Simulated aeromedical evacuation after severe injuries in a small animal model

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Disclaimer

The authors have no commercial associations that might create a conflict of interest.

The study protocol was approved by the Walter Reed Army Institute of Research/Naval Medical Research Center Institutional Animal Care and Use Committee.

Background

Hemorrhage and traumatic brain injury are common injuries in recent conflicts. They can be lethal if left unattended.

Important complications could develop for severely wounded casualties upon aero-evacuated to higher care.

Also, this could become a serious concern at the time of mass casualty evacuation or for prolonged field care scenarios when resources might be limited.

We addressed these questions in a pre-clinical polytrauma injury model.

Background

Aeromedical evacuation consists of various physical events that could affect the physiology of the wounded.

- Decrease of barometric pressure in altitude
- Decrease of partial pressure of oxygen
- Decrease of temperature

By flying in an pressurized aircraft at 2440m (8000 ft).

- The barometric pressure falls from 760 to 570 mmHg
- The PaO_2 is reduced from 160 to 120 mmHg; this corresponds to a 76% of surface equivalent without O_2 supplement.

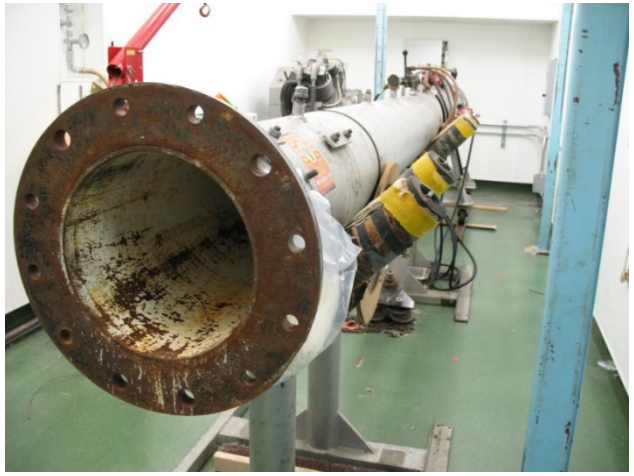
Background

We studied the impact of hypobaria (altitude) as a component of aeromedical evacuation in a polytraumatized rat model when blood is not available, evacuation vehicles may not be equipped with necessary oxygen.

This simulates evacuation in the least optimal environment.

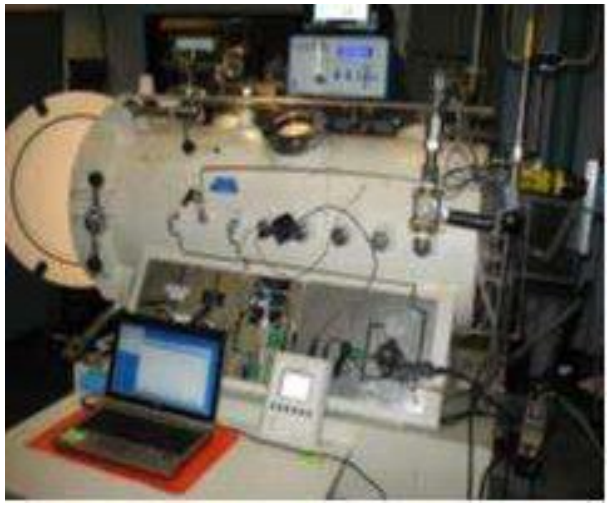
Methods

Anesthetized rats (~350 g) were either non-injured (NON-INJ) or injured (INJ).



