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PHYSIOLOGY AND OPERATIONAL COMPARISON OF MC-1 AND MC-3 (MC-4) PARTIAL PRESSURE SUITS

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FOREWORD

This work was performed under Project No. 6333, "Development of Pressure Suit Systems," Task No. 63612, "Development of Pressure Suit Garments," under the direction of Capt. Terence F. McGuire, USAF, MC, of the Respiration Section, Physiology Branch, Aerospace Medical Division, Wright Air Development Division.

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ABSTRACT

A theory on the physiological limitations of partial pressure suits, with supporting evidence, is discussed. Loss of "effective" blood volume, workload placed on the heart, available oxygen, a number of reflexes that can work separately or together to the individual's disadvantage, and other contributory mechanisms are stressed. The comparative operational characteristics of the MC-1 and MC-3 (MC-4) partial pressure suits are presented.

PUBLICATION REVIEW

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INTRODUCTION

This report discusses the physiological limitations and other related considerations of a "get-me-down" type suit, i.e., the MC-1 partial pressure type suit which led to the development of the more effective MC-3 and MC-4 ensembles. The contents of this report constitute Part I of a total study; Part II (ref. 138) summarizes the time-altitude capabilities of the MC-1 and MC-3 (the MC-4 is the same as the MC-3 suit but also includes a g suit) ensembles with particular attention given to the operational problems associated with such partial pressure type suits.

To develop a simple and readable working hypothesis of the normal and abnormal physiology of partial pressure type suits for dissemination to physiological training officers, flight surgeons, and other interested personnel was the purpose of this study. Although some portions of this working hypothesis have not been completely proven, this present theory appears to be the most practical in the light of available information.

Some form of external counterpressure must be used to balance the high breathing pressures that are necessary to maintain adequate oxygenation and prevent the adverse effects suffered at high altitudes without a pressurized cabin. At 40,000 feet without a pressurized cabin, 100 percent oxygen is only sufficient to produce an environment comparable to breathing free air at 10,000 feet, a condition of mild deficiency in blood oxygen saturation not ordinarily imposing hazard. Survival above 40,000 feet without either a pressurized cabin or a pressure suit is tenuous; since the atmospheric pressure cannot present sufficient oxygen to the lungs, even with the use of 100 percent oxygen, exogenous pressure must be added to the breathing system to insure adequate oxygenation of the blood. An elevation of altitude from 40,000 to 45,000 feet requires an additional 31 mm. Hg breathing pressure for sufficient oxygenation. However, under this pressure, an A-13 mask, even with advanced harness, cannot hold its seal against the face. Further, internal pressure causes the eardrum to bulge painfully; increasing eyeball

pressure produces pain; the reflexively active upper respiratory tract is disturbed; and a series of cardiovascular effects is set in motion which greatly limits the ability of man to remain at this altitude. Even slight increases beyond this 45,000-foot altitude would considerably increase these adverse effects.

In case of cabin pressurization loss, an altitude of 50,000 feet is recognized as the physiological dividing line above which only a few personnel could be expected to compensate sufficiently to retain consciousness for a minimal "get-down" time. If decompression is rapid at 55,000 feet and above, useful consciousness on 100 percent oxygen can be measured only in seconds, about 12 seconds or less, which would permit only sufficient time to become aware of the condition but not enough time to effect compensating measures. Although some pilots have said, "I can hold my breath for almost 2 minutes; surely I can do the same in my aircraft and return safely to 40,000 feet," they have neglected consideration of the fact that such breath holding is preceded by deep inspiration and the lungs are relatively emptied by the decompression process. Rapid or explosive decompression rarely offers any warning and, even if it did, locking the glottis or back of the throat against decompression would lead to pulmonary damage as the trapped gases expanded. Also, the ground-level air has higher density and contains more usable oxygen than is available in a "just decompressed" cabin, even if the pilot were on 100 percent oxygen.

The most logical means of providing this counterpressure would be to encase man in a formfit, flexible, nonpermeable garment inflated with air, i.e., a full pressure suit. However, at the outset, problems of bulk, mobility, temperature, and humidity precluded the field practicability of such a garment. Therefore, the Air Force concentrated on the development of a formfitting counterpressure garment, the "partial pressure" suit for field use. Along the torso and extremities of this garment are inflatable tubes (capstans) attached to the cloth of the suit by means of interdigitating tapes. These tubes are so designed in relation to varying body segments that a capstan pressure pulls on the interdigitating tapes and tenses the cloth of the suit to cause a pressure to be exerted on the body surface; for example, a capstan pressure of 5 psi would cause a pressure of 1 psi on the body (the 5 to 1 ratio).

Initially, the T-1 type garment sufficed to fulfill the Air Force requirement for a "get-me-down" type suit. The comfort and mobility of the partial pressure suit were increased by a readjustment of the capstan size and by addition of a bladder along the anterior portion of the trunk which made breathing less difficult. The gradual evolution of the prototype garment resulted in the MC-1. An evaluation of the MC-1 capability was prompted in 1954 when Headquarters, USAF, initiated consideration of a mission completion garment. This investigation demonstrated the ineffectiveness of the MC-1 and led to the development of the MC-3.

DISCUSSION

A. Rectifying Four Basic Fallacies

To properly understand the causes of the physical limitations observed in the use of the MC-1 and discussed later in this report, four basic fallacies should be rectified.

1. Hypertension of Pressure Breathing

The expression "hypertension of pressure breathing" is believed to be a misnomer. Many investigators have noted that, when a subject breathes under pressure, while there is an observed blood pressure elevation, the elevation in blood pressure is always less than the increase in breathing pressure. The author investigated rather

thoroughly this phenomenon with a pressure-differential tank and concluded that the wrong frame of reference had been used in the past. If a subject is placed into the pressure-differential tank, the helmet and body surface pressures are raised equally, and a mercury manometer for measuring blood pressure is left outside the tank, the blood pressure rises as virtually a 100 percent additive factor. However, if the manometer is carried inside the tank and is "backloaded" by the breathing pressure, no significant blood pressure alteration is observed. Similarly, if the subject is seated outside the altitude chamber and the manometer is placed inside, an artificial hypertension, proportional to the degree of vacuum around the manometer, will be produced. The converse is true if the positions of the subject and manometer are reversed. If the manometer is placed outside and an altitude of about 6,000 feet (a pressure drop of 159 mm. Hg) is simulated, it fails to register any blood pressure at all. If breathing pressures are not balanced, a reactive vasoconstriction will take place in an attempt to compensate for the cardiovascular stress imposed by the pressure breathing. However, although ophthalmoscopic examination of the fundus of the eye is difficult while a fullhead helmet is in place, such examination failed to reveal the vasoconstriction that would be expected with a hypertension of, for example, 280/230. Accurate blood pressure are determined only by using the breathing pressure as the frame of reference, either by subtracting the added breathing pressure from the apparent blood pressure or by backloading the manometer with breathing pressure; this is the case during the usual clinical determination of blood pressure where the atmospheric pressure one breathes is also backloading the blood pressure manometer. That the hypertension observed during pressure breathing without counterpressure garments or with equipment like the T-1 or MC-1 suit is not a directly additive factor between blood pressure and added breathing pressure is a reflection of the combination of inadequacy of the counterpressure and the inability of the individual to compensate entirely physiologically. Although it would be more accurate to use changes in the intrapleural pressure as a baseline, for practical purposes in the pressure suit situation, the breathing pressure serves well as a rough baseline. Some degree of difference is expected in pleural pressure curves, as related to altered airway pressures, depending on the relaxation of the subject (higher pleural pressure rise with active respirations and muscular tenseness) and the elasticity of the lungs (greater pressure transmission in less elastic pulmonary tissue).

