Intensive Care Med (1987) 13:203–207

Intensive Care Medicine

Hemorrhagic shock with paradoxical bradycardia

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Received: 1 October 1985; accepted: 26 June 1986

Abstract. Two hundred and seventy-three acute hemorrhagic shocks were treated in 1984 in a pre-hospital emergency care unit. Twenty patients (7%) had a paradoxical bradycardia: they were conscious, 9 of them had an undetectable systolic arterial pressure with the sphygmomanometric method but the femoral pulse was still present. All of them recovered from bradycardia with fluid loading alone. The comparison between patients with paradoxical bradycardia and those with tachycardia showed that the former had more severe and rapid hemorrhages. During 1985, 7 new cases of acute hemorrhagic shock with paradoxical bradycardia were treated with an antishock trouser. These patients recovered from bradycardia more quickly ($p < 0.01$) and with a less important fluid loading ($p < 0.01$) than those previously treated without the antishock trouser. Two other patients were treated with atropine before antishock trouser inflation and experienced ventricular premature beats and one developed ventricular fibrillation. A paradoxical bradycardia can occur in hemorrhagic shock and denotes a rapid and severe hemorrhage requiring a massive and rapid fluid loading. The preliminary results of the antishock trouser in this setting are encouraging. The treatment of bradycardia per se may be deleterious and atropine must be avoided in conscious patients with hemorrhagic shock and paradoxical bradycardia.

Key words: Hemorrhagic shock – Acute hypovolemia – Bradycardia – Antishock trouser

Heart rate usually increases in hypovolemic shock: parasympathetic inhibition and sympathetic activation are the two components of the reflex tachycardia induced by hypovolemia [16]. Vasovagal syncope with bradycardia has been well described in healthy volunteers [1], and bradycardia is sometimes observed in unconscious patients with dramatic hemorrhagic shock and undetectable blood pressure, at the point of death: bradycardia just precedes cardiac arrest. Very few reports have emphasized the occurrence of bradycardia in conscious patients with hemorrhagic shock [4, 12] and most authors have considered the paradoxical bradycardia to be similar to the vasovagal faint. We have studied the occurrence of bradycardia in patients with acute hemorrhagic shock (AHS). Thereafter, antishock trousers [5] were used, and treatment with atropine was evaluated.

Methods and patients

The study was performed during 1984 in a pre-hospital emergency care unit. Criteria for inclusion were evidence of external or internal (afterwards confirmed at the hospital) hemorrhage, initial systolic arterial pressure (SAP) $\leqslant$ 70 mmHg, no head or spinal injury. Patients initially with cardiocirculatory arrest as established by a physician were excluded. Patients previously taking medication known to cause a bradycardia were excluded. Age, time between emergency-call and physician's arrival, time of physician's care before admission to the hospital, heart rate (HR) and SAP measured initially and finally at the hospital, consciousness, treatment administered, and the outcome of patients after admission to the hospital were recorded. Consciousness was defined as a Glasgow's coma score $\geqslant$12. When SAP was undetectable using sphygmomanometer then the femoral pulse was noted as being present or absent. When bradycardia was observed an electrocardiogram was performed and during the last part of the study the HR was recorded every 5 min

* This study has been presented in part at the 3rd International Symposium on Applied Circulatory and Respiratory Physiology in Anesthesia and Intensive Care, Rotterdam, 27–29 November 1985

until arrival at the hospital. Whenever possible survivors were tested with simple tests of parasympathetic functions [3]: upright tilt, carotid massage, oculocardiac reflex. Parasympathetic functions were considered within normal limits when carotid massage and the oculocardiac test decreased HR between 10 to 20 bpm and when upright tilt decreased SAP less than 10 mmHg. Patients received no cardiovascular drugs and atropine was carefully avoided in conscious patients with paradoxical bradycardia. Fluid loading was usually performed with 2 or more teflon catheters (14 to 16 Gauge, $1\frac{1}{4}$ inch − 3 cm), using colloids (Plasmion, Plasmagel); central venous catheters were rarely used.

