

XIIth US/USSR Joint Working Group Meeting
on Space Biology and Medicine
Washington, D.C., U.S.A.
November 9-22, 1981

Comparison of the Cardiovascular (CV) Changes Between Supine and
Orthostatic Positions as Measured by Invasive Techniques

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Comparison of the Cardiovascular (CV) Changes Between Supine and Orthostatic* Positions as Measured by Invasive Techniques

The most common gravitational reactions are the usual daily changes in body position (postural reactions). The transition from horizontal to orthostatic position is associated with shifts in the CV system expressed mainly as hydrostatic changes in blood pressure. This problem has evoked considerable interest, although whatever research has been done on the subject has been essentially theoretical or math modelling. Direct measurements of true blood circulation in a healthy man during postural changes were rarely made and were episodic in character. Because of that, qualitative studies of pressure changes in different parts of the CV system in the entire body, and the intensity of these changes depending on the body's angle of inclination, suffer from experimental paucity.

Transition to an orthostatic position first causes an increase in transmural pressure in the vessels of the lower extremities and an increase in venous volume in that area; secondly, intrathoracic blood volume and the load on the right heart are decreased. In 0-g, or in 0-g simulation on Earth, such conditions can be created using LBNP. Most work on this subject is conducted by using indirect methods, and for this reason little is known about changes during LBNP application in such important parameters as central venous pressure, pulmonary artery pressure, right heart blood volume, oxygenation and blood acid-base balance, etc.

*When Soviets say "Orthostatic", it means any positive angle of inclination from supine to a 90° head-up position. (NT)

The purpose of the present research is the following:

1. Study of orthostatic responses to various body angles of inclination on blood pressure and oxygenation in different areas of the CV system.
2. Study of various LBNP rates on the central and peripheral blood circulation.
3. Comparison between orthostatic and LBNP test responses on the most informative blood circulation data and above all, data on central venous and pulmonary artery pressures.

Methods

Invasive monitoring of the CV system is the principal method of research. In the last few years nearly 400 catheterizations of various circulatory areas, were performed on 50 healthy volunteers, under various conditions (gravitational tests, immobilization, physical loads and others) (V. E. Katkov, et al., O. G. Gizenko, et al., V. E. Katkov, V. V. Chestukin, V. E. Katkov, et al., V. E. Katkov, et al.) (1-5). It must be noted that we used not only the "sharp" catheterization method, where the probe remained in the venous bed or in the heart for 3 to 5 hours, but we also implanted catheters, specifically into the pulmonary artery for periods up to 12 days.

Slides 1 and 2 show a typical catheter positions in the upper internal jugular bulb and pulmonary artery. Catheter presence allows simultaneous recording of blood circulation and metabolism, i.e. the evaluation of adequate blood supply level to tissue oxidation-reductive processes.

Results and Discussion

Tilt Tests. Areas of catheterization, Catheters used, and positioning of manometers are shown on Slide 3. It shows our attempts to catheterize almost all "strategically" important points of the CV systems from the base

of the skull to the dorsalis pedis. Catheterization was done with the subject in the supine position and upon completion the subject was raised to an angle of 10, 30 and 75° for 5 minutes each.

Slide 4 shows that during a gradual transition to a head-up position, there was a pressure drop in the jugular vein and the right atrium. Noteworthy is that during the 30 to 75° transition, the right atrium pressure kept dropping to below atmospheric, whereas jugular vein pressure remained practically unchanged. As a result, at 75°, the jugular vein-right atrium pressure gradient doubled in comparison with the supine position. Pressure curves from the areas, while similar when in supine position, were quite different at an angle of 75° (Slide 5).

Results show that, in orthostasis, the pressure gradient between the bulb of the internal jugular vein and right atrium can increase. We know that in orthostasis, jugular vein pressure below that area becomes sharply negative. Surrounding tissue pressure also contributes to the venous collapse and a sharp increase in blood flow resistance. Under these conditions the stability of the latter can be attained only with a pressure gradient increase in the section from the upper bulb to the right atrium. In other words, these changes are very similar to those observed in the Starling resistor.

It is interesting to note that in orthostasis, a similar interdependence was characteristic of the pulmonary diastolic pressure and the end-diastolic pressure in the left ventricle which, in a healthy man is essentially equal to the left atrial mean pressure. Slide 6 shows that the left atrial pressure drop was higher than the mean pressure drop in the pulmonary artery, which was reflected as an increase of the transpulmonary BP gradient

(pulmonary artery mean pressure less left atrial mean pressure). Slide 7 shows the original data of such a change in orthostasis in one of the subjects. The increase in transpulmonary gradient along with a decrease in cardiac output confirms the increase in vessel resistance in the pulmonary circulation.

In an orthostatic position there is considerable irregularity of pulmonary blood flow, expressed in "Vest Z zone". According to experimental data, a pressure drop in the pulmonary artery and veins is accompanied by a drop in pulmonary capillaries and venules, which can considerably increase vessel resistance in this area (Permutt, et al) (6). Based on this premise our colleagues at the Institute have run tests on a mathematical model with results which corresponded to our experimental data (A. I. Dyachenko, V. G. Shabelnikov) (7).

