



Review Article

Acupuncture Treatment for Low Blood Pressure and Shock

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Abstract

In 1980-1990 we have a series of animals (dogs, rats and rabbits), which undergone experiments using nitroprusside-induced low blood pressure and hemorrhage or endotoxic shock, by using Electroacupuncture (EA) applied on S36 and P6 acupoint to increase BP and antishock. These effects are related to the activation of central cholinergic system in hypothalamus and lower brain stem. These results can be a strong spot for clinical application of EA to treat low BP and shock.

Keywords: Central cholinergic function; Electroacupuncture (EA); Endotoxic shock; Hemorrhage; Neostigmine; Nitroprusside

Introduction

Usually in many references acupuncture and electroacupuncture are used to treat pain and hypertension. However, in China, in some clinic doctors also use acupuncture to treat hypotension or shock. 1973, Hunan Medical College Hospital reported that acupuncture can improve low blood pressure and shock treatment [1]. Thus, acupuncture is not only used to treat hypertension but also for low pressure and shock. Therefore, after we have done many experiments to analyze the mechanism of acupuncture inhibits hypertension effect, we started to study acupuncture effect on hypotension or shock.

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Experimental Studies

In 1983 Xiao and Li began to study acupuncture treatment on experimental low blood pressure [2]

They injected sodium nitroprusside (NP) slowly into the vein of conscious dogs 30µg/kg/min. Dog's Systolic Blood Pressure (SBP) decreased from 116±6 to 78±6 mmhg, Heart Rate (HR) increased from 103±8 to 189±4 per minute. In the infusion period, BP could be stabled at a low level for more than an hour. After cessation of infusion the SBP and HR recovered slowly to the contrast level within 5-10 minutes. The breathing did not change significantly.

The acupuncture effect

In the above-mentioned low blood pressure dogs Electroacupuncture (EA) at St36 or P6 for 20min with low frequency and low current (1-2mA, 2Hz/min) the blood pressure recovered toward normal level within 5-10 minutes significantly. When EA stopped, blood pressure and heart rate declined again. The pressor effect of EA can also be observed in anesthetized dogs. It is worth noting that the acupoint and the stimulating parameters are same as used in the treatment of high blood pressure dogs. However, the current of EA to increase the effects of Blood Pressure (BP) in conscious dogs are lower than in the anesthetized dogs. In this series of experiments we only use St36 or P6, because in our previous experiments already show that other control acupoints such as Li6-7 or G36-39 are not effective.

Cardiac output and organ blood flow changes

When NP was injected into the conscious dogs to low BP, the cardiac output and renal blood flow are reduced considerably, but mesenteric and femoral blood flows changed only a little. When EA at St36 or P6 to increase BP; the cardiac output was increased significantly, the renal blood flow was further reduced. The mesenteric and femoral arterial blood flow had no significant change. Therefore, EA increase of BP is primarily depends on the increase in cardiac output and reduction of renal blood flow; thus results in reduced urine formation and increased water and sodium retention.

Baroreceptor reflex resetting

During EA, BP increases and the relationship between BP-HR curve in the EA period moved to the right, especially when BP is low, so there is a reset of baro-reflex during EA.

The neurotransmitters related to the pressor effect of EA

In our study, Naloxone (0.2mg/kg iv) was not able to block EA pressor effects, and morphine (0.1mg/kg iv) did not affect the NP infusion induced low blood pressure. So, the EA pressor effect is not related to the opioid system.

Central cholinergic function in the EA pressor effect [3]

In conscious dogs with normal BP application of atropine (0.15mg/kg iv) or scopolamine (0.25mg/kg iv), did not affect the low blood pressure induced by NP infusion. However, atropine and scopolamine

could block the EA pressor effect on NP induced low blood pressure. If microinjection of scopolamine or atropine into the brain around the central gray in the brainstem, or the reticular formation of midbrain, EA's pressor function could be blocked. When Acetylcholine (ACH) was microinjected into this area, a pressor effect lasts approximately 10 minutes could be shown. Injection of saline or ACH into the surrounding area would have no such effects. In the brain of the top cortex there are a lot of cholinergic neurons, but microinjection of ACH, atropine did not show significant influence on BP and the EA effect. Therefore, the cholinergic mechanism of EA pressor effect is mainly in the lower brain stem.

Because of the Deep Peroneal Nerve (DPN) is underneath the acupoint St36, and in the following experiments instead of EA we only used DPN stimulation (2-4 mA, 0.5 ms duration. 20Hz) [4].

Effect of DPN stimulation on NP infusion induced low blood pressure

After DPN stimulation the BP and left ventricular pressure LVP, dp/dt_{max} and cardiac power rings are all increased.

Effect of somatic nerve stimulation on endotoxin shock

When IV injection i.e, coli endotoxin (5mg/kg) is injected to the dog, the blood pressure was reduced considerably. After stimulation of the DPN, the BP, LVP, dp/dt_{max} and mesenteric vascular resistance are all significantly increased. There was no apparent change in heart rate.

