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大量抗坏血酸注射液对心血管系统的作用

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摘要:

本文报导大量抗坏血酸注射液(injectio acidi ascorbici,以下簡称IAA)对动物血压、心脏及血管的影响。IAA为用碳 酸氫鈉中和的10%抗坏血酸灭菌溶液,pH約为5.6,內加焦亚硫酸鈉作稳定剂。靜脉注射IAA 0.2克/公斤及0.4克/公 斤可使麻醉犬、猫和家冤动脉血压輕度短时上升;在因手术創伤、失血及数种降压莉物(氯丙嗪、亚硝酸鈉、組織 胺、罌粟碱)所致低血压的动物,IAA的升压作用較为明显持久。同剂量抗坏血酸溶液(solution acidiascorbici,以下 簡称SAA)靜脉注射常引起血压下降,用碳酸氫鈉将SAA中和后即有升压作用,焦亚硫酸鈉无升压作用,故IAA的升压作 ▶加入引用管理器 用是中和后的抗坏血酸所引起。靜脉注射IAAO.2克/公斤可增加麻醉犬心搏量、心輸出量及心脏指数,对总外周阻力 无明显影响;对在体冤心亦呈現兴奋作用。1:1,000和1:500的IAA灌流离体冤心后,可見心縮振幅加大,心率略增,冠 脉流量增多。IAA对离体冤耳、腎、下肢血管无明显影响。在体冤下肢及腎血管血流量在血压上升时均有增加。犬 脾容积在IAA注射后有輕度扩张現象,而SAA却使脾容积短时減少。实驗結果表明,IAA的升压作用主要是由于心脏功 能改善,心輸出量增加所致。

关键词:

THE CARDIOVASCULAR EFFECT OF LARGE DOSES OF INJECTIO ASCORBICI ACIDI

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Abstract:

Ascorbic acid has been used in treatment and prevention of hypo-or avitaminosis C as well as in many other clinical conditions including some cardiovascular diseases. The intravenous injection of large doses of Injectio acidi ascorbici (IAA) (pH=5.6) was known to be effective in the management of cardiogenic shock in Kershan disease. The present paper reports the experimental results on the effect of IAA on blood pressure, hearts, and blood vessels with a view to elucidating certain mechanisms of its efficacy in cardiogenic shock. In urethan-or Na-pentobarbital-anaesthetized dogs, rabbits, and cats, an intravenous injection of IAA at the dosage of 0.2, 0.4 g/kg moderately and temporarily raised the arterial blood pressure to the extent of about 10-20 mm Hg, lasting about 5-10 minutes. No significant change in heart rate was observed, but during the early phase of the elevation of blood pressure the respiration became quickened and deepened. As the blood pressure of animals was reduced below 80 mm Hg by operation and haemorrhage or various hypotensive drugs (chlorpromazine, sodium nitrite, histamine, papaverine), the pressor effect was more pronounced and sustained than that in anaesthetized animals. Solutio acidi ascorbic (SAA) always produced a drop of blood pressure, but after being neutralized with sodium bicarbonate to pH=5.6, it could exert the same pressor effect as IAA. It seemed, therefore, that the pressor effect of IAA was due to the partially neutralized ascorbic acid in it. Cardiac output, was estimated on Fick's principle in urethan-anaesthetized dogs, an intravenous injection of IAA 0.2 g/kg increased the cardiac output, cardiac index, stroke volume, and aortic mean pressure, especially in hypotensive animals. The total peripheral resistance was not obviously altered. In using IAA (1:500, 1:1000), both the contractile amplitude of Cushny's rabbit's heart in situ and of isolated rabbit's heart were enlarged; at the same time, heart rate and coronary flow of the latter were also increased. In perfused isolated rabbit's ears, hind legs, and kidneys, IAA (1:500, 1:1000) showed no remarkable effect, except a slight dilatation. During the rise of blood pressure after IAA administration, the blood flow of hind leg and kidney by Kaverina's method was increased. IAA slightly increased the spleen volume of anaesthetized dogs, while SAA always diminished it. From these results, the pressor effect of IAA seemed to be chiefly due to the improvement of cardiac function, resulting in an increase of cardiac output. It was considered that IAA might serve as an effective drug in combating cardiogenic shocks other than those in Kershan disease.

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