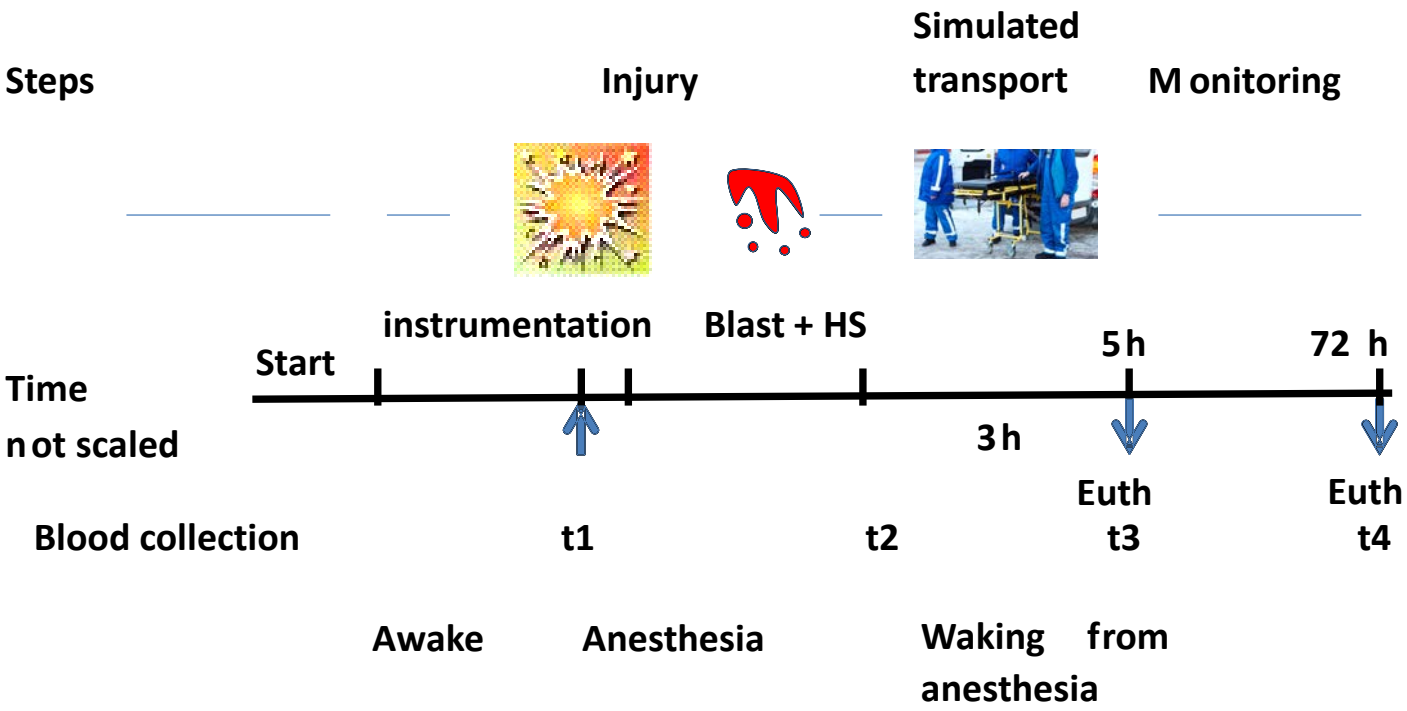
The injury consisted of a 75kPa blast followed by a 30% hemorrhage.

After 15 minutes shock, all rats received 2:1 0.9% sodium chloride followed by the simulated evacuation either at sea level or at 2440 m in an hypobaric chamber.



The chamber was flushed with 21% O_2

Methods



Schematic diagram for experimental design:
t1: After instrumentation
t2: After polytrauma
t3: After 3 hours evacuation
t4: After 3 day following evacuation

Methods

Groups:

t1	**t2**		**t2**	
	No Injury		Injury	
Normobaria	**t3**: 3h	**t4**: 72h	**t3**: 3h	**t4**: 72h
Hypobaria	**t3**: 3h	**t4**: 72h	**t3**: 3h	**t4**: 72h