It is extremely important to the circulation that the thoracic cavity maintain a "subatmospheric" pressure (refs. 96, 99) because of its gross influence on the effective filling pressure of the heart, which pressure multiplies when the effects of the negative intrathoracic pressure and the peripheral venous pressure are added. The author is certain that the hypertension of pressure breathing is apparent rather than real and is actually a relative hypotension in situations of unbalanced pressure increases in the chest.

2. Pressure Differential Tolerated at Ground Level Can Be More Than Duplicated at Altitude.

The expression "Since pressure breathing is more difficult at ground-level than at altitude, any pressure differential you can tolerate at ground-level can be more than duplicated at altitude" needs rectifying. Work with the pressure differential tank and the altitude chamber demonstrated that not only is the degree of positive pressure breathing important, but also its relationship with the surrounding or ambient pressures. A subject with a breathing pressure of 30 mm. Hg at 30,000 feet (225 mm. Hg ambient pressure) is in a much more precarious position than breathing the same pressure load at ground-level (760 mm. Hg ambient pressure), even though he is receiving the same amount of oxygen to the blood via the full-head helmet. This is an important point because it means that much of the work on pressure breathing done heretofore at ground-level is applicable to the altitude situation only in the sense of physiological pattern, not in the sense of time duration. The author has been able to demonstrate a significantly

decreased cardiac output, longer circulation time, and augmented electrocardiogram changes for a constant degree of uncompensated pressure breathing (the same situation prevailing in a partial pressure suit whose counterpressure is inadequate) by the mere expedient of reducing the surrounding ambient pressure, for example, from ground level to 18,000 feet (half an atmosphere). At 18,000 feet, a subject on 100 percent (actually 99.6 percent) oxygen is still receiving more oxygen than he can use and is below altitudes where gaseous distention of the intestine (bends) is a problem. But the amount of "counterpressure" supplied by the atmosphere to the surface of the body has been reduced by 7.3 psi, apparently making "pooling" of blood of vascular circuits much easier.

An interesting observation has been the finding of Hurtado and his group of Morococha, Peru (ref. 89), where their studies on the local inhabitants at that altitude (14,900 feet) have shown hypertension to be virtually unknown. As the atmospheric pressure applied to the body surface forms part of the total peripheral resistance to blood flow and thus affects blood pressure, one wonders if the reduced atmospheric pressure (430 mm. Hg or 8 psi as compared with 760 mm. Hg or 14.7 psi at sea level) played a significant part in this observed condition.

Why, then, is it subjectively easier to breathe under pressure at altitude though not so well protected? The exhalation valve on USAF full-head helmets is held shut by a combination of breathing pressure backload, spring tension, and atmospheric pressure Above 40,000 feet, while the atmospheric pressure continues to fall, the inner environment of the helmet remains at the pressure of 40,000 feet. Atmospheric pressure is 140.7 mm. Hg at 40,000 feet, and it drops to 20.8 and 8.0 mm. Hg at 80,000 and 100,000 feet, respectively. Thus, there is less resistance to the valve. Also aiding in this subjective improvement is the movement of less dense air through the respiratory tract in a more laminar and less turbulent manner and with a consequent reduction of resistance; this experience is noted even before reaching pressure suit altitudes.

3. 40,000-Foot Equivalent

The adequacy of the "40,000-foot equivalent" needs rectifying when considering an acute change in altitude. Currently, partial pressure suits attempt to return the man exposed to extreme altitudes to the breathing pressures and body surface counterpressures which he experienced at 40,000 feet on 100 percent oxygen. At 40,000 feet without a pressurized cabin, 100 percent oxygen is only enough to produce a situation comparable to breathing free air at 10,000 feet, a condition of mild deficiency in blood oxygen saturation. Although there is a colony of people living at 17,500 feet in South America, they have had the necessary time to become acclimated to this situation with the required major physiological and biochemical adjustments. Obviously, the case of the man taken acutely to 10,000 feet is quite different. The "mountain sickness" frequently experienced by people as they drive from the plains to the heights of the Rocky Mountains is caused by mild hypoxia. By virtue of their slow ascent, they have a greater length of time to compensate for this condition than does the pilot. If "get-me-down" was all that was required of a partial pressure suit, this mild hypoxia would not constitute a great difficulty. However, the burden to the cardiovascular system imposed by the partial pressure suits up to and including the MC-1 is such that the compensating effort to adjust to the 40,000-foot equivalent becomes excessive.

The author conducted a series of 12-hour altitude chamber runs at 10,000 feet without supplemental oxygen which showed that subjects are forced to draw somewhat heavily on their cardiac reserve and their abilities to compensate and that mental acuity and alertness diminish. The nadir of performance for most subjects fell between 6 and 9 hours. The interesting findings of this study—including blood volume, cardiac output, EKG studies, etc.—will be the subject of a separate report by the author and his

coworkers. However, there are numerous allusions in the literature to the toil taken by reduced availability of oxygen caused by increased altitude. An attempt to go to a 38,000-foot equivalent in the MC-1 failed to improve performance because the increased pressures served to augment the imbalance inherent with the suit, giving increased compensatory strain which even more offset the advantage of increased availability of oxygen.

4. Bends Are Necessarily Progressive

The misconception that bends are necessarily progressive is fortunately not nearly so prevalent as it had been; this fact is mentioned only for the sake of emphasis. Those experiencing bends at altitude, especially after having had some denitrogenation, must not be allowed to feel that this, of necessity, means the "beginning of the end." In the experience of this facility, the majority of cases with bends were progressive and severe only in a minority of cases which had adequate denitrogenation. The majority of cases fluctuated in severity, disappeared, reappeared elsewhere, or remained constant (ref. 138).

B. Selecting and Testing Subjects

1. Selecting

Although volunteers were not specially selected, the author did subject them to a specifically designed physical examination (see Appendix I) intended to eliminate those with incipient cardiac disorders, pulmonary pathology, and other maladies that would normally preclude their participation in any flight activity. This extensive physical examination also provided valuable information in ascertaining the degree of certain of the physical and physiological variables which were measured during their performances.

2. Testing

To insure safety as well as to acquire information, each volunteer subjected to reduced ambient pressures was carefully instrumented to permit physiological monitoring. Electrocardiograms (SE I, SE II, SE III, AVR, AVL, AVF, V1, V2, V4, and V6), vectorcardiograms, blood pressures, respiratory variations, and similar measurements were taken while the volunteer was at reduced ambient pressures as simulated in an altitude chamber; both the electrocardiograms and vectorcardiograms are forms of monitoring heart activity.