The results show that the NP induced low blood pressure and the endotoxin induced shock in rats could be helped by the EA or DPN stimulation [5].

In another experiment, venous blood of nine rats was withdrawn to below 55 percent of total blood volume in approximately 5-10 minutes, BP dropped immediately to the reference value of 55%. The blood pressure within an hour returned to the reference value of 66 percent and no further recovery is observed. Two of the rats died after 30 minutes. If the BP down to 59% of its original value, stimulation of sciatic nerve will increase BP to 81% of the original value. When sciatic nerve stimulation is stopped, and BP will continue to remain at 80 percent of the original values for at least 2 hours. At the same time the splanchnic nerve discharge displayed in the sciatic nerve stimulation period increased. The rats were usually demonstrating good posture and drinking water. No one died in the 36-hour lab period.

Sciatica exciting charge effect could not be blocked by naloxone. However, when scopolamine is intravenous injected into these low blood pressure rats, sciatic nerve stimulation induced pressor response greatly diminished. Therefore, in such low-blood pressure model sciatic nerve stimulation induced pressor effect is also related to the activation of cholinergic system in the brain.

Brain Cholinergic Mechanism in the EA or Somatic Nerve Stimulation Induced Pressor Response [6].

In this series of experiments we used urethane-chloralose anesthetized rabbits.

The pressor effect of brain cholinergic activation

When neostigmine was injected into lateral Ventricle (ICV), the

mean BP, left ventricular pressure and renal nerve discharge all increased, but heart rate decreased significantly within five min. These changes continued for more than one hour.

- In another series of experiments with rabbits, the mesenteric arteries were occluded for one hour and then released for reperfusion (release some toxic substance). MAP dropped from 104 ± 5 mmHg to 68 ± 5 mmHg, ICV neostigmine ($200 \mu\text{g}/200 \mu\text{l}$) is injected and then the MBP returned to 77 ± 5 mmHg after 20 min, no rabbit is died. If neostigmine ICV is not injected, 2/3 rabbits died within two hour
- Microinjection of neostigmine or carbachol into bilateral RVLM, the MBP and stimulation of PAG induced pressor response increases significantly. If atropine was microinjected into RVLM, ICV neostigmine has no pressor response. Suggesting that the pressor effect induced by ICV neostigmine is related to the activation of cholinergic M-receptor in RVLM
- Stimulating superficial nerve induced pressor response is related to central cholinergic system activation [7]. Superficial Peroneal Nerve (SPN) contains more type IV fibers. Stimulation of SPN (0.1-0.3 mA, 5-10 Hz, 0.5 ms) the MBP and pressor response (MBP max) induced by stimulation of dorsal PAG increased significantly. If atropine was microinjected into RVLM, the MBP and MBP max decreased significantly
- Thus, SPN stimulation could activate cholinergic system in the brain, increase sympathetic activity and defense reaction related cardiovascular response. EA and somatic nerve stimulation could increase BP in low BP animals
- Change of brain cholinergic concentration during defense area or SPN stimulation. During stimulation of defense area or SPN, the Ach concentration of perfusion fluid from ventricle and RVLM increased markedly, and returned to normal level after stop stimulation for 30 min. Modulation of neurally mediated vasodepression and bradycardia by electroacupuncture through opioids in nucleus tractus solitaries [8]
- Stimulation of vagal afferent endings with intravenous Phenylbiguanide (PBG) causes both bradycardia and vasodepression, simulating neurally mediated syncope. Activation of μ -opioid receptors in the Nucleus Tractus Solitarius (NTS) increases blood pressure. Electroacupuncture (EA) stimulation of somatosensory nerves underneath acupoints P5-6, ST36-37, LI6-7 or G37-39 selectively but differentially modulates sympathoexcitatory responses. We therefore hypothesized that EA stimulation at P5-6 or ST36-37, but not LI6-7 or G37-39 acupoints, inhibits the bradycardia and vasodepression through μ -opioid receptor mechanism in the NTS. We observed that stimulation at acupoints P5-6 and ST36-37 overlying the deep somatosensory nerves and LI6-7 and G37-39 overlying cutaneous nerves differentially evoked NTS neural activity in anesthetized and ventilated animals. Thirty-min of EA-stimulation at P5-6 or ST36-37 reduced the depressor and bradycardia responses to PBG while EA at LI6-7 or G37-39 did not. Congruent with the hemodynamic responses, EA at P5-6 and ST36-37, but not at LI6-7 and G37-39, reduced vagally evoked activity of cardiovascular NTS cells. Finally, opioid receptor blockade in the NTS with naloxone or a specific μ -receptor antagonist reversed. P5-6 EA-inhibition of the depressor, bradycardia and vagally evoked NTS activity. These data suggest that point specific EA stimulation inhibits PBG-induced vasodepression and bradycardia responses through a μ -opioid mechanism in the NTS.

Conclusion

EA and somatic nerve stimulation can raise the low BP induced by iv NP, hemorrhagic, endotoxic shock, and PBG induced bradycardia and low BP. It is related to the activation of cholinergic activity in the hypothalamus and lower brain stem, including NTS.

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