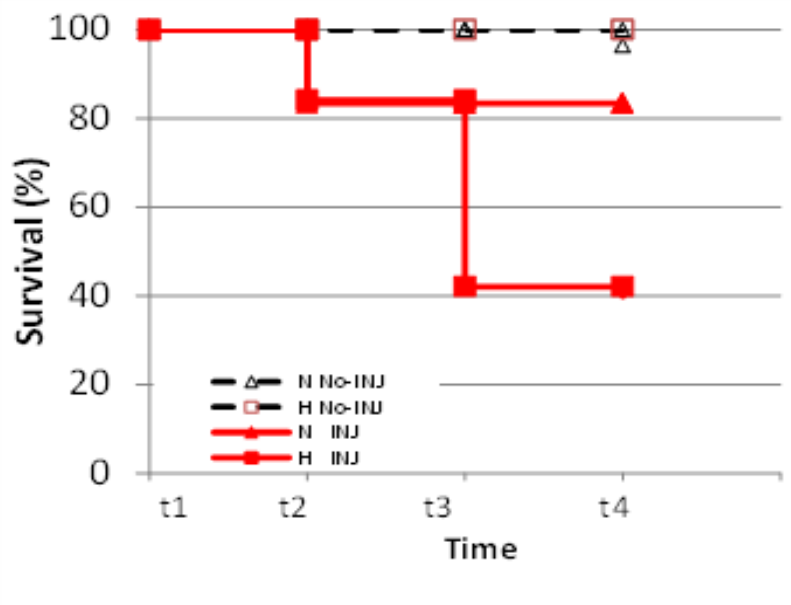
Methods

Physiology: MAP, HR, sPO2, temperature.

Metabolic parameters: hematology, blood gases, chemistry.

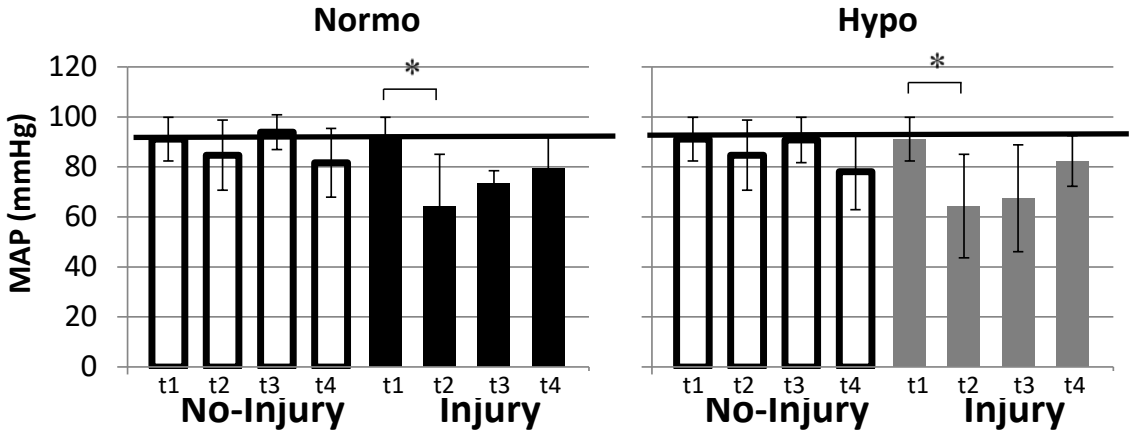
Inflammation markers

Results



Survival is reduced by 16% after injury
Survival is reduced by 50% under hypobaria
Animals died between 130 and 160 mins