In addition to the monitoring of the chamber run as mentioned above, X-ray studies, extensive blood and urine studies, pressure breathing experiments, etc., were made at ground level and at altitude.

An isotope technique (ref. 1) was developed for determining cardiac output, i.e., the amount of blood pumped by the heart per unit time. It proved repeatable, accurate, and highly adaptable to the design purposes. This technique has been checked against other existing methods, including those involving catheterization techniques, with very satisfactory results. Very small doses of isotopes were used to determine shifts of blood and fluids in the body, total volume of circulating blood, and time of blood circulation from one spot to another.

A special pressure differential tank (see figures 1 and 2) was constructed to study the effects of unbalanced pressures, i.e., more pressure on the body surface than around the head and in the respiratory tract, or vice versa. The subject is seated in the apparatus with his head protruding through a port in the upper surface of the tank proper. The

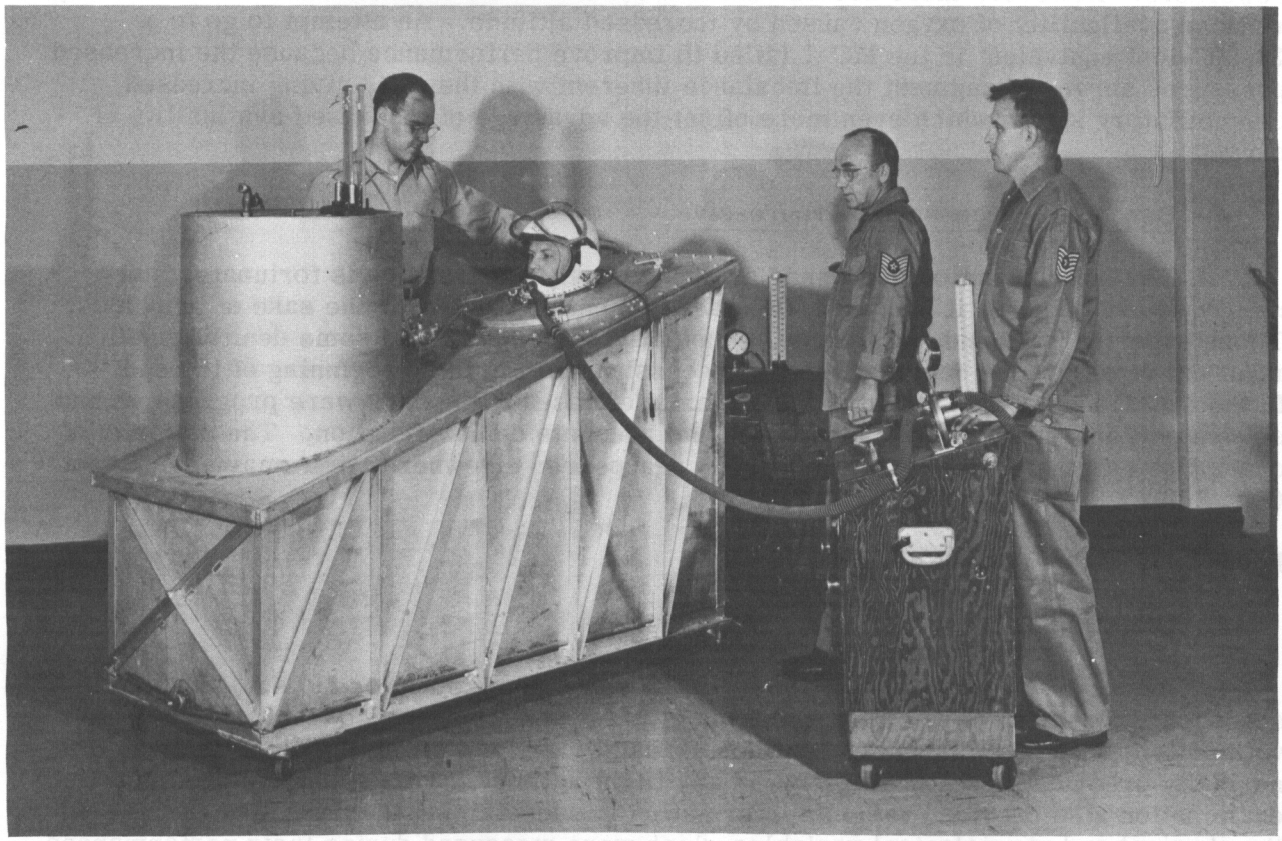


Figure 1. Pressure Differential Tank

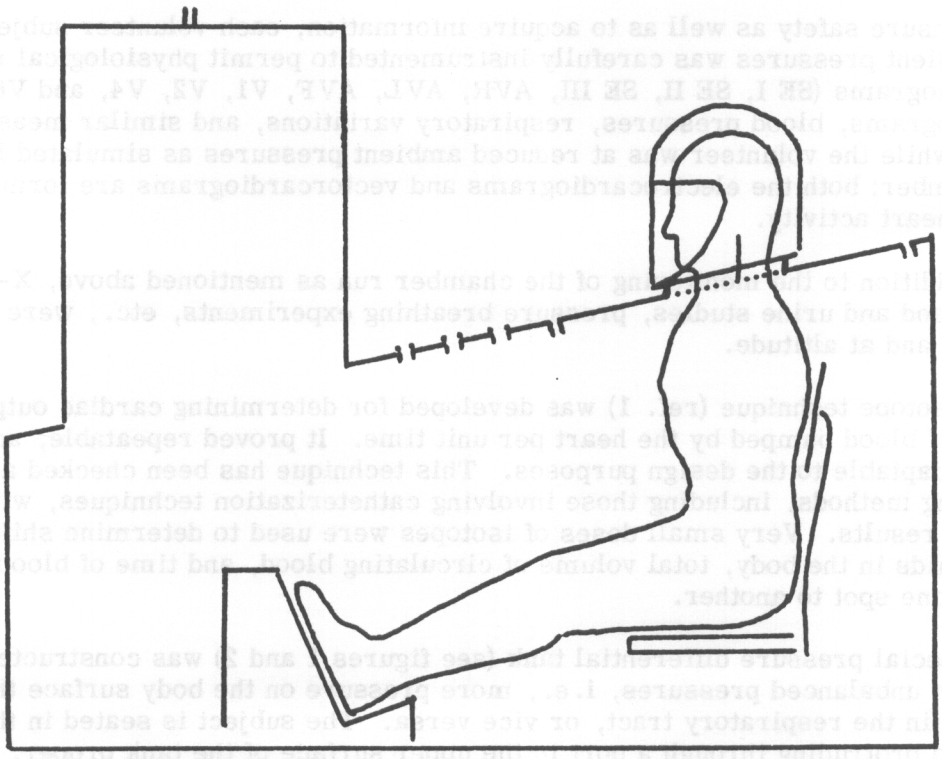


Figure 2. Pressure Differential Tank

head area is pressurized by means of a USAF full-head helmet fastened securely to the surface of the tank. The "compartments" are separated by a special pressure seal which doesn't compromise the pressure-sensitive areas of the neck. This apparatus allows any degree of imbalance to be produced and maintained. It was operated in altitude chambers at both ground level and reduced ambient pressures. One of the reasons for the construction of the tank was to provide the means of accurately evaluating the counterpressure inequities among men in partial pressure suits. The tank also permits an accurate determination of the effects of age, body build, physical fitness, and autonomic nervous system activity on the ability to compensate satisfactorily for imbalance. Further, for those believing that the body can sense counterpressure differences, the tank permits comparison of mechanical counterpressure in the form of water in the "tank" portion, as in the partial pressure suit, with pneumatic counterpressure, as in the full pressure suit. In the case of mechanical counterpressure, the pressure is equated at heart level.