At the same time, we realize that in these conditions postural responses caused catheters to shift not only in relation to the manometer, but to each other as well, which could affect the measurement accuracy.

Because of that, a final conclusion on transpulmonary intravascular pressure gradient changes can be made only with pressure data obtained through invasive techniques.

Transition to an orthostatic position caused a pressure increase in lower extremities vessels. Slide 8 shows that pressure increase in arteries and related veins was not uniform. Pressure increase in the artery was always higher than in the vein and generally corresponded to modelling figures over the entire area from the right atrium to the dorsalis pedis.

Hydrostatic venous pressure increase was always below expected values. This peculiarity was already noted in 1911 by Hooker (8) who said that

experimental data corresponds with modelling figures only in cases of paralysis of the extremities or in deep narcosis. In the Pollack and Wood (9) experiments the venous pressure increase at the shin level in orthostasis was only 75 mm Hg, i.e. lower than model figures by about 20 mm Hg. According to these authors, the reason for this discrepancy is the lower extremity muscle tone increase in orthostasis. Our data agrees with this. At the same time, a more elaborate analysis shows that venous pressure decreases do not take place in the lower extremities but rather in areas located higher.

Slide 9 shows that the femoral-dorsalis pedis venous pressure increase corresponded to model figures, whereas in the area from the right atrium to the femoral vein it was about 15 mm Hg lower. The apparent conclusion is that the effect of hydrostatic fluid columns leveled off above the dorsalis pedis level.

Following Pascal's law, arterial and venous pressures, located at the same distance from the heart, should increase uniformly, as in an U shaped tube. This could be caused only by compensatory mechanisms which decrease venous pressure. Our results showed that in a healthy man standing quietly compensatory mechanisms are located in the abdominal cavity, i.e. in an area of the hydrostatic indifference point.

The principal mechanism which provokes this effect is an increase in abdominal muscle tone and intra-abdominal pressure, as well as an increase in negative pressure in the heart and thorax. Existing valves in the iliac veins kept a return blood flow from occurring when the intra-abdominal pressure increased. This increase also led to a pressure decrease in the inferior vena cava and a relief in the venous flow to the heart. According to A. Guyton (10) an intrathoracic pressure drop has a considerable influence

on this effect in orthostasis, inasmuch as it brings about a reflex which automatically increases the muscle tone of the abdominal wall. If a man begins moving, however, the lower extremities' "muscle pump" joins up with the "abdominal muscle pump". As a result shin venous pressure can drop to levels found in a supine position (Pollack and Wood) (9).

Slide 10 shows arterio-venous difference changes (in %) in O_2 for various organs and tissue in a 75° orthostasis. Following Fick's principle, this effect is closely related to cardiac output. Results obtained show that it does not change uniformly in different organs. Thus, if blood flow remains relatively constant in the brain or the heart, it slows down considerably in other areas. In fact, the further (lower) away from the heart, the slower it becomes. In spite of distinct pressure and blood flow changes in some areas during orthostasis, acid-base balance remained constant, which points to an adequate blood supply to organ and tissue metabolic requirements.

Lower Body Negative Pressure (LBNP)

As in tilt tests, LBNP causes a transmural pressure increase in vessels of the lower extremities and a decrease in intrathoracic blood volume.

In simultaneous recording of Dorsalis pedis and arterial venous pressure, Slide 11 shows that LBNP application causes an immediate pressure drop in the vein, which after 2-3 minutes returns to the original level, whereas the arterial mean pressure of the Dorsalis pedis remains constant throughout the entire decompression period. The results, while not indicative of transmural pressure change in tissues, show that venous vessels are affected by this reaction and immediately respond to an even small level of decompression. According to Loeppky, et al. (11) a small level of decom-

pression (-20 mm Hg) brings about a sharp (51%) decrease in blood flow in a tibialis without any change in its diameter. It is thus apparent that basic venous changes take place even with small degrees of rarefaction. In other words, decompression leads to a venous transmural pressure increase with consequent capacity and blood volume sequestration increase which, in turn causes a proportional drop in central blood volume, central venous, and pulmonary artery pressures.

This assumption agrees with results obtained in measuring central circulation, mainly central venous and pulmonary artery pressures (Slide 12). The slide shows that the most visible changes took place during light decompression but further increases caused less apparent shifts. These results also agree with published works. In particular, Zoller et al (12) showed that in a LBNP range from 0 to 5 mm Hg, CVP had an average drop of 8% at 1 mm Hg, then at -2 mm Hg this drop averaged only 1% of mm Hg of decompression. We show that such changes are characteristic not only for CVP but for the pulmonary artery pressure as well.

A more detailed analysis on pulmonary artery mean pressure affected by different levels of decompression is given in a semilogarithmic scale (Slide 13). These pressure figures were taken 10 to 15 seconds after a relative rate of rarefaction had taken place. The abscissa represents rates of rarefaction and the ordinate—a natural logarithm of pulmonary artery pressure. This curve can be conditionally divided into two areas: in the rarefaction range of 0 to -30 mm Hg and from -30 to -60 mm Hg, with each being different based on decompression level. The most apparent decrease is within the 0 to -30 mm Hg range. Changes in the second range are not so apparent.