Since March 1985, an antishock trouser (Gladiator Antishock Pants, JOBST Ltd) was used in new cases of paradoxical bradycardia in AHS. Fluid loading was always initiated within 5 min of the physician's arrival and the antishock trouser (AST) was inflated between the 5th and the 10th min. The inferior limb compartments of the AST were inflated to 80 mmHg and the abdominal compartment to 60 mmHg. HR was recorded every 5 min until arrival at hospital. During the same period atropine was also used but was administered only after fluid loading was begun and the AST was in place but not inflated.

Statistical analysis

Whenever appropriate, group means were compared by Student's t test and proportions by Fisher's exact method. When parametric hypothesis could not be assessed, Mann and Whitney's U test was performed. A p value < 0.05 was necessary for rejection of a null hypothesis. Group data are expressed as the mean ± standard error of the mean (m ± sem).

Results

Two hundred and seventy-three patients met the criteria of severe AHS most were secondary to road accidents (197), violence with side or fire-arms (47), and others were due to non traumatic hemorrhage (ectopic pregnancy, gastroduodenal hemorrhage, ruptured aortic aneurysm).

Two hundred and forty-eight patients had an adequate tachycardia (HR: 151 ± 1 bpm) usually found in AHS and defined group T. Twenty-five patients had an absolute bradycardia (HR $\leqslant$ 60 bpm) and none had a relative bradycardia (HR 60 to 100 bpm). Among these 25 patients, 2 distinct clinical groups existed. 5 patients were unconscious, with undetectable blood pressure, no femoral pulse but non-dilated pupils and electrial activity on the electrocardiogram. These 5 pa-

Table 1. Clinical and hemodynamic characteristics of 20 patients with acute hemorrhagic shock (AHS) and paradoxical bradycardia

Patient (no)	Sex	Age (years)	Etiology of AHS	HR initial-final (bpm)	SAP initial-final (mmHg)	Fluid loading (l)	Outcome after admission to the hospital
1	F	21	EP	50 − 140	F − 50	3.0	Dead
2	M	29	H(hv)	60 − 160	50 − 60	3.5	Dead
3	F	25	S	55 − 150	F − 60	3.0	Alive
4	F	27	EP	60 − 140	60 − 70	4.0	Alive
5	M	35	H	60 − 140	50 − 80	3.0	Alive
6	M	19	H(hv)	60 − 160	F − 60	3.5	Dead
7	M	18	S	45 − 130	F − 70	3.5	Alive
8	M	26	H	50 − 150	70 − 50	3.5	Alive
9	M	38	AWL	50 − 140	60 − 60	4.0	Alive
10	M	34	AWL	60 − 140	F − 60	4.5	Alive
11	F	32	EP	55 − 150	50 − 60	4.0	Alive
12	M	42	H(hv)	50 − 160	F − 70	3.0	Dead
13	F	30	AWL	45 − 150	F − 80	3.5	Alive
14	M	35	S	60 − 160	F − 60	5.0	Alive
15	M	43	H	60 − 160	60 − 70	3.5	Alive
16	M	39	AWL	60 − 140	70 − 70	3.0	Alive
17	M	21	AWL	40 − 150	60 − 60	3.0	Alive
18	M	41	H	55 − 120	60 − 80	3.0	Alive
19	F	20	S	60 − 140	F − 60	3.0	Alive
20	M	22	T	60 − 70	50 − 70	3.0	Alive
m ± sem		30 ± 2		55 − 142 ± 1 ± 5	/ − 65 ± 1	3.5 ± 0.1	

S: splenic trauma, H: hepatic trauma, hv: rupture of hepatic veins, EP: ectopic pregnancy, AWL: arterial wound of the limbs, T: thoracic trauma, HR: heart rate, SAP: systolic arterial pressure, F: presence of femoral pulse. Data are expressed as m ± sem

Table 2. Comparison between patients with acute hemorrhagic-shock (AHS) and tachycardia (group T), or paradoxical bradycardia (group B)