C. MC-1 Partial Pressure Suit Ensemble

1. Time-Altitude Capability

The time-altitude capability studies of the MC-1 partial pressure suit ensemble (MC-1 suit, K-1 or MB-5 helmet, and pressure gloves) clearly demonstrated its gross inadequacy to meet the stated USAF, maximum time-altitude requirements. Figure 3 depicts the MC-1 capability as measured in the altitude chamber runs. The additional test findings since a previous study (ref. 2) reduced the earlier postulated capability at higher altitudes. Using the information of figure 3 to cite examples, the probability of 3 crewmembers being functional at an altitude of 60,000 feet after 1 hour is only about 1 in 7 or 8; after 1 hour at 55,000 feet, there is approximately a 3-out-of-4 chance that all 3 would be functional. This reasoning is based on the consideration of typical jet-qualified fighter and bomber pilots, excluding specially selected crews.

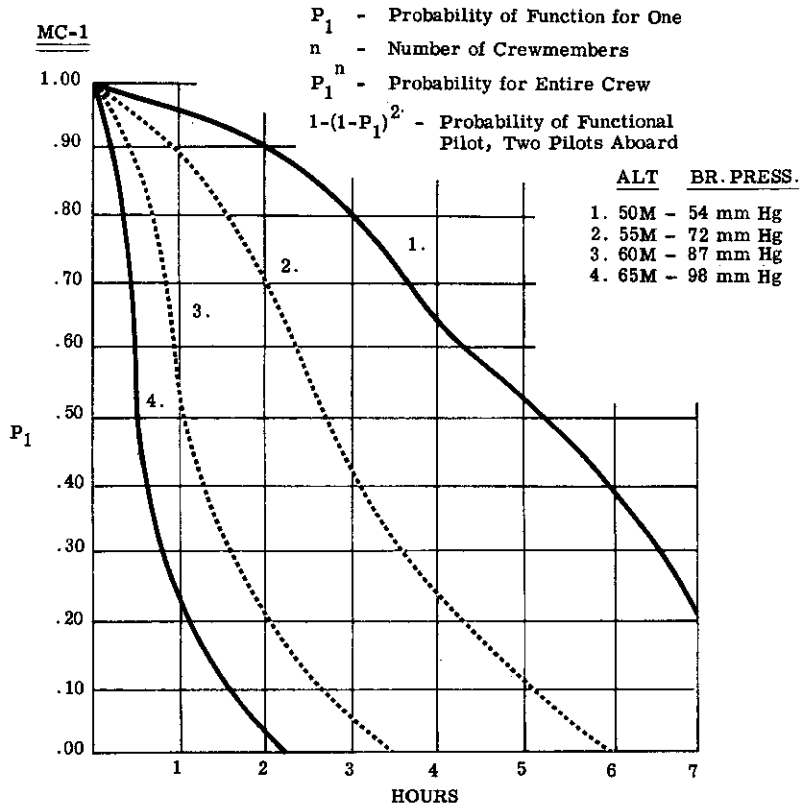


Figure 3. Probability Chart for the Time-Altitude Capabilities of the MC-1 Ensemble

2. Limitations of Test Runs

Altitude chamber runs were terminated at that stage which the author would have chosen as the cut-off point if he were the test subject. Impending syncope and electrocardiographic (EKG) changes prompted about 60% of the terminations, with advanced degrees of bends, neck seal "blowouts" which caused "dumping" of the helmet pressures, hand or neck pains, etc., accounting for the remaining 40% of the terminations. The reasons for termination are discussed at greater length in Part II (ref. 138) of this study.

Table II in Part II (ref. 138) shows that one of the major reasons for altitude chamber flight termination was impending syncope which implies a fainting reaction. As a warning period precedes actual collapse and is evidenced by pallor, nausea, sweating, weakness, malaise, a sensation of being "over-whelmed," etc., the subjects could be and were removed prior to any actual faint. The duration of this period varies with the altitude: at 50,000 feet, there is usually a 5-minute warning; at 65,000 feet, the warning period may be less than a minute.

At simulated altitudes of 60,000 feet or above, about 6 percent of the subjects sustained very great elevations of pulse rate. Within 1 or 2 minutes, the pulse rate would exceed 160 pulses per minute. At rates between 160 and 180 pulses per minute, the minute volume of blood moved by the heart ceases to rise significantly with increase in the pulse rate. For the average human with a pulse rate above 160 pulses per minute, the minute volume falls off sharply because the heart is unable to fill adequately between contractions. Therefore, the author considered a pulse rate of 160 pulses per minute as the cutoff point to avoid syncope.

On the basis of current medical knowledge, the abnormal EKG changes were interpreted as myocardial hypoxia. Subjects were not permitted to continue under this stressful condition since it is considered to be physically injurious. As those in the group experiencing the EKG changes evidenced the characteristics associated with impending syncope if the EKG changes continued and as those in the syncope group usually evidenced EKG changes of lesser magnitude but becoming more marked as termination neared, the division between these two groups is, therefore, somewhat artificial.

3. Mechanisms of the Physiological Limitations

Without considering in detail the methodology and results of the isotope experiments, electronic monitoring, X-ray studies, blood and urine studies, etc., the basic reason for the failure of the MC-1 partial pressure suit assembly and its predecessors to permit sustained human endurance at extreme altitudes is the lack of sufficient counterpressure to balance properly the delivered breathing pressure. Because of this failure, the respiratory cycle is reversed, inspiration being passive and expiration active, with subsequent fatigue of the respiratory muscles. This failure also explains why X-rays reveal distended lungs and a depressed diaphragm while the MC-1 is "inflated," and the functional residual air of the lung is increased and the inspiratory reserve volume is decreased. Further, this failure causes the right axis deviation of the heart as observed on the EKG.

Because of the inadequate counterpressure to balance properly the delivered breathing pressure, four factors evolve which limit the time-altitude capability of the MC-1 partial pressure suit assembly. These four factors are: (1) loss of effective circulating volume, (2) mild deficiency in blood saturation and added compensatory strain in adjustment to the 40,000-foot equivalent, (3) cardiac strain, and (4) reflex activity. The combination of these factors, given sufficient time, precipitates a vasodepressor type of syncope with an acute fall in blood pressure and slow pulse.