This peculiarity, i.e. shifts in low pressure system with low rarefaction magnitude-is apparently caused by a change in venous capacity in the decompression area. It increases sharply with a slight increase of transmural pressure (approximately up to -30 mm Hg). Further increase causes a less apparent capacity change.

Data thus obtained shows that, first, this response causes the veins in the decompression area to react almost immediately; second, low magnitude LBNP application causes significant shifts in the low pressure system; third, increased decompression below -30 mm Hg does not cause any drastic shifts in the system.

The comparison of responses in LBNP rates and tilt-tests on central circulation, using CVP and pulmonary artery pressure, was made for the following considerations:

- These figures are predominant in the formation of such physiological factors as preloading and afterloading of the right ventricle.

- They are extremely sensitive to gravitational reactions, or their simulation.

- Their quantitative measurement can be accurately taken in both stable and transitional periods.

Slide 14 shows changes in these figures, as well as the right ventricle function and the O_2 arterio-venous differences for systemic circulation in response to various LBNP and tilt-test rates (dotted line). At a -30 mm Hg rate, LBNP has the same responses as tilt-tests at -60 mm Hg. Pulmonary artery pressure and right ventricle function were lower but the arterio-venous difference was higher than in orthostasis. Thus, to simulate gravitational response on low pressure system with LBNP, the optimal rate should not

exceed -30 mm Hg. Invasive techniques, therefore, enabled us to establish a number of peculiarities, which take place in the cardiovascular system in response to gravitation or the simulation of its effects. In spite of significant circulation changes, blood supply to tissue metabolic requirements remains adequate. This is reflected by the continuity of acid-base balance of blood taken from various regions of the cardiovascular system.

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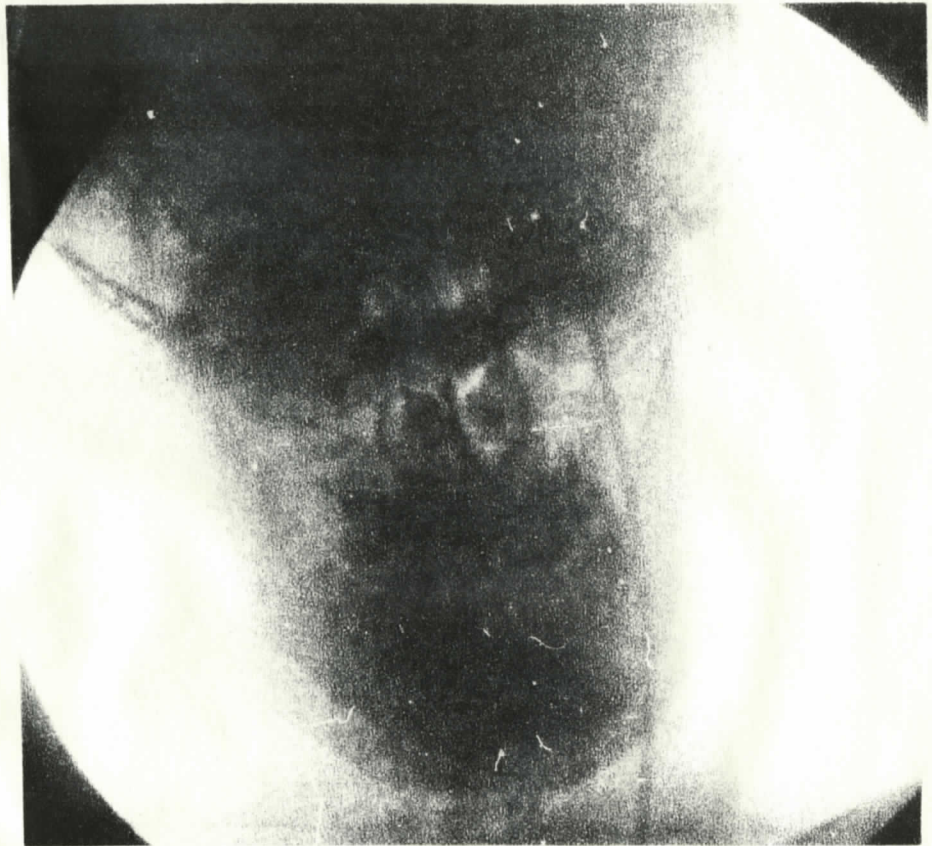