	Group T $n = 248$ No (%)	Group B $n = 20$ No (%)
Age (years)	37 ± 1	30 ± 2[a]
Sex (F – M)	88 – 160 (35 – 65)	6 – 14 (30 – 70)
Undetectable initial SAP	52 (21)	9 (45)[a]
SAP at the hospital (mmHg)	67 ± 1	65 ± 1
HR at the hospital (bpm)	140 ± 1	142 ± 2
Timing before care (min)	13.1 ± 0.2	12.2 ± 0.6
Timing of care before hospital (min)	62.6 ± 3.5	57.5 ± 2.1
Fluid loading (l)	3.31 ± 0.03	3.48 ± 0.13
Death	20 (9)	4 (20)
Etiology of AHS		
Abdominal trauma	42 (17)	11 (55)[a]
Thoracic trauma	68 (27)	1 (5)[a]
Limbs and pelvic trauma	115 (46)	0 (0)[a]
Arterial wounds of the limbs	12 (5)	5 (25)[a]
Ectopic pregnancy	7 (3)	5 (25)[a]
Others	19 (8)	0 (0)

SAP: systolic arterial pressure, HR: heart rate. Statistical analysis performed by Student's test or Fisher's exact method. [a]: $p < 0.05$. Data are expressed as m ± sem, or number of patients (No) followed by percentage in brackets

tients constituted the dying patients group D and all required cardiopulmonary resuscitation associated with epinephrine and atropine infusion, they died before arrival at the hospital. Twenty patients fulfilled the criteria for paradoxical bradycardia and constituted group B (7% of AHS – 95% confidence interval: 4 – 10%). They were all conscious, had no previous cardiac disease, received no medication, and bradycardia was noted before any invasive care. In 9 out of 20 patients SAP could not be determined but a femoral pulse was still present (data are summarized in Table 1). ECGs showed sinus rhythm without major ST–T changes. Patients recovered from bradycardia after fluid loading, only one had a persistent relative bradycardia at the time of admission, a few minutes later, however, after a larger fluid loading the HR was 130 bpm. Only 2 patients were intubated: patient 12 because he became unconscious and patient 15 because of restlessness requiring sedation. Only one deep venous catheter was placed. Table 2 compares groups T and B. Group D was excluded from the comparison since it did not fulfill the criteria. Patients from group B had AHS due to rapid and massive hemorrhage (hepatic trauma with hepatic vein rupture, ectopic pregnancy, arterial wounds) whereas most

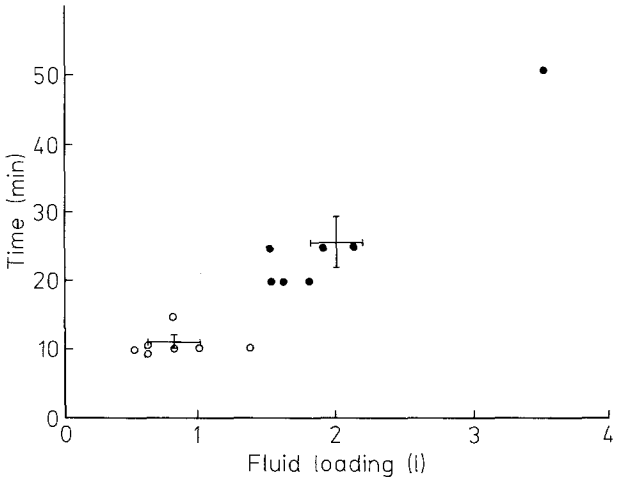

Fig. 1. Comparison of the recovery from paradoxical bradycardia in patients with acute hemorrhagic shock and treated with (○) or without the antishock trouser (●) $p < 0.01$. Recovery is defined as heart rate ⩾ 100 bpm. Time is time after physician arrival. Fluid loading was begun before the 5th min, and AST was inflated between the 5th and the 10th min. Mann und Whitney's U test

AHS in group T were due to pelvic and limb trauma responsible for a more gradual hemorrhage. The number of patients with undetectable SAP was greater in group B. Probably because they experienced more severe AHS.

Five surviving patients from group B agreed to be tested several months after they were discharged from hospital; tests of parasympathetic function, electrocardiogram, and blood pressure were within normal limits.