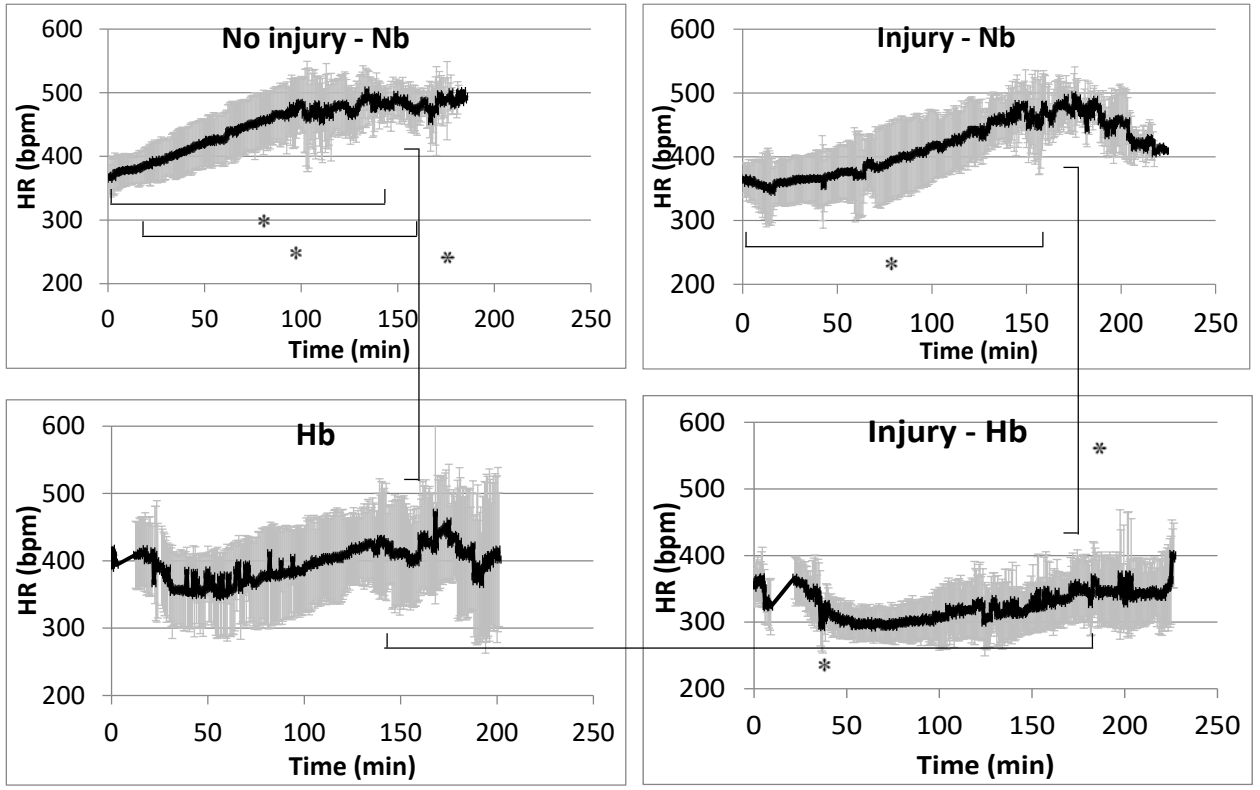
Results



MAP is reduced following injury at t4

No difference between normo and hypobaria

Results

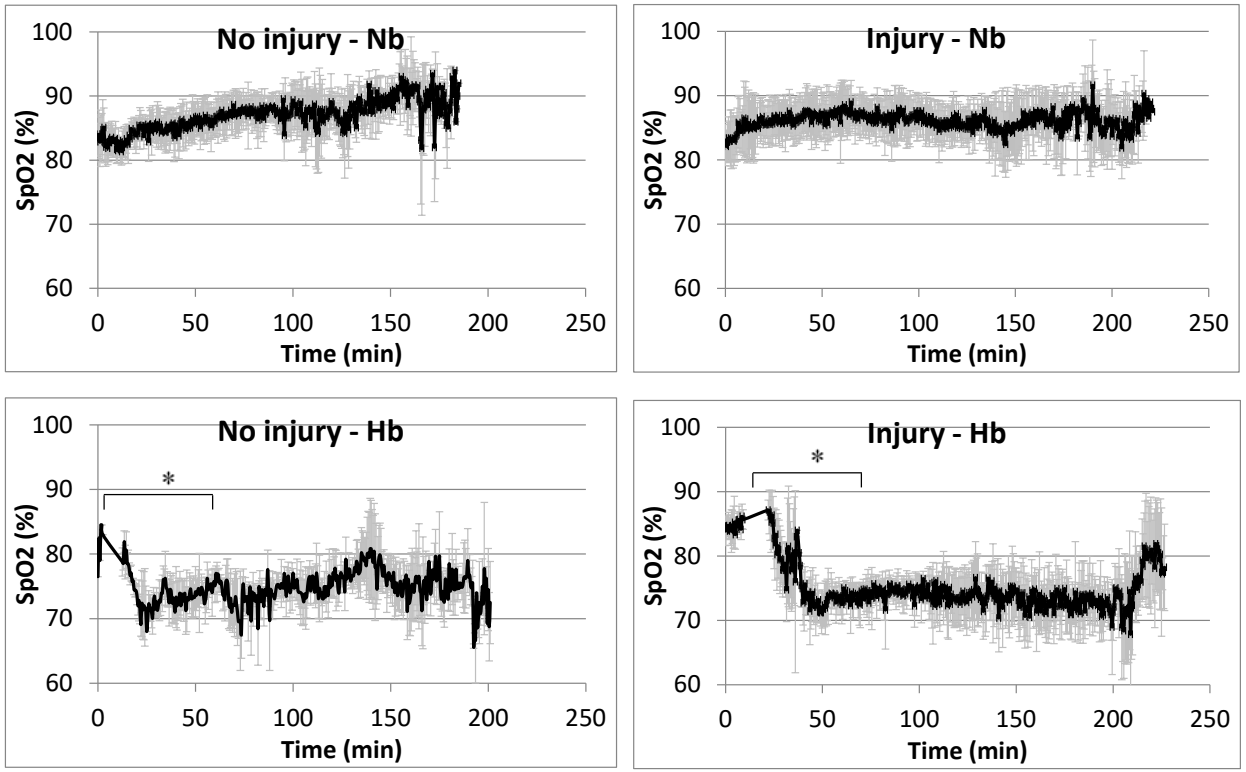


At t3 Normo: no effect of injury