Fig. 1



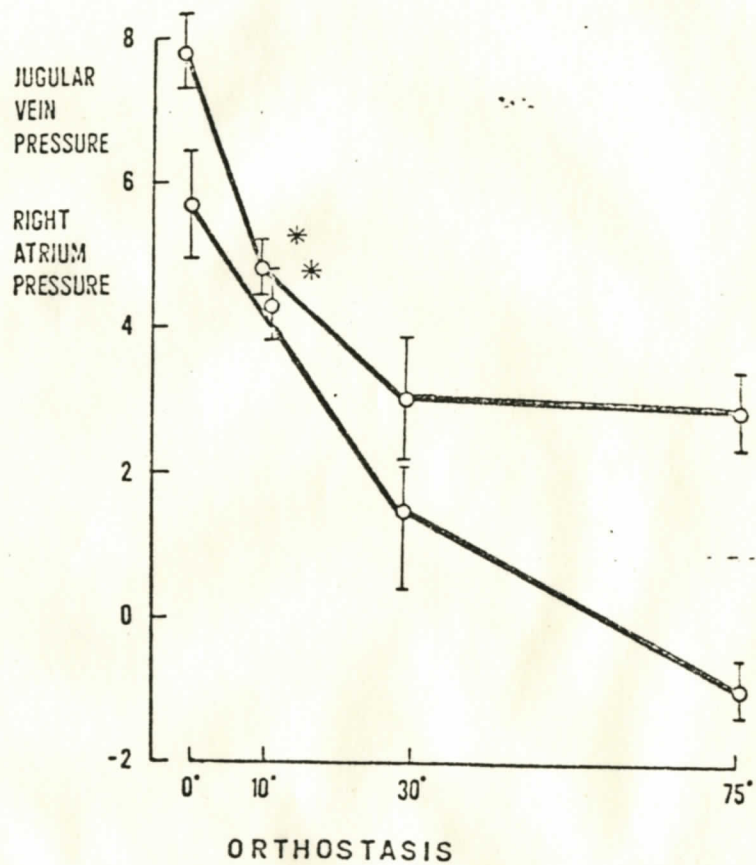
Fig. 2
(Mirror Image)

METHODOLOGY OF THE STUDY

SERIES	AREA OF CATHETERIZATION	CATHETERS AND CANNULAS	LEVELS OF LOCATION OF MANOMETERS
I	UPPER BULB OF THE INTERNAL JUGULAR VEIN	ODMAN-LEDIN	TIP OF THE CATHETER
	RIGHT ATRIUM	ODMAN-LEDIN	TIP OF THE CATHETER
II	LEFT VENTRICLE	ODMAN-LEDIN	RIGHT ATRIUM
	CORONARY SINUS	COURNAND No. 8	RIGHT ATRIUM *
	PULMONARY ARTERY	COURNAND No. 8	RIGHT ATRIUM
	FEMORAL ARTERY	CANNULA	RIGHT ATRIUM *
III	FEMORAL ARTERY	ODMAN-LEDIN	TIP OF THE CATHETER
	FEMORAL VEIN	ODMAN-LEDIN	TIP OF THE CATHETER
	FOOT ARTERY	CANNULA	TIP OF THE CANNULA
	FOOT VEIN	CANNULA	TIP OF THE CANNULA
	(ONE OF THE BRANCHES OF THE		
	RETE VENOSUM DORSALE PEDIS)		

