imaging procedures in patients, however, and we would welcome alternate suggestions by Dr Charnley. We have at least one year follow up of all participants which does not challenge any diagnoses in the main report.

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Family occurrence of achalasia
Sir,—The familial occurrence of achalasia or diffuse oesophageal spasm in four families described by Dr Frieling (Gut 1988; 29: 1595–602) and colleagues is of considerable interest as is their review of other recorded instances of the disease involving members of the same family. The preponderance of horizontal transmission leads them to suggest inheritance through an autosomal recessive gene. This pattern of involvement, however, would be compatible with common exposure to some agent in the family environment in the past because the development of clinical symptomatology almost certainly lags many years behind the initiation of the pathological process.

We have been able to find only one instance of achalasia in monozygotic twins which suggests that the disease is not usually genetically determined. In a study of 1012 first degree relatives of 167 patients with achalasia we were unable to detect one established case of achalasia and none of the 447 siblings of patients with achalasia had the disease although several had oesophageal symptoms. This indicates that in the vast majority of patients no hereditary factor is present and those in which there is a family history of the disease are an interesting but atypical minority. In our view it would be wrong to suggest that achalasia or diffuse oesophageal spasm are inherited disorders and thus divert attention from the search for an environmental causative agent.

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Family occurrence of achalasia.

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Gut 1989 30: 559
doi: 10.1136/gut.30.4.559

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