Similar effects to those in goldfish were obtained in sunfish with 1 and 4 p.p.m. In rainbow trout and in seafish 1/4 p.p.m. was still effective and induced stage II. In young salmon 2 p.p.m. led rapidly to loss of righting reflex, and laboratory experiments performed with *Gambusia* were very promising[2].

In frogs, 2 p.p.m. paralysed the hind legs after an immersion period of 30 min; 16 p.p.m. produced loss of righting symptoms within 10 min.

Immersion of adult species of salamanders in 4 p.p.m. for 15, 30 and 60 min was carried out without fatalities; stages II and III were easily obtained after 5 and 20 min of immersion. Allowed to recover in an empty aquarium, all animals became normal after 6 h.

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## Effect of Reserpine on Atrial and Ventricular Rates in Atrial Fibrillation in Man

INVESTIGATIONS into the intrinsic rhythmicity of normal cardiac pacemakers suggest that the catecholamine content of the myocardium is an important determinant of their activity[1]. Reserpine depletes stores of catecholamines; in the dog heart–lung preparation after initial acceleration it slows the sinus rate[2], and in the dog heart *in situ* with heart block it markedly slows the ventricular pacemaker[1]. In man reserpine slows the normal heart[3], but since the sinus node is under extracardiac control this observation provides no basis for speculation concerning the mechanism of reserpine effects on the cardiac pacemaker.

Atrial fibrillation is a special circumstance in which a rapid ectopic pacemaker is relatively independent of extracardiac autonomic control. In atrial fibrillation in man, reserpine slows the ventricular rate[4], but its effect on the atrial rate has not been reported. The slowing of the ventricular rate does not mean that there is necessarily a corresponding change in atrial rate, for in atrial fibrillation where the ventricle responds to atrial impulses, changes in atrial rate often result in reciprocal changes in ventricular rate, for example, after quinidine the atrial rate falls and the ventricular rate rises. In addition, ventricular rate may change as a result of effects on AV conduction. An acceleration of ventricular slowing by atropine in one patient as reported by Marangoni and Cavusoglu[4] does not establish increase of vagal tone by reserpine since atropine could accelerate the ventricle if reserpine had not been given.

After a two-week control period, 7 patients with atrial fibrillation, suffering from mental disease but not in congestive failure, were given reserpine in daily oral doses of 2·0 mg for one week, and 3·0 mg for the week following, after which the reserpine was withdrawn. There were no other changes in medication during the investigation period. The atrial rates were counted according to the method of Gold *et al.*[5] in 1-min electrocardiographic strips (sternal lead), before the administration of reserpine, twice weekly during treatment with reserpine and at the end of each week after withdrawal. Ventricular rates were determined from the same strips of electrocardiogram.

The averages of the counts were plotted. The curves (Fig. 1) indicate that the ventricular rate fell after the first week of treatment, continued below the control during the entire treatment period (but did not fall further when the dose was raised from 2·0 mg to 3·0 mg per day, indicating that the former was a ceiling dose)

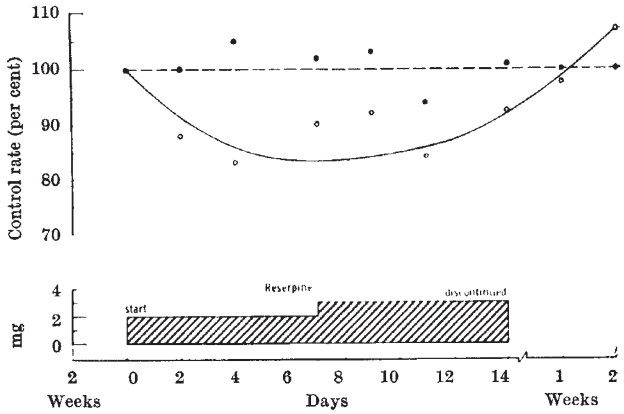


Fig. 1. Atrial and ventricular rates in seven patients with atrial fibrillation receiving reserpine. ———, Ventricular rate (100 per cent = 85·7/min); — — — —, atrial rate (100 per cent = 407/min)

and rose after the first week of withdrawal, returning to control at the end of the second week. The atrial rate was not changed by the reserpine. The average rates after reserpine were paired with control rates in each case and examined by the sign test. The $P$ value for the atrial slowing compared with control was 0·31 during treatment and 0·4 after treatment. The $P$ value for ventricular slowing was 0·005 during treatment and 0·39 after withdrawal. Our findings are diametrically opposed to those of Arora[6], who observed atrial slowing but no ventricular change after reserpine in atrial fibrillation induced by aconite in the dog heart *in situ*.

Since in our cases there was no change in atrial rate, there could have been no significant effect of catecholamine depletion on the ectopic mechanism, and since all ventricular beats were responses to atrial impulses, the only simple and reasonable explanation for the ventricular slowing is depression of AV conduction by reserpine. Nothing in this experiment bears on the mechanism of the effect; there are the possibilities of unopposed vagal activity as a consequence of catecholamine depletion, a central action to increase vagal tone, and, as already suggested by Innes, Krayer and Waud[7] on the basis of observations in the dog heart–lung preparation, of a direct depressant effect on AV conduction.

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# HAEMATOLOGY

## Polypeptide Chains of Buffalo Haemoglobins

THAT many breeds of cattle exhibit polymorphism involving two haemoglobin types is now well established[1]. On the other hand, buffalo haemoglobin seems to be monomorphic in that all buffaloes examined possess two major haemoglobin components[2,3]. Bovine haemoglobins have been fairly thoroughly characterized[1] and it has been suggested that the two bovine haemoglobins may contain one polypeptide chain in common[4].