialysis should be commenced. We believe that regular dialysis should commence with a GFR of less than 5 ml/min, the standard Seattle procedure set down by Scribner 14 years ago, but that in places where good dietary therapy is available using a selected low-protein diet, it is possible, but not desirable, to postpone dialysis until the GFR has fallen to 2 ml/min. In places less fortunate than Beersheva, Manchester or Pisa in their dietetic staff, it is better to dialyse earlier rather than too late. There is no point delaying dialysis until neuropathy or bone disease are disabling, or until there is widespread arterial calcification. On the other hand, there is no need to dialyse too early, in patients who are well maintained and content on a 20-gram selected low protein diet without neuropathy or overt problems with divalent ion metabolism. The reason for this is simple: patients who are not dialysed do not die from accidents of dialysis. No patient on a selected low-protein diet has developed air embolism from a dialysis he was not having! It would, on the other hand, we feel, be remiss of us to refuse to hear a voice crying in the wilderness who says that our preconceptions are wrong, and that early dialysis at levels of GFR of 15–25 ml/min is indicated. We have published the evidence in the paper of Bonomini and his colleagues. We believe that dialysis at GFR's of 15 ml/min is unjustified (with the rare exception of occasional cases of severe hypertension or massive proteinuria and as a temporary measure only) for several reasons: (1) dialysis facilities would be swamped overnight and those who can't live without dialysis would die because of lack of places; (2) dialysis should be reserved for those in whom there is no alternative; (3) at GFR's of 15–25 ml/min, when no diet

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is needed (other than phosphate binders in Western Europe and the USA, to prevent hyperparathyroidism) there is no doubt in our minds that dialysis is worse than the untreated renal failure at this early stage; (4) regular dialysis is a procedure having both morbidity and mortality in its own right and in these patients it may possibly shorten their lives or at least cause them illness which they would not suffer from were they not on this premature therapy. We are not convinced that there is a need for dialysis to prevent complications measurable solely by minor disturbances in nerve conduction or biochemical parameters.

Nevertheless, we feel that the school of Bonomini has a right to be heard. We invited professors Scribner and Maher to put their views, and professor Scribner has a detailed article in which he appraises the data of Bonomini. It is clear that Scribner, as the father of chronic haemodialysis, has remarkably good results which most of us would envy, and that he was somewhat reluctant to publish a detailed criticism of a colleague's views. Nevertheless, we thought that his views

should be juxtaposed with the paper of Bonomini and his colleagues, so that our readers can judge for themselves. For us, the problem remains theoretical, largely because of the pressure of patients in very advanced renal failure who need dialysis to remain alive. We do not have the dialysis places to engage in dialysis in persons with GFR's above 3 **ml**/min.