* CATHETERS WERE USED ONLY FOR BLOOD WITHDRAWAL

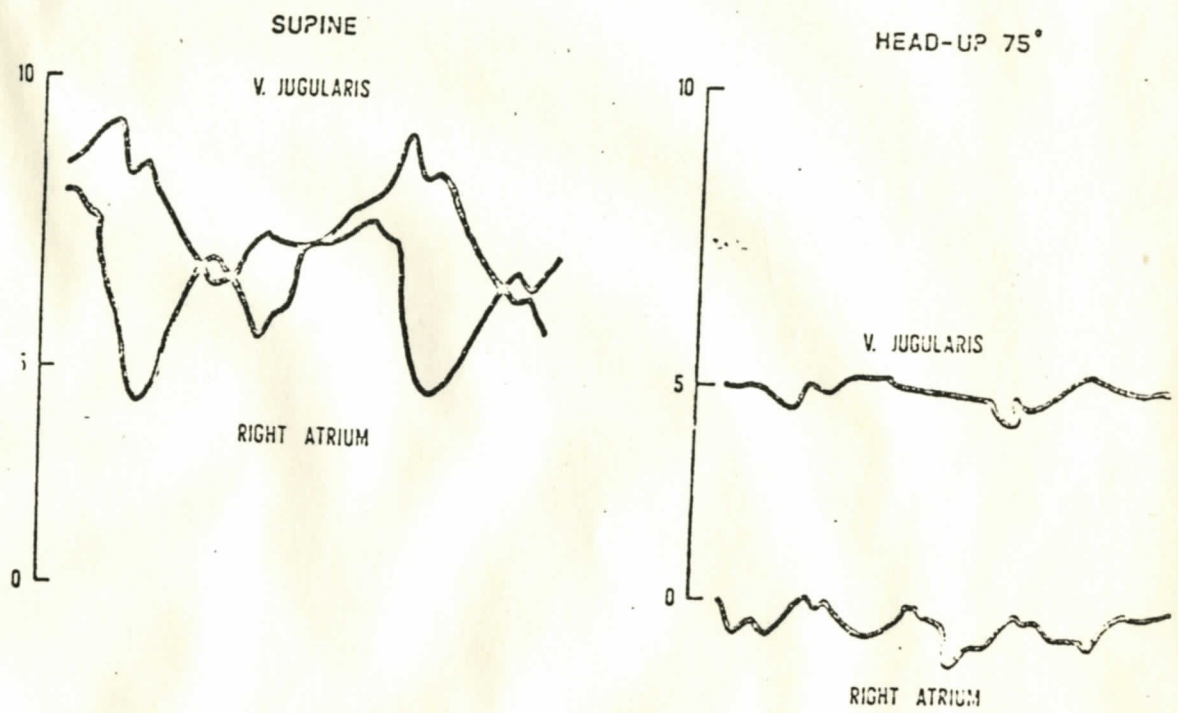
Fig. 3



MEAN PRESSURE (mm Hg) IN THE
JUGULAR VEIN AND RIGHT ATRIUM
DURING ORTHOSTASIS

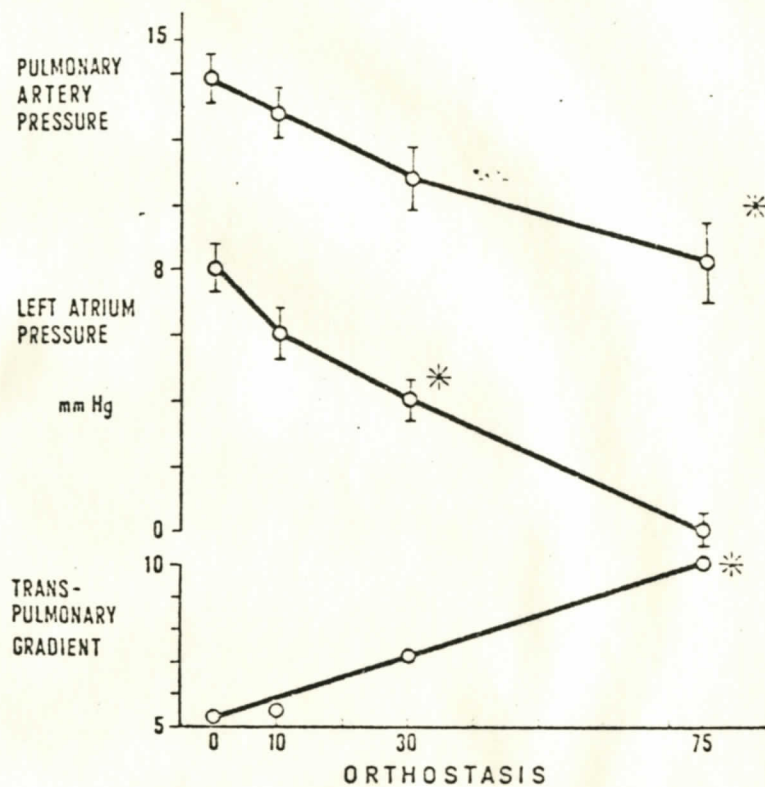
* - SIGNIFICANT

Fig. 4



TYPICAL CHANGES OF THE JUGULAR VEIN AND RIGHT ATRIUM PRESSURES (mm Hg) IN ORTHOSTASIS 75

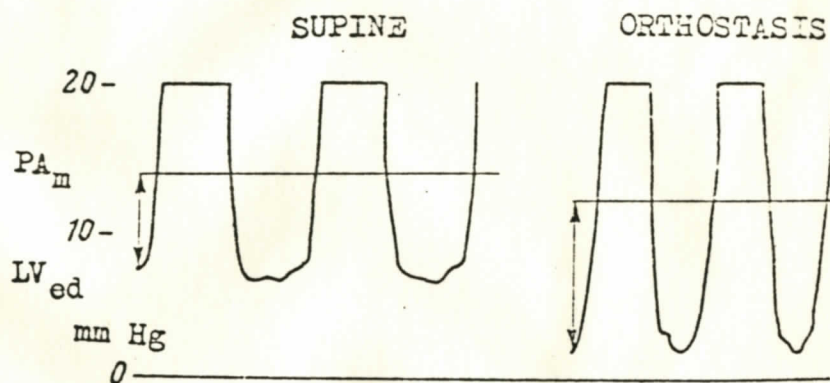
Fig. 5



MEAN PRESSURE IN THE PULMONARY ARTERY,
LEFT ATRIUM PRESSURE (END-DIASTOLIC PRESSURE
IN THE LEFT VENTRICLE) AND TRANSPULMONARY
GRADIENT OF THE INTRAVASCULAR (VENOUS)
PRESSURE DURING ORTHOSTASIS

