

A240

TITLE : HEMODYNAMIC EFFECTS OF DOBUTAMINE DURING SEPTIC SHOCK TREATED BY NOREPINEPHRINE.
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After failure of volume expansion and dopamine infusion in septic shock, norepinephrine (NOR) is administered to restore mean arterial pressure (MAP).¹ Because of myocardial depression during septic shock, dobutamine (DOB) could increase cardiac index (CI) and hence oxygen delivery (DO₂).² The aim of our study was to investigate the hemodynamic effects of DOB in patients with septic shock already treated by NOR.

Methods: After approval of local Ethic Committee ten patients with septic shock aged 62 ± 13 yrs (mean ± S.D.), were included in this study. Dose of NOR was 1.48 ± 0.54 µg.kg.⁻¹.min⁻¹ to maintain MAP between 70 and 90 mmHg. Hemodynamic parameters were measured and calculated before DOB infusion (0), after increasing doses of DOB (5, 7.5, 10, 15 µg.kg.⁻¹.min⁻¹) and 15 min after DOB was stopped (0bis). During DOB infusion, volume expansion (Albumin 4%) maintained baseline filling pressures. Values were expressed as mean ± SD. Statistics were ANOVA followed by Scheffe-F-test. p<0.05 was considered as significant.

Results: They are summarized on the table. DOB at 10 µg.kg.⁻¹.min⁻¹ decreased systemic vascular resistances (SVRI). At this dose, heart rate (HR) greatly increased in 2 patients with atrial fibrillation in one. In all patients but these two, lowering of SVRI was maintained at 15 µg.kg.⁻¹.min⁻¹. Volume expansion during DOB infusion was 1011 ± 195 ml. When DOB was stopped, pulmonary wedge pressure (Pwp) was higher than before DOB was started. All the other hemodynamic parameters remained unchanged (table).

Conclusion : DOB did not increase CI, DO₂ and oxygen consumption (VO₂). DOB induced arrhythmias. We concluded that DOB offered no advantages when CI is maintained at its upper limits with NOR during septic shock.

References: 1. Ann. Int. Med, 113 : 227-242, 1990.
2. Am. Rev. Respir. Dis., 142 : 2-7, 1990.

DOB µg.kg. ⁻¹ .min ⁻¹	0	5	7.5	10	15 (n=8)	0bis
HR b.min ⁻¹	115 ± 15	121 ± 17	122 ± 16	130 ± 23	126 ± 18	116 ± 21
MAP mmHg	78 ± 6	71 ± 13	74 ± 16	69 ± 14	73 ± 13	78 ± 12
Pwp mmHg	7.9 ⁺ ± 0.7	7.4 ⁺ ± 2.1	7.3 ⁺ ± 1.9	8.1 ± 2	8.3 ± 1.7	9.9 ± 1.7
CI l.min ⁻¹ .m ⁻²	4.8 ± 1.5	4.7 ± 1.4	5.2 ± 1.4	5.0 ± 1.6	5.4 ± 1.4	4.9 ± 1.6
SVRI dynes.cm ⁻⁵ .m ⁻²	1285 ± 341	1169 ± 326	1106 ± 397	1026* ± 215	1019* ± 247	1217 ± 345
VO ₂ mlO ₂ .min ⁻¹ .m ⁻²	154 ± 40	147 ± 51	164 ± 39	145 ± 41	145 ± 37	157 ± 36
DO ₂ mlO ₂ .min ⁻¹ .m ⁻²	685 ± 178	643 ± 174	697 ± 163	639 ± 198	683 ± 174	632 ± 177
Lactates mmol.l ⁻¹	4.9 ± 2.4	4.8 ± 2.6	4.6 ± 2.6	4.7 ± 2.7	5.0 ± 3.1	4.4 ± 2.6

moy ± SD; * p<0.05 vs 0; ⁺ p<0.05 vs 0 bis.

A241

Title: NOREPINEPHRINE (NOR) VS EPINEPHRINE (EPI) IN SEPTIC SHOCK.
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After failure of volume expansion and dopamine infusion in septic shock, NOR is administered to restore mean arterial pressure (MAP).¹ EPI has been recently advocated for this purpose instead of NOR.^{2,3} This study was designed to compare NOR with EPI and to show if the latter may improve oxygen uptake (VO₂) by improving oxygen delivery (DO₂).

Methods: After approval of our local Ethic Committee, 16 patients with septic shock were included (age 63±13 yr, mean±SD). The following hemodynamic parameters were collected: HR, MAP, diastolic arterial pressure (DAP), RAP, PWP, CI, VO₂ and DO₂ were calculated from hemodynamic and metabolic data. Arterial lactate were measured. Patients were randomized in two groups, to receive firstly NOR (group I) or EPI (group II). After at least 1 to 3 hours of hemodynamic stability with either NOR or EPI, hemodynamic and metabolic parameters were recorded (T0). Then in group I EPI was substituted to NOR and inversely in group II. After one hour of cardiovascular stability, similar recordings were done. Values are expressed as means ± SD, comparison within groups were made with paired Student t test.

Results: Mean doses of catecholamines (in µg.kg.⁻¹.min⁻¹) were: in group I, NOR: 1.55±0.8, EPI: 0.89±0.7, in group II, EPI: 0.82±0.6, NOR: 1.37±0.7. Main results are summarized for 14 patients in the table. Two patients in group I were excluded because of atrial fibrillation after substitution by EPI.

	NOR Gr I	EPI Gr I	EPI GR II	NOR GR II
HR b/min	100±18	111±18 **	115±19	108±13
CI l.min ⁻¹ .m ⁻²	3.4±0.9	4.5±2 *	4.8±1.8	5.2±1.6
MAP mmHg	76±11	71±11	76±12	79±10
DAP mmHg	59±10	52±9 *	52±10	59±7 +
PWP mmHg	10.6±3	10±2	11.7±4	8.6±3 +
DO ₂ ml.min ⁻¹ .m ⁻²	430±140	577±290 *	585±166	644±167
VO ₂ ml.min ⁻¹ .m ⁻²	121±47	120±58	160±29	156±19
Lactate mmol/l	6±4.4	7.6±5	5.1±2.6	5.1±2.2

* p<0.05 and ** p<0.01 EPI vs NOR in group I, + p<0.05 NOR vs EPI in group II.

Conclusion: EPI after NOR in group I resulted in significant increases in HR and CI due to β adrenergic effect without improvement in VO₂ despite increase in DO₂; the latter phenomenon was observed by ³ but not by ². The low DAP observed in both group under EPI was probably due to a vasoconstriction relatively less important with EPI than with NOR. It could be detrimental for organ perfusion especially for coronary and cerebral perfusion. Thus, there is no evidence for EPI use (lower DAP, arrhythmogenicity) during septic shock in the absence of severe cardiogenic impairment.

References: 1. Ann. Int. Med., 113, 227-242, 1990.
2. Chest, 98, 949-953, 1990.
3. Intens. Care. Med.; 17, 36-39, 1991.