Hypobaria: HR decreased initially and was lower after injury 346 ± 54 bpm vs 293 ± 24 bpm $p<0.01$

At t3 HR remained lower after injury.

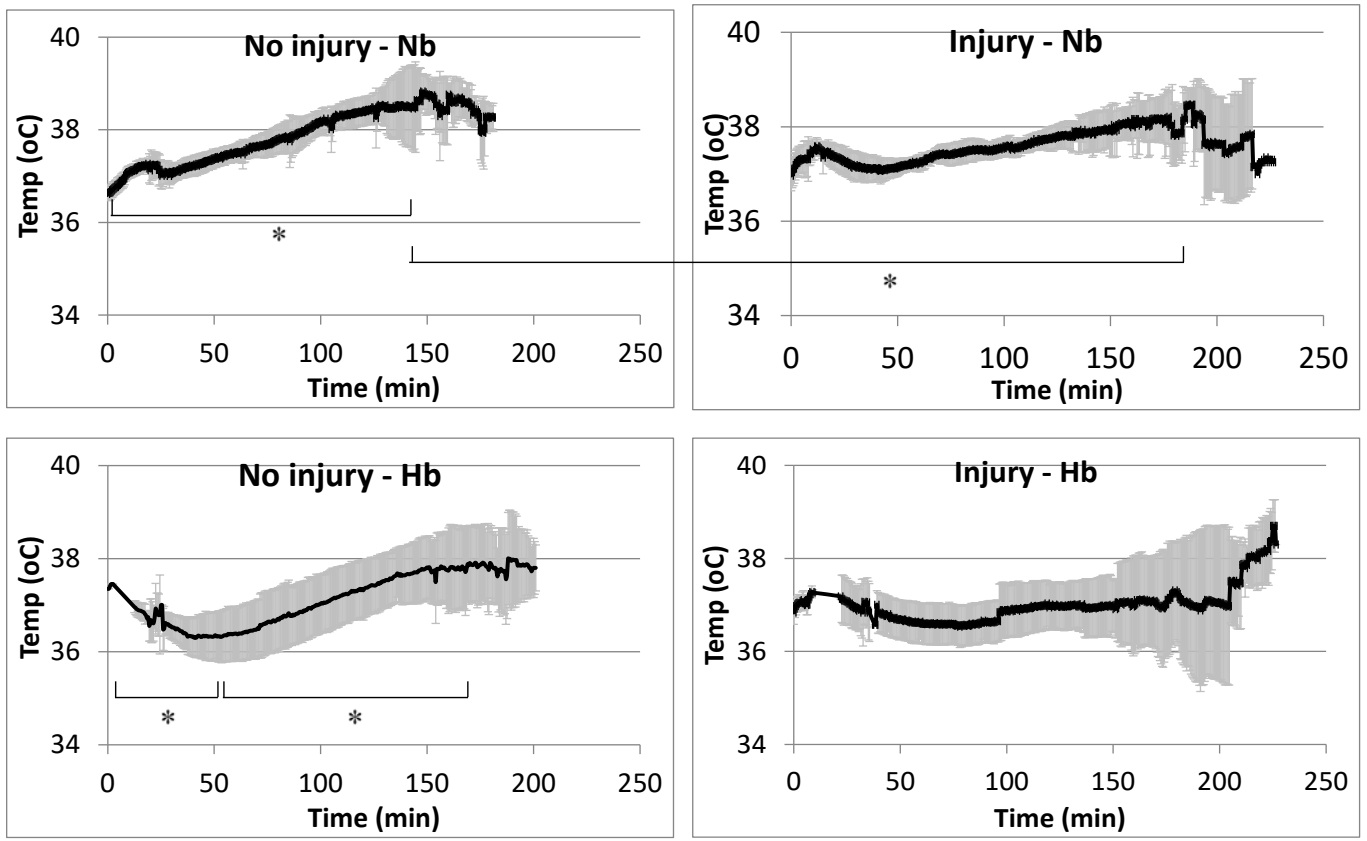
Results



At t3 Normo: no effect of injury

Hypo SpO_2 decreased initially to ~ 74%.