Treatment with AST

Since March 1985, AST were used in 7 new cases (Table 3). As shown in Fig. 1, patients treated with AST recovered more quickly (11 ± 1 min vs 26 ± 4 min in group T) and with less fluid loading (0.83 ± 0.13 L. vs 2.00 ± 0.26 L. in group T). In patient 4 the AST was accidentally deflated and HR then decreased from 125 to 45 bpm; after reinflation HR was 110 bpm. All patients recovered from bradycardia within 5 min of AST inflation.

Treatment with atropine

From March 1985, atropine was used in 2 cases (Table 3), patient 8 experienced multiform and repetitive ventricular premature beats immediately after atropine (1 mg). No change in SAP was observed after atropine infusion and thus AST were inflated. Patient 9 experienced multiform and repetitive premature ventricular beats and then ventricular fibrillation immediately after atropine (1 mg). External countershock was administered, AST was inflated, and the patient was successfully resuscitated.

Table 3. Clinical and hemodynamic characteristics of patients with acute hemorrhagic shock (AHS) and paradoxical bradycardia, and treated with antishock trousers (patients 1 to 7) and with atropine (patients 8 and 9)

Patient (no)	Sex	Age (years)	Etiology of AHS	HR initial-final (bpm)	SAP initial-final (mmHg)
1	M	23	AWL	65–120	F–50
2	M	32	S	60–120	50–65
3	F	37	S+P	50–130	45–65
4	M	28	AWL	55–130	F–60
5	M	23	H	40–130	F–50
6	F	18	RPH+P	60–135	50–60
7	M	37	S	55–150	F–60
8	F	23	EP	45–130	50–60
9	F	32	H	60–150	F–50

HR: heart rate, SAP: systolic arterial pressure, F: presence of a femoral pulse, AWL: arterial wound of the limbs, S: splenic trauma, H: hepatic trauma, P: pelvic and limbs trauma, RPH: retroperitoneal hemorrhage, EP: ectopic pregnancy

Discussion

All patients from group B had AHS and paradoxical bradycardia with mechanical activity of the heart sufficient to maintain adequate cerebral blood flow since they were conscious. None had taken drugs known to induce bradycardia and none had previous cardiac disease. The simple tests performed in surviving patients suggested that they had normal parasympathetic function. Paradoxical bradycardia is clearly related to an extreme and rapid blood loss and the ensuing hypovolemia: (1) was observed in the most severe and rapid cases of blood loss; (2) recovery occurred with correction of hypovolemia by massive fluid loading or AST; it is remarkable that the HR of patients from group B on arrival at hospital was not different from that in group T; (3) patients treated with AST recovered more quickly and AST are known to induce a rapid correction of hypovolemia [5]; (4) the accidental deflation of the AST in patient 4 also demonstrated the relationship between extreme hypovolemia and paradoxical bradycardia, and its reversible feature. Our study demonstrated that paradoxical bradycardia existing in AHS, was not related to irreversible forms of shock but to extreme and rapid hypovolemia. Paradoxical bradycardia supervened in 7% of AHS as defined.

Barcroft et al. [1] have described vasovagal faints in acute hemorrhage with both bradycardia and vasodilation inducing loss of consciousness. In late severe AHS, bradycardia and vasodilation overpowering normal vasoconstriction has been reported [2] and is considered a reliable index of the onset of irreversible shock. The literature contains few studies of relative bradycardia during AHS; Jansen [4] and Secher [12] each reported 4 cases. Pavek and Wegmann [11] found that bradycardia sometimes occurred during anaphylactic shock and seemed to relate to the severity but not to the degree of hypovolemia. Secher and Bie [13] in a review concluded that when blood pressure falls in AHS a decrease in HR and in peripheral resistance is the rule, and that bradycardia in AHS is remarkably similar to events associated with a vasovagal faint. We believe that it is unlikely that our patients experienced a vasovagal faint since this would have certainly induced loss of consciousness and rapid cardiac arrest. Some patients treated with AST were conscious enough to tell the physician that inflation of AST was uncomfortable. These data suggest that systemic resistance was not decreased, allowing sufficient cardiac output and adequate cerebral blood flow. The hypothesis of high sympathetic activity in paradoxical bradycardia responsible for high systemic resistance is supported by the results of atropine administration: in our 2 cases atropine infusion was immediately followed by ventricular premature beats and ventricular fibrillation in one case suggesting a high sympathetic activity previously masked by parasympathetic activity.