However, the experiencing of this resulting condition is not considered dangerous provided that the subject is removed from the situation immediately after the inception of the state of the condition. Reduced mobility in the semi-upright position probably also contributes to this vasodepressor reaction. Yet, remote possibility of cardiac standstill or fibrillation does exist at the moment of the vagal discharge that precipitates the syncope. In fact, the author has observed a cardiac standstill on the oscilloscope lasting a number of seconds. However, vagal "escape" with resumption of cardiac systoles at bradycardiac rates have been evidenced in each case. Obviously, continuation at altitude with overt signs of cardiac strain would be prejudicial to the best interests of the subject.

a. Loss of Effective Circulating Blood Volume

"Pooled" blood and fluid loss as edema of unprotected tissues are the main contributions to the loss of effective circulating blood volume. Smaller contributions to this loss are made through respiration and insensible perspiration; both the artificial breathing atmosphere and the actual surrounding atmosphere of high altitudes are exceptionally dry and, therefore, desiccating.

A blood loss of approximately 25 percent would cause the healthiest subject in the horizontal position to faint; some men in the horizontal position would become syncopic with only a 4 percent loss. If the subject were seated, a loss of only 18 percent would bring the "nonsusceptible" individual to the point of syncope. As the average adult male carries about 6 liters of blood, 18 percent is about 1 liter or approximately 1 quart. Considering the number of potential areas of "pooling," this is not a large volume.

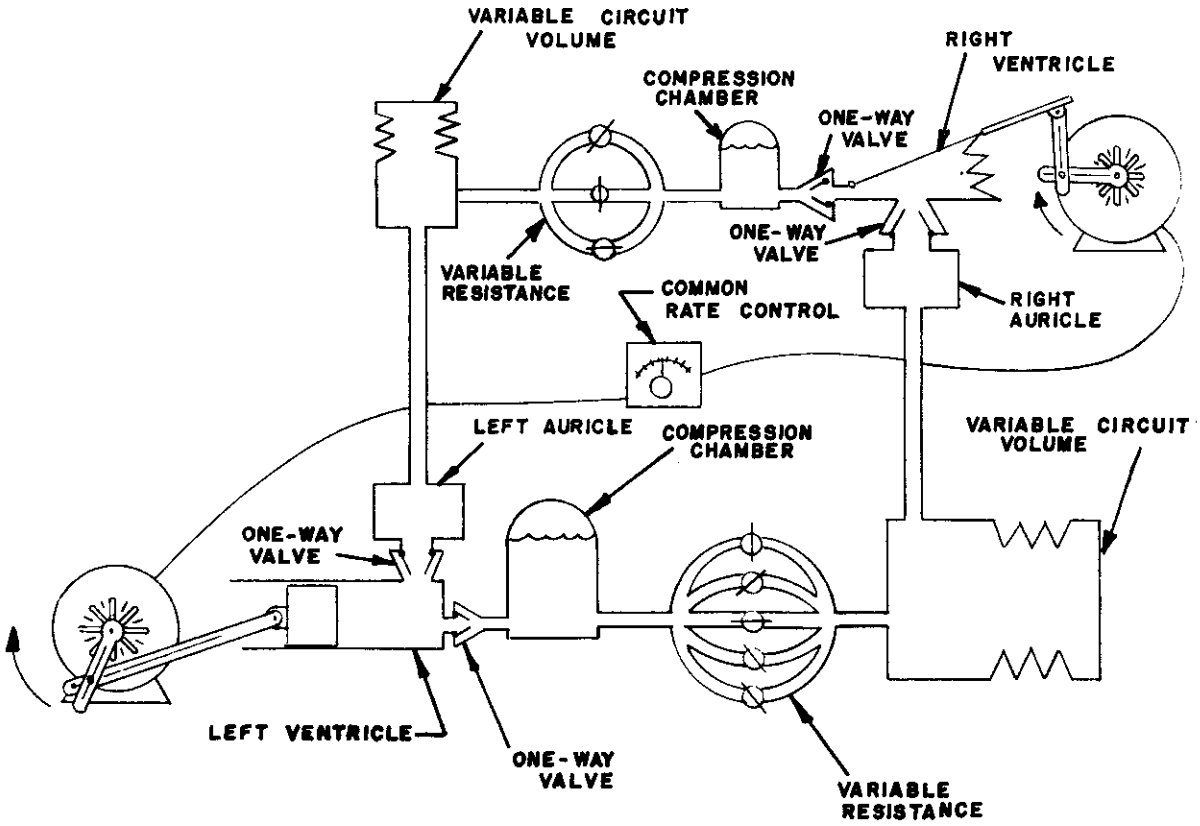
Treatment of some basic cardiac and blood circulation physiology, considered in a more mechanical sense, will provide information leading to a clearer understanding of the functions which contribute to the loss of effective circulating blood volume.

(1) Basic Cardiac and Blood Circulation Physiology

Figure 4, a simple schematic representation of the circulatory system, shows the circuit that the blood follows from the left side of the heart, through the body for delivery of oxygen to the tissues, and back to the heart. The heart mechanism is like two pumps in series with no significant shunts between the two major divisions of the circuit. The hydraulic system connecting the two pumps can accept shifts of volume from one part of the circuit to another only within limits. Therefore, except for brief transition periods, the two pumps must maintain an average output balance, even when one pump has been loaded more than the other or its function has been impaired.

The two sides of the heart pump, the ventricles, differ anatomically and functionally. Whereas the left side is designed to move smaller quantities of blood at high pressure, the right side is designed to move larger quantities at low pressure; the pressure; the pressure in the pulmonary circuit, i.e., from right heart to lungs to left heart, is only about one-sixth of the pressure in the systemic circuit. Hence, the right side of the heart has less reserve than the left side and becomes more acutely stressed during pressure breathing.

A factor compensating for the above is that the traction on the right ventricular wall resulting from contraction of the left ventricle (figure 5) contributes to the bellows action of the right ventricle. The free wall of the right ventricle can be virtually destroyed either by cauterization in dogs (refs. 25, 27) or by occlusion of that part of the coronary blood supply in man (ref. 24) without apparent serious loss of circulatory efficiency in the resting state. With the above impairment, right ventricular output could be maintained only by the effect of left ventricular tension on the free right ventricular wall. Fortunately, the anatomical design of the right ventricle is such that



Left Auricle
Right Auricle > Reservoir Function Indicated

Left Ventricle
Right Ventricle > Major "Pump" Elements

Figure 4. Schematic of Cardiovascular System

minimal myocardial shortening expels relatively large amounts of blood. However, the anatomical design is not adapted to initiating elevated intraventricular pressures (ref. 38). Much higher myocardial tensions are required to elevate intraventricular pressure on the right side than on the left. Since the pulmonary (lung) tree normally offers little resistance, the right ventricle is adapted for handling volumes of wide variation, such as an adaptation, under low pressure, to sudden changes in venous return and central venous volume. But, as an extreme example, sudden pulmonary arterial pressures, as in massive pulmonary embolism, often result in death because the right ventricle cannot maintain the higher pressures needed to sustain pulmonary blood flow. If the need for higher pulmonary pressures is chronic and it does not develop acutely, as in pulmonary fibrosis or pulmonary stenosis, the right ventricle will become more muscular and assume a more cylindrical shape similar to the high pressure-low volume pump of the left ventricle.