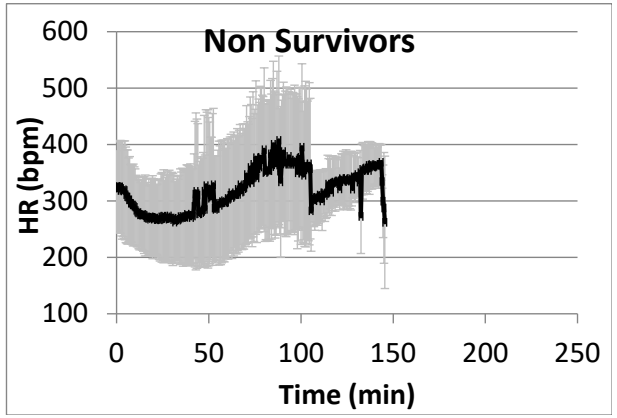
Results

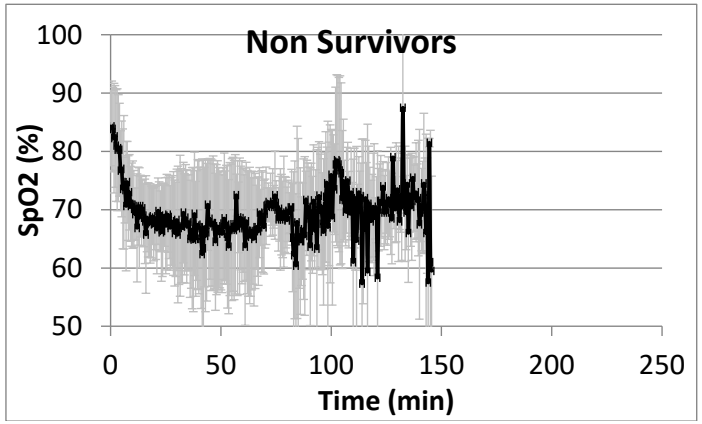


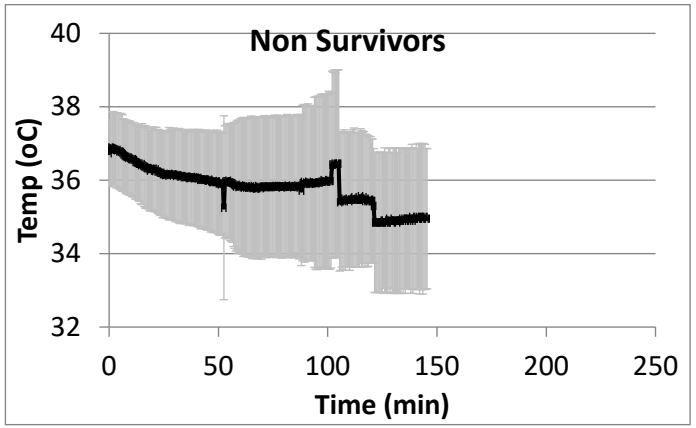
During the simulated hypobaric evacuation, the body temperature decreased initially (36.4 ± 0.4 ^{o}C, $p<0.01$)

And increased thereafter if there was no injury (37.8 ± 0.4 ^{o}C).