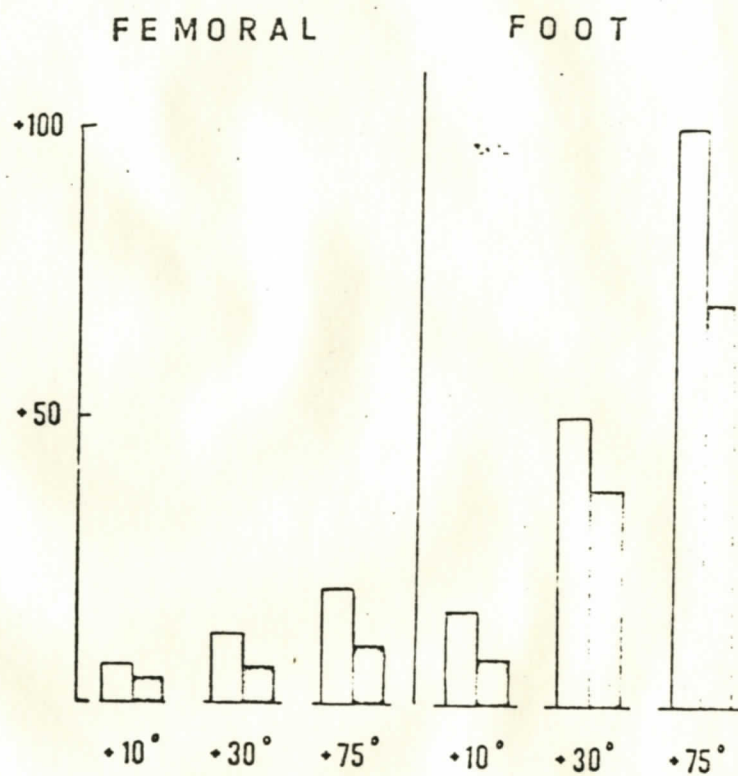
* - SIGNIFICANT

Fig. 6



END-DIASTOLIC PRESSURE IN THE LEFT VENTRICLE (LV_{ed}) AND MEAN PRESSURE IN THE PULMONARY ARTERY (PA_m) RECORDED IN THE TEST SUBJECT GM IN THE HORIZONTAL AND HEAD-UP POSITION. THE TRANSPULMONARY GRADIENT OF THE INTRA-VASCULAR PRESSURE IS SHOWN BY THE ARROW WHICH COINCIDES WITH THE R WAVE IN THE II STANDARD ECG LEAD.

Fig. 7



INCREMENT OF PRESSURE (mm Hg)
IN LEG VEINS AND ARTERIES
DURING ORTHOSTASIS
 □ - ARTERY ■ - VEIN

Fig. 8

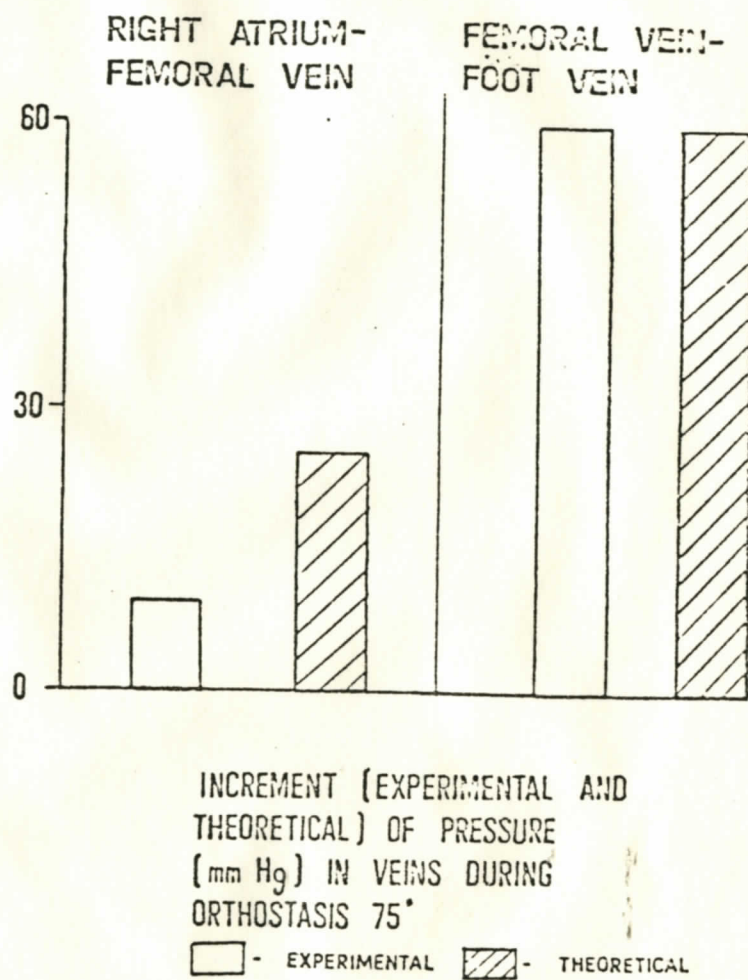
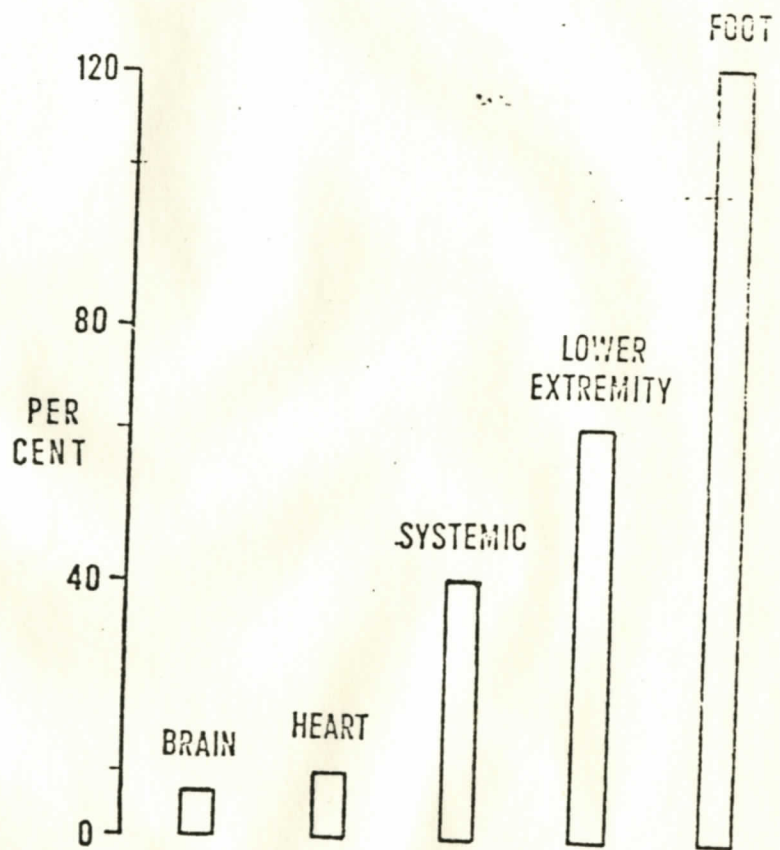
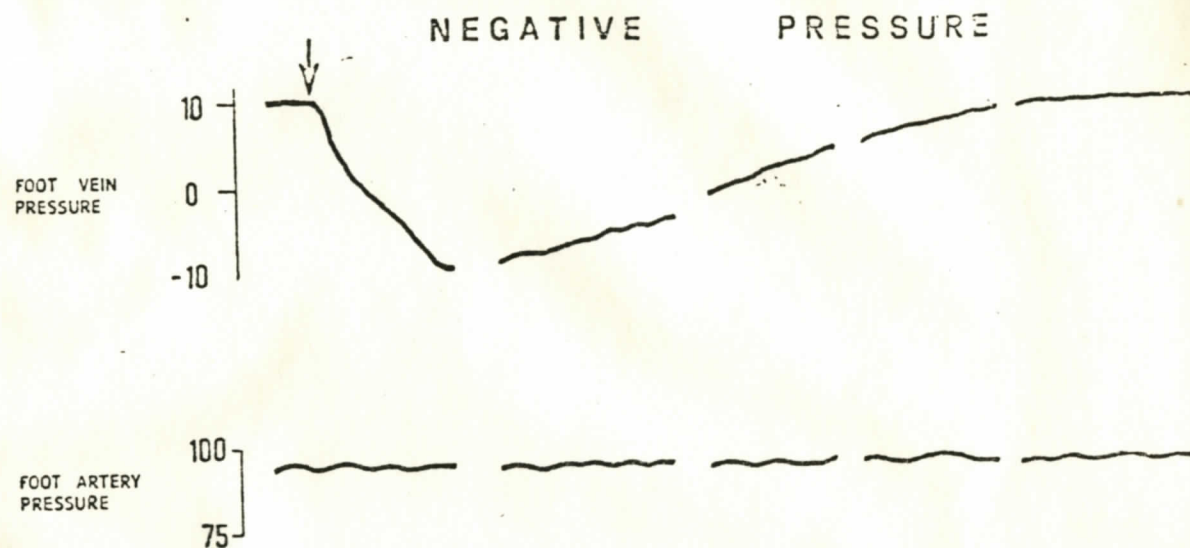