As the two ventricles are in series and stroke at the same rate, they must maintain an average output balance, even when one ventricle is under stress or its function has been impaired. As each ventricle responds to variations in the effective filling pressure, i.e., the pressure at the inlet of the pump, a simple servomechanism is provided for output balance between the two pumps. If this balancing mechanism is unable to readjust the two outputs to equal levels, circulatory collapse may result.

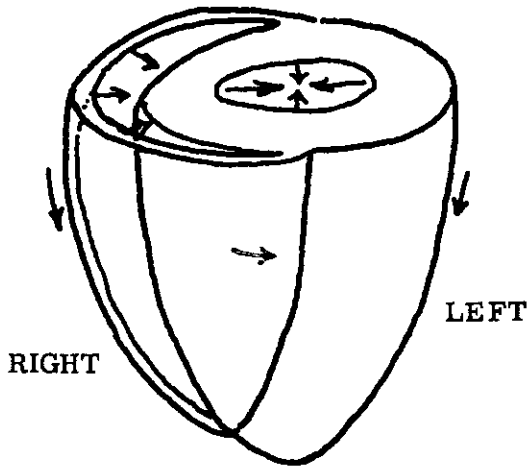


Figure 5. Diagrammatic Sketch of the Heart.

The right side of the heart is built like a bellows, with a large surface area compared to its contained volume. It is adapted to movement of large volumes at low pressures. The left side has a small surface area compared to its contained volume. It is adapted to moving small volumes at high pressures. However, the output of both sides over a period of time is obviously equal.

Pressure breathing imposes a resistance between the two pumps by increasing the pressure in the lungs. It also produces a "tamponade" or compression effect on the heart and great vessels of the thorax. This requires added pressure in the right ventricle to overcome the added resistance. This also causes a "backup" of venous blood since it, too, must build up added pressure to overcome the new intrathoracic threshold and restore flow to the right side of the heart. During the Valsalva maneuver (transiently) increased intrathoracic pressure), X-ray and contrast media have detected the obstruction to venous return at the level of the first rib where the major vessels enter the thorax (ref. 109). Prior to the author's findings, other investigators have observed this pressure rise (refs. 6, 7, 119). Normally, about three-fourths of the blood is in the veins (ref. 8) and has passed the point where oxygen is given off and the waste products are picked up, i.e., the capillaries where arteries and veins meet. With the added pressure, a larger portion of the blood is diverted from the arteries to the veins. As the intrathoracic pressure rises, venous return to the heart is impeded and systemic venous pressure rises because the arterial side of the system continues to supply blood. When the new intrathoracic pressure threshold is exceeded, return flow to the right side of the heart increases, but the effective blood volume has been reduced due to the blood being "pooled" in the venous circuits outside the thorax. Scientists, such as Chadwick (ref. 91), Barach (ref. 92), and Henry (ref. 90), have conducted extensive study on "pooling" during pressure breathing which is generally applicable to the MC-1 investigation. The pooled blood reduces the effective circulation as realistically as if a subject had been bled by the slashing of his wrist. With sufficient time and imbalance, some of the fluid leaves the vessels and moves out into the unprotected tissues to cause edema.

Though more time is required for the larger molecules to cross the capillary barrier, the rapidity of the ion and smaller molecule transitions is amazing. Cowie et al. (ref. 30) demonstrated with tracer techniques that the whole capillary bed is extremely permeable to such constituents as sodium (a 60 percent exchange between intravascular and extravascular compartments is possible within 60 seconds), chloride (a 64 percent exchange), and the water molecule (a 140 percent exchange). The author has observed (ref. 31) that the larger plasma proteins are rather slow in crossing the barrier.

Since water molecules can easily move between the blood and tissues and intracapillary pressure greatly exceeds extravascular pressure during pressure breathing, why is it that "water" does not flood out into the tissues? The probable explanation

is that not only is the tissue osmotic pressure increased, but also the increased transudation causes hemoconcentration only to the point where the increased osmotic pressure of the concentrated blood forces a new equilibrium at a higher level. Incidentally, excepting the most brief tests, hematocrits rise during the MC-1 runs. The filtration barrier depends mainly on five factors: (1) capillary hydrostatic pressure, (2) tissue hydrostatic pressure, (3) intracapillary osmotic pressure, (4) tissue osmotic pressure, and (5) capillary permeability. The ultimate fluid redistribution effect during pressure breathing depends on such diverse factors as adequacy of counterpressure, physical fitness (althletes have a significantly larger blood volume and many more capillaries), hydration, cardiac and vascular response, and all important changes in vascoconstrictive tone. Undoubtably, part of the loss of effective volume is offset by depletion of some of the blood reservoirs. The spleen, for instance, can contribute 200 to 250 cc. which is small compared to the potential of the liver or the highly reactive mesenteric vessels. The blood displaced from the thorax by pressure breathing is another "autotransfusion." Also, the subpapillary plexus of the skin is a potential contributor. Varying degrees of pallor (vasoconstriction) of many individual are often seen for significant lengths of time during the compensatory efforts preceding the advent of the pallor which prompts termination of a pressure suit run. Other investigators (ref. 32) have observed that pressure breathing causes vasoconstriction in the fingers.

Contrary to the common nonscientific misconception, most of the compensation for volume alteration must be made by reduction of the venous compartment under neurohumeral influence. As the capillaries and the veins hold, respectively, 5 and 75 percent of the total blood volume, the arteries contain only 20 percent of the total supply. A constriction of an arterial system which would reduce the volume of the system by 50 percent would displace less than a pint of blood; it is questionable from a physiological viewpoint that such a constriction would be possible and advantageous. The vasoconstrictive efforts of the body during pressure breathing can be easily obscured by passive venous pressure change if an attempt is made to measure volume changes plethysmographically. Yet, Fenn et al. (ref. 28) were able to demonstrate the vasoconstrictive effect by blocking the passive pooling with an inflated cuff. As mentioned above, this vasoconstriction can be observed in the eyegrounds during a pressure-breathing imbalance episode.

Although some investigators (refs. 34, 36) have contended that a significant quantity of blood might be pooled within the pulmonary circuit itself during high levels of continuous positive pressure breathing, X-ray and isotope work in this field have failed to support this belief.

The capstan principle, originally developed by Lamport et al. (ref. 93) and applied to pressure suits by Drury and Henry (ref. 94), works satisfactorily on tubular portions of the body, such as the limbs. However, as the capstan bridges a body concavity, it cannot be directly applied to the surfaces within the depression. Since the pressure suit does not provide sufficient counterpressure in such areas as the trough of the back, crease of the buttocks, groin, shoulder area (excess cloth must be positioned here to allow adequate shoulder movement), around the blunt edges of the MC-1 chest bladder (see figure 6), etc., the body develops its own tissue pressures hydrostatically by withdrawing fluid from the circulatory blood which further depletes the subject of available circulatory volume.