Paradoxical bradycardia has not been reported in experimental study in man. Murray et al. [7] have used the application of a negative pressure to the lower body to determine a reproducible hypovolemia and to effect vasovagal syncope. In their study, when HR fell, then arterial pressure and systemic resistance also decreased inducing syncope. In these experiments of graded hypovolemia, arterial pressure was not decreased until the vasovagal faint; thus, the sympathetic tone would have been certainly not as high as in AHS with low arterial pressure.

Bradycardia has been reported rarely in experimental studies and most of the experiments conducted in anesthetized animals in which parasympathetic tone is reduced and sympathetic tone intensified before any reduction in arterial pressure [16]. Overall, hemorrhage is usually carried out slowly. When circulatory effects of rapid hemorrhage were studied, then paradoxical bradycardia occurred and could be reversed to a marked tachycardia with retransfusion of part of the shed blood [9]. Little et al. [6] have also described bradycardia in experimental AHS in rats when some 25% of the blood volume was withdrawn. Moreover, when tissue ischaemia was superimposed on blood loss, more blood had to be removed to induce a bradycardia [6]. This might explain why some patients did not experience paradoxical bradycardia in AHS in spite of a rapid and massive hypovolemia.

The pathophysiology of paradoxical bradycardia in AHS is not clearly understood. Öberg et al. [9] considered that paradoxical bradycardia might be a protective reflex enabling improvement in diastolic ventricular loading in massive and rapid hypovolemia. Bradycardia in AHS in cats was eliminated by either cooling the cervical vagi, atropine administration, or cutting cardiac vagal nerves, all demonstrating that a vago-vagal reflex loop was responsible [9]. A previous study suggested that the fibers concerned with this reflex are not tonically active vagal afferents [8]. Öberg and Thoren [10] suggested that this bradycardia originated from left ventricular mechanoreceptors in non-medullated vagal afferents. The involvement of non-medullated C-fibre afferents in the cardiac nerve is also supported by experiments in rats treated with capsaicin which selectively destroys these fibres: more blood has to be removed to elicit bradycardia in capsaicin-treated rats [14].

Atropine was not used in conscious patients in the first part of this study and all patients recovered from bradycardia with fluid loading alone. HR in patients with paradoxical bradycardia seems to be a reliable index of hypovolemia correction and atropine might mask inadequate fluid loading. A high HR is probably not suitable in AHS with massive hypovolemia and poor diastolic filling of the heart. Secher et al. [12] have reported on one fatal outcome in a patient with paradoxical bradycardia who was given epinephrine resulting in a higher HR but unaffected blood pressure. They concluded that treatment of bradycardia per se may be fatal. Moreover, our study suggested that atropine can induce ventricular arrhythmias because of a high concomitant sympathetic activity.

The treatment of paradoxical bradycardia in AHS is not the treatment of bradycardia per se but rapid and massive fluid loading. AST seem useful because the recovery from bradycardia is achieved rapidly and with lower fluid loading. AST inflated at high pressure (60 to 80 mmHg), that is often above SAP in severe AHS, corrects hypovolemia by 'autotransfusion' of venous blood pooled below the diaphragm to the arterial circulation above it, by increasing the systemic resistance, and reducing the blood volume returned to the lower extremities, and by reducing any hemorrhage below the diaphragm [5].

Conclusion

We suggest that paradoxical bradycardia is an important sign of major and rapid hemorrhage, requiring massive and rapid fluid replacement. The correction of bradycardia seems to be a reliable index of correction of hypovolemia. Preliminary results of AST in this setting are encouraging. The treatment of bradycardia per se is not indicated and may be deleterious. Atropine must be avoided in conscious patients with paradoxical bradycardia, it is indicated in vasovagal faint.

Acknowledgement. We are indebted to Professor André Lienhart, Department of Anesthesiology, Hôpital Saint-Antoine (Paris), and to Didier Payen, MD, Department of Anesthesiology, Hôpital Lariboisière (Paris), who reviewed the manuscript and provided valuable advice.

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