Results





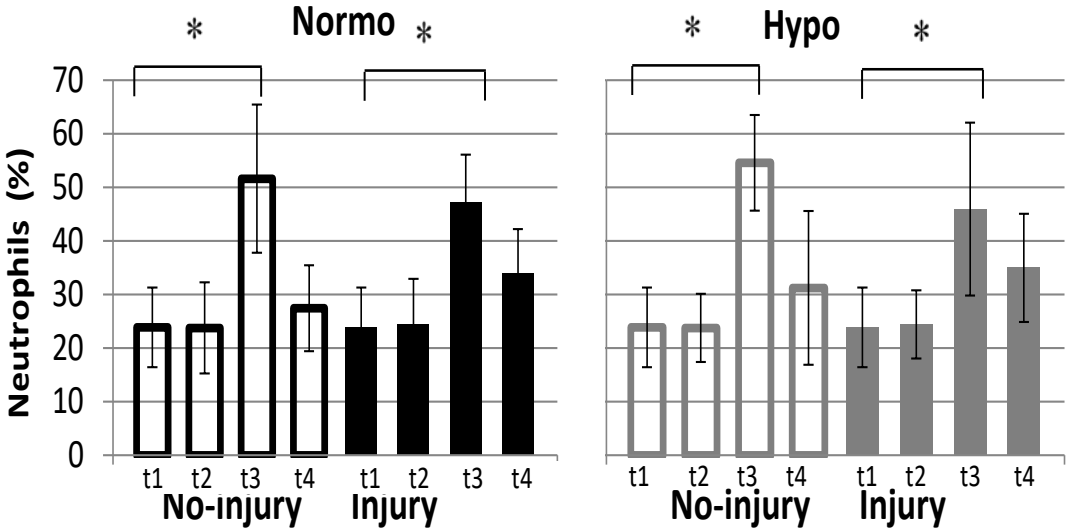


HR: overcompensation ~90 min in flight

SpO_2 :remained lower

Temp: constant decrease

Results


Normo
Hypo
*
*
*
*
Neutrophils (%)
70
60
50
40
30
20
10
0
t1 t2 t3 t4 t1 t2 t3 t4
No-injury
Injury
t1 t2 t3 t4 t1 t2 t3 t4
No-injury
Injury

Results

		TNF-a	IL-1 B	IL-4	IL-10	IL-18	IL-1 a	IFN-G	Fractalkine	GM-CSF	IL-6	RANTES	MCP-1
Nb	t1	1.19	6.33	16.07	21.61	19.18	25.42	52.10	35.10	59.21	133.60	87.06	248.70
	t2	1.11	3.03	19.97	23.19	14.67	18.31	62.13	37.27	70.27	127.75	125.86	258.97
	t3	1.38	3.77		32.27	45.89	12.63	40.99	42.22	53.96	160.92	145.93	298.23
	t4	0.92	2.13	11.06	15.36	15.46	16.41	28.36	20.86	33.14	160.92	180.18	318.35
Hb	t1	1.19	6.33	16.07	21.61	19.18	25.42	52.10	35.10	59.21	133.60	87.06	248.70
	t2	1.11	3.03	19.97	23.19	14.67	18.31	62.13	37.27	70.27	127.75	125.86	258.97
	t3												
	t4	0.85	4.64	3.14	29.87	26.48	9.93	14.13	54.35	28.65	107.19	204.34	761.77
Nb-Inj	t1	1.19	6.33	16.07	21.61	19.18	25.42	52.10	35.10	59.21	133.60	87.06	248.70
	t2	0.64	2.99	11.86	23.56	9.61	19.88	34.02	26.63	47.89	125.57	141.56	266.87
	t3	2.18	6.71	19.80	65.09	40.66	37.61	78.54	75.17	61.68	227.14	106.02	331.84
	t4	1.27	4.36	21.59	73.16	45.55	26.37	65.64	34.03	67.43	197.44	435.81	580.64
Hb-Inj	t1	1.19	6.33	16.07	21.61	19.18	25.42	52.10	35.10	59.21	133.60	87.06	248.70
	t2	0.64	2.99	11.86	23.56	9.61	19.88	34.02	26.63	47.89	125.57	141.56	266.87
	t3	2.86	9.17	30.66	117.82	83.35	56.07	98.40	79.88	104.39	112.27	344.77	315.17
	t4	0.43	7.34	12.02	36.56	17.25	17.89	29.65	29.30	33.38	165.38	347.20	668.38

Conclusion

Overall, AE altered the physiology of the injured animals (combination of blast and hemorrhage).

Hypobaria significantly affected the survival of rats during the 3 hour simulated AE, particularly under hypotensive conditions and relative hypoxia.

Non-survivors exhibited poorer control of hemodynamics during the flight.

These findings warrant further investigation into more specific effect of brain injury including metabolic markers and cognitive behavior.

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Questions?