

unknown. Although auto-immune related thyrotoxicosis has been reported after amiodarone treatment,⁶ neither of our patients had a goiter or thyroid antibodies before amiodarone treatment and antibodies did not develop during follow up.

These data suggest that amiodarone induced thyrotoxicosis has a peculiar pathogenesis. When intrathyroidal amiodarone concentrations exceed a threshold, cell damage leads to thyrotoxicosis when the contents of the thyroid leak into the bloodstream. The intra-thyroidal concentration of amiodarone too would decrease, allowing repair and the restoration of euthyroidism. If this hypothesis is true, continuation of amiodarone treatment might eventually lead to a recurrence of thyrotoxicosis when the intrathyroidal amiodarone concentration again exceeds the threshold.

In our patients the follow up period was probably too short for a second period of thyrotoxicosis to develop.

- 1 Trip MD, Wiersinga WM, Plomp ThA. Incidence, predictability and pathogenesis of amiodarone induced thyrotoxicosis and hypothyroidism. *Am J Med* 1991;91:507-11.
- 2 Smyrk TC, Goellner JR, Brennan MD, Carney JA. Pathology of the thyroid in amiodarone-associated thyrotoxicosis. *Am J Surg Pathol* 1987;11:197-204.
- 3 Martino E, Aghini-Lombardi F, Mariotti S. Treatment of amiodarone associated thyrotoxicosis by the simultaneous administration of potassium perchlorate and methimazole. *J Endocrinol Invest* 1986;9:201-7.
- 4 Reichert LJM, de Rooy HAM. Treatment of amiodarone induced hyperthyroidism with potassium perchlorate and methimazole during amiodarone treatment. *Br Med J* 1989;298:1547-8.
- 5 Brennan MD, van Heerden JA, Carney JA. Amiodarone-associated thyrotoxicosis (AAT): Experience with surgical management. *Surgery* 1987;102:1062-7.
- 6 Martino E, Macchia E, Aghini-Lombardi F, et al. Is humoral thyroid autoimmunity relevant in Amiodarone-induced thyrotoxicosis (AITT)? *Clin Endocrinol* 1986;24:627-33.

CORRECTION

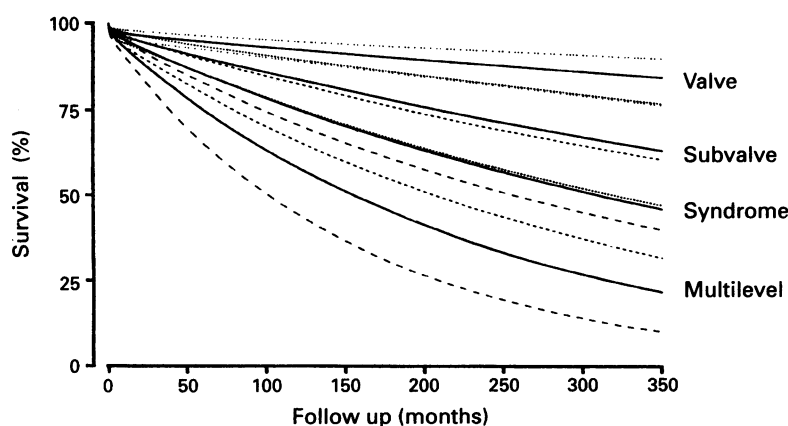


Figure 1 Predicted risk of death (and 70% CIs) for a patient presenting at 13.9 months of age with moderate obstruction of the left ventricular outflow tract plotted against level of obstruction from a solution to equations developed by means of hazard analysis (appendix 1).

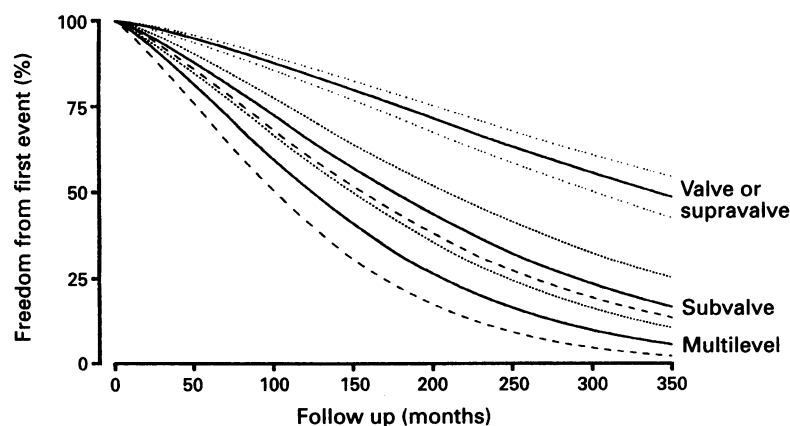


Figure 2 Predicted freedom from first clinical event (operation, balloon dilatation or endocarditis, and 70% CIs) for a patient presenting at 13.9 months of age with mild obstruction of left ventricular outflow tract without aortic regurgitation plotted against level of obstruction from equations developed by means of hazard analysis (appendix 1).

Incidence and prognosis of obstruction of the left ventricular outflow tract in Liverpool (1960-91): a study of 313 patients

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We regret that owing to a printer's error figure 1 and figure 2 in this article in the June issue (*Br Heart J* 1994;71:588-95) appeared in the wrong order and with the wrong legends. The corrected versions are reprinted on the left.