If an area, such as a hand, is left unprotected at altitudes above 65,000 feet, edema is augmented by vaporization of fluids beneath the exposed skin and in unprotected joint areas which results in a painless emphysema (see figure 7), termed "vapo-edema" by Rosenbaum (ref. 137). If the gas moves beneath the suit edge or if the atmospheric pressure is increased, it becomes pressurized and cannot escape. The gas cannot enter the blood stream because both arterial and venous pressures are too high and the vessel integrity remains intact. The author and Rosenbaum have shown that this

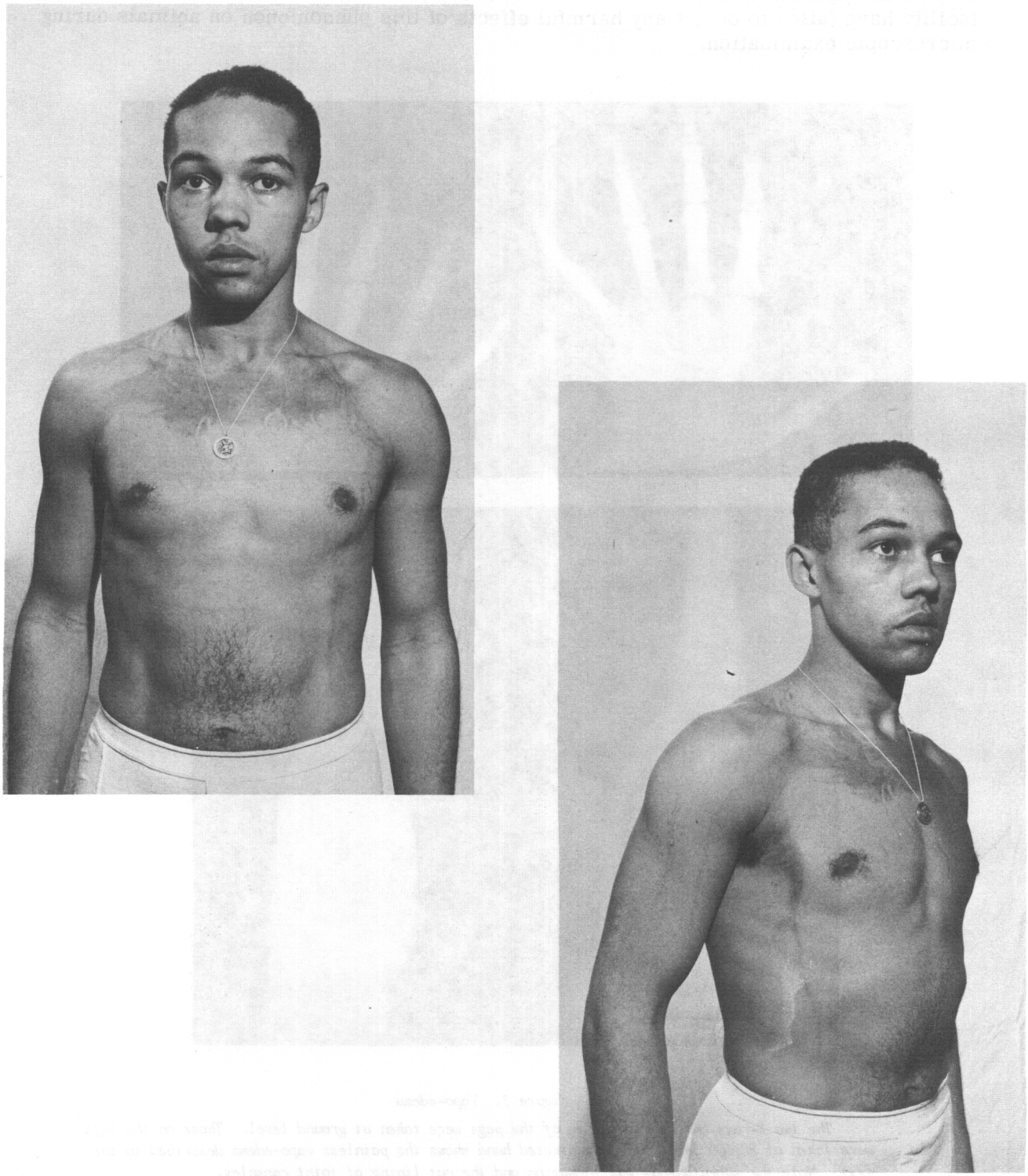


Figure 6. Ecchymosis Following Pressure Suit Runs

This subject shows obvious evidence of ruptured blood vessels in areas where the chest bladder's blunt edges created unprotected areas. These are good examples of the potential "pooling" areas. The lines across the ecchymotic area result from the placement of small EKG leads down the neck to the chest.

phenomenon will occur not only on terminal portions of limbs, but also wherever there may be an exposed unpressurized gap. However, pathologists associated with this facility have failed to detect any harmful effects of this phenomenon on animals during microscopic examination.