Fig. 9



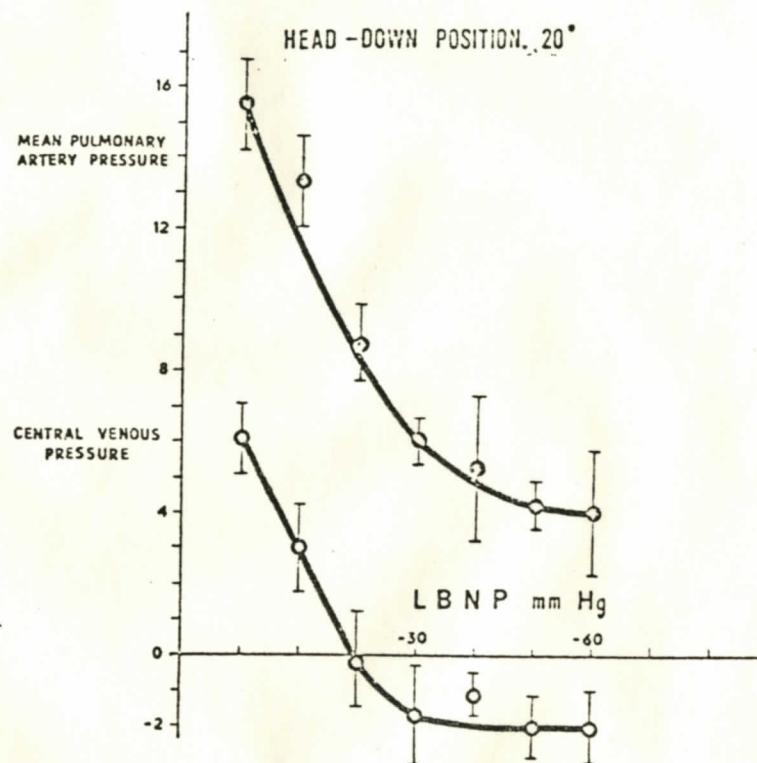
PERCENTAGE CHANGES OF THE OXYGEN
ARTERIOVENOUS DIFFERENCE IN DIFFE-
RENT ORGANS AND TISSUES DURING
ORTHOSTASIS AT 75°

