

stannic salts failed to show any noteworthy difference. The effect of concentration indicated marked differences both when injected intravenously or subcutaneously. Thus 20 milligrams of tin per kilo, injected intravenously, produced a moderate albuminuria when the amount of tin per c.c. was 1.5 milligrams; much greater amounts of albumin and large amounts of sugar when each c.c. contain 8 milligrams of tin; acute death when each c.c. contained 20 milligrams of tin.

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The influence of tartrates, citrates and oxalates on the isolated heart.

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Sodium tartrate and citrate in Locke's or in Ringer's solution or in defibrinated blood perfused through the isolated heart of the frog, dog and cat caused a decrease of cardiac activity which became more marked with increased concentrations. The action of the citrate was considerably greater than that of the tartrate, the ratio being about 5 : 1 with weak solutions. The difference was even more marked when the action of more concentrated solutions was compared, an $N/100$ citrate was as active as $N/10$ tartrate, as observed on the frog's heart. The action of citrate and oxalate was compared by its effects in calcium-free solutions (Ringer and Locke minus calcium). The effect of citrate and oxalate was found to be the same in some experiments but in others the action of the oxalate was distinctly less toxic than that of the citrate. Since the solubility in water of calcium citrate is twice as great as calcium tartrate, while calcium oxalate is practically insoluble, it is apparent that the action of these salts is not due to the precipitation of the calcium. Again since the equimolecular solutions, calcium tartrate and calcium citrate, have the same effect as CaCl_2 it would also indicate that a possible decrease in ionization of the calcium in the tissues is not a factor which determines the action of tartrate, citrate and oxalate on the heart.