AJMALINE IN CARDIAC ARRHYTHMIAS:

Ineffectivity or Indifference?

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Ajmaline, an indolin base, was first isolated from the Indian plant rauwolfia serpentina by Dr. Salimuzzaman Saddiqui (1) and named after Hakim Ajmal Khan, a pioneer of Tib in India. It is a member of a second group of rauwolfic derivatives which have no sedative, hypnotic, or hypotensive effects. Arora et al. (1956) in the subcontinent and Kleinsorge (1965) in the Europe confirmed its anti-arrhythmic efficacy and this has been substantiated (Ilyas 1970, 1981, Klein et al. 1980). It is also useful in the treatment of arrhythmias associated with Wolff-Parkinson—White syndrome (Wellens et al. 1974, Khaliullah et al. 1980). Despite proven efficacy, ajmaline is not in use in Pakistan.

Electrophysiologically, ajamline produces prolongation of A-V interval and QRS duration, and haemodynamically it slows down the pulse rate and lowers arterial pressure and stroke volume in increasing doses. It significantly depresses the intraventricular conduction as the main mechanism of antiarrhythmic effect and leads to prolongation of conduction intervals in healthy subjects and patients (Bohme et al. 1968). and also has some sympatholytic activity (Schmitt et al. 1960).

In an intraindividual comparative study in 15 patients with chronic stable ventricular extrasystoles of various origins, in the order of effectiveness were ajamline, propafen and lidocaine and suppression of extrasystolies was most marked after ajmaline (Klein 1980). In 40 patients with 87 episodes of tachycardia, 85 episodes (96%) were sinoverted; 17/27 cases of atrial fibrillation and 4/7 cases of atrial flutter were were also sinoverted (Forster et al. 1966), and ajmaline was ineffective in chronic atrial fibrillation. Serious side effects in this series were observed in 2/66 (3%) cases. In one case with bundle branch block short asystole occurred and in the other transient ventricular flutter was observed. Ajmaline has been used safely and effectively in cases of arrhythmias in children (Keele 1968, Kast 1968), and in post-infarction tachycardias (Brauch 1964).

Ajmaline diagnostically blocks ventriculo-atrial conduction, without stimulateous effect on A-V conduction in cases of ventricular tachycardia with retrograde conduction to atria (Sandoe 1972). It has been postulated that ajmaline blocks anomalous bundle but not conduction in the normal heart (Chiale et al. 1977). Ajmaline in 24 cases of pre-excitation syndrome lengthened P-R interval in 75%, delta-wave disappeared in 64% and changes in QRS time were recorded in 58% of cases (Sepulveda et al. 1976). In 35 cases of WPW syndrome ajmaline intravenously caused temporary interruption of pre-excitation in 60% of cases (Rosantranz et al. 1965) and abolished WPW syndrome in 19/27 cases (70%; Forster et al. 1966).

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Toxic effects of ajmaline include hypotension, decreased cardiac output and atrioventricular block. It is contraindicated in atrial flutter and severe conduction disorders. Side effects were reported in 3/66 cases (3%: Forster et al. 1966). Rarely ajmaline has been used suicidally by over-dosage (Hager et al. 1968).

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