Fig. 10



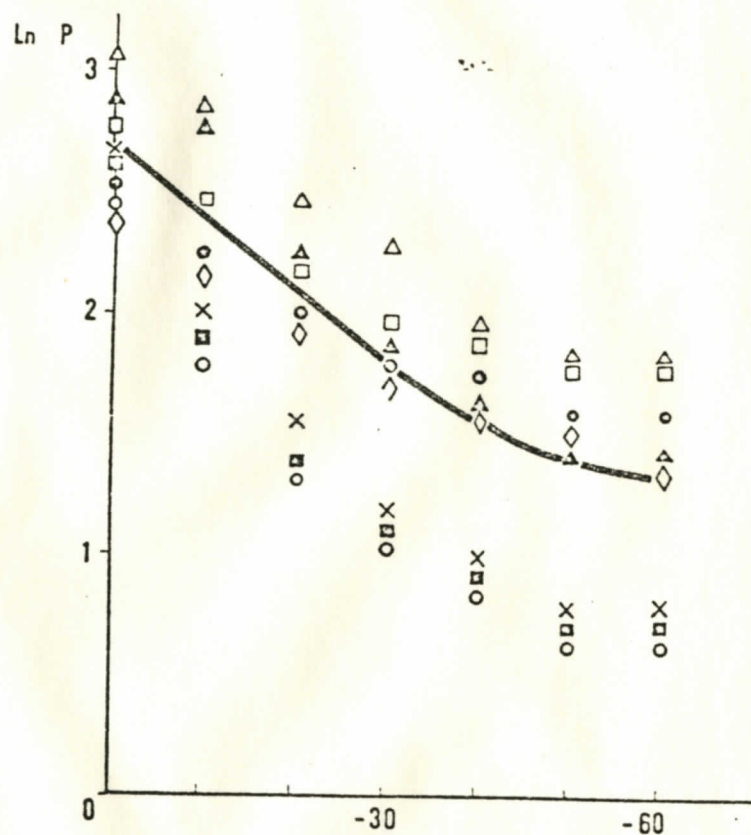
PRESSURE (mm Hg) OF THE DORSAL VEIN AND ARTERY OF THE FOOT
DURING LBNP (ORIGINAL RECORDING)

Fig. 11



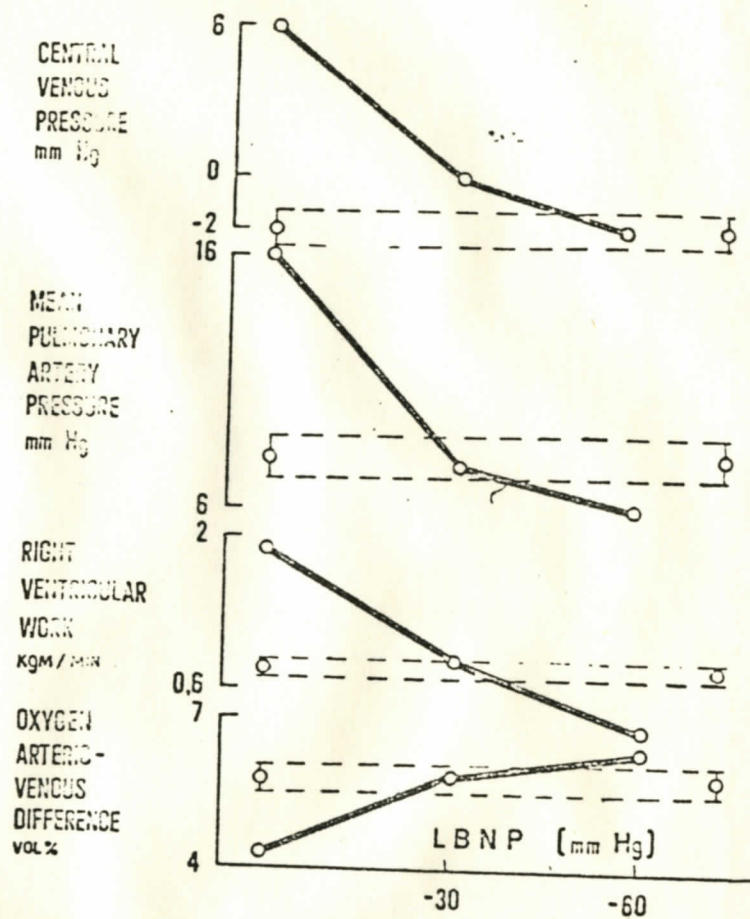
MEAN PULMONARY ARTERY PRESSURE AND CENTRAL VENOUS PRESSURE (mm Hg) DURING LBNP (CHIBIS SUIT WITH THE DECOMPRESSION LEVEL INCREASED)

Fig. 12



LBNP DEPENDENCE OF THE
NATURAL LOGARITHM OF MEAN
PRESSURE IN THE PULMONARY
ARTERY [mm Hg]

Fig. 13



EFFECT OF THE TILT TEST AT
70° AND DIFFERENT LEVELS OF
LBNP ON CENTRAL CIRCULATION
AND OXIDATIVE METABOLISM

Fig. 14

Tilt Test Shown in Dotted Lines