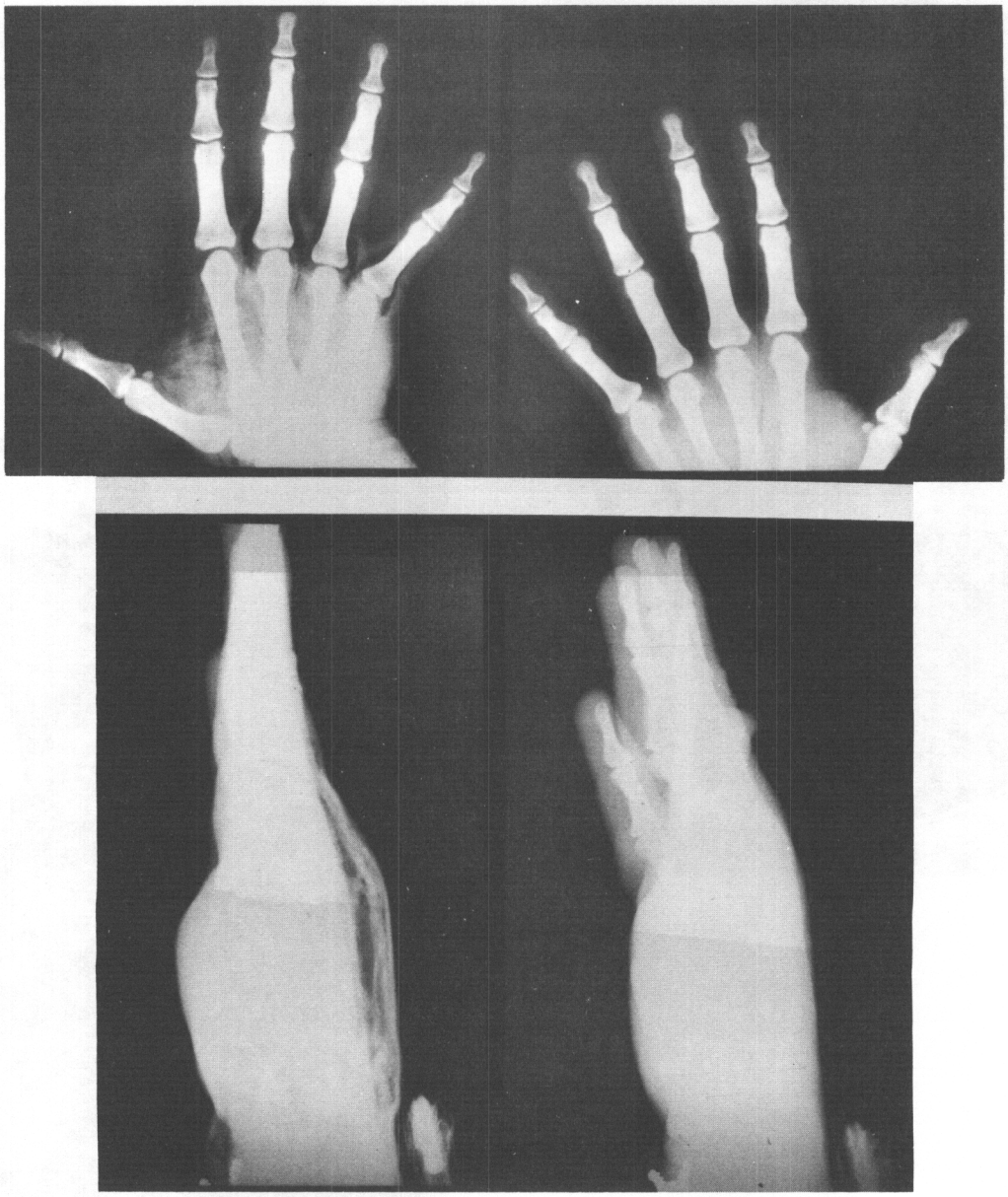


Figure 7. Vapo-edema

The two X-rays on the right side of the page were taken at ground level. Those on the left were taken at 80,000 feet. The unprotected hand shows the painless vapo-edema described in the text. Note the subcutaneous gas formation and the out lining of joint capsules.

As the subject is breathing dry 100 percent oxygen, his lungs must contain a constant water vapor pressure to function properly. With each exhalation, he loses more moisture to the dry environment, thus tapping his fluid reserves.

The conglomeration of all these factors appreciably reduces the effective circulating volume and minimizes the extent of time which can be endured at altitude.

b. Adjustment to the 40,000-Foot Equivalent

As discussed in Section II, 3, adjustment to the 40,000-foot equivalent causes an added compensatory strain and a mild deficiency in blood saturation.

c. Cardiac Strain from Imbalance

The heart muscle itself must receive sufficient oxygen to function properly. If more oxygen is needed to perform additional work, the heart muscle must acquire it either by extracting more oxygen from the same amount of blood or by increasing the blood flow through the heart muscle supply vessels, the coronary arteries. Active skeletal muscle can not only greatly increase the amount of blood flow through the muscle, but it can also more than double the amount of oxygen it can extract from a given amount of blood. However, a characteristic of heart muscle is its constant functioning at very high oxygen extraction.

With the heart in the "resting" state, oxygen extraction from the blood passing through the coronary system approaches 70 percent of complete extraction. A complete 100 percent extraction is not physiologically possible in any tissue. Since the percent of the extraction is so high in the myocardium, the heart has relatively little reserve of venous oxygen. Therefore, to meet increased demands, it must depend primarily on an increase in coronary flow. Even in advanced congestive heart failure where the cardiac output is greatly reduced, the blood flow is reduced to all tissue, even the brain, but with exception of the myocardium. All other tissues compensate primarily by increased extraction of oxygen from each unit of blood. As indicated above, adjustment to the 40,000-foot equivalent in the partial pressure suit has already reduced the amount of oxygen available in each unit of blood.

Although an increased blood flow would appear as the preferable means of acquiring the needed oxygen because of the limitations of increasing the percent of oxygen extraction from the blood, there should be consideration of the path of the coronary arteries. These arteries originate on the left side of the heart at the point where the main pipe (aorta) leaves the left ventricle, then turn back into the heart muscle (myocardium) to supply both the right and left sides, and finally empty into the right side of the heart. The heart could increase the flow by raising the pressure gradient between the left and right sides. But pressure breathing with insufficient counterpressure raises the pressure on the right side. As noted previously, rather than raising the pressure on the left side, it has been lowered since, in reality, the "hypertension of pressure breathing" is a relative hypotension. Thus, development of the coronary pressure gradient has been retarded rather than aided. This decrease in the effective pressure gradient would not be so critical were it not for the simultaneous reduction in the amount of oxygen in each unit of blood accompanying the adjustment to the 40,000-foot equivalent and other factors still to be enumerated. The reduction in the effective pressure gradient during pressure breathing has been demonstrated in both humans and animals by catheterization techniques; both the right side of the heart and the base of the aorta were catheterized. Demonstrations were performed as follows: on humans by the author, Capt. Frank J. Leary, USAF, MC, and Dr. Denmark; on animals by the author, D. Rosenbaum, and Capt. Dale Smith, USAF, VC. Recent dog work performed for other purposes at the National Institute of Health (ref. 29) and at Ohio State University (ref. 100) substantiated the demonstrated reduction.

Without a coronary gradient pressure, the heart could attempt to increase coronary flow by increasing its rate which would thus increase the blood output per unit

time from the left side of the heart (left ventricle). Actually, the heart rate is forced reflexively to increase to a certain extent by the reduced effective circulating blood volume of the body. The relationship between central blood volume and heart rate has been demonstrated. A carefully calibrated workload was performed both before and after experimental pooling of blood in the legs (ref. 9). The latter was accompanied by lower cardiac output and higher heart rate, implying that a larger available central blood volume allows slower rates and higher stroke volumes. Similar findings were reported after phlebotomy (ref. 10) and heavy fluid loss from excessive perspiration (ref. 11). Consideration of another heart characteristic is prompted at this point of the discussion. Whereas the blood flows best through the coronary arteries when the heart is relaxed, i.e., between contractions, circulation through the heart is greatly impeded at the moment when the heart contracts. Since the coronary vessels are imbedded within the ventricular wall, ventricular contraction compresses them and, thereby, obstructs flow. Gregg et al. (ref. 33) demonstrated that coronary flow during diastole is greater than during systole by an average of 2.4 to 1. Tachycardia markedly reduces diastolic intervals and proportionately decreases coronary flow unless coronary dilation can take place. Cardiac output is more efficiently increased by greater stroke volume than by tachycardia because of both greater mechanical efficiency of the myocardium itself and proportionately greater coronary blood flow. The myocardium requires more oxygen to sustain a given cardiac output by increased rate than by increased stroke volume with a slow rate. Tachycardia can very effectively increase cardiac output to meet a sudden need, but this increase would be performed at the expense of efficiency in oxygen consumption, ventricular contraction, diastolic filling, and, at higher rates, coronary flow. Thus again, in pressure breathing with inadequate counterpressure, the heart would be forced to use a less efficient mode of compensation.

This relationship between central blood volume, tachycardia, and coronary flow undoubtedly explains the very rapid onset of marked EKG indications of myocardial hypoxia as experienced in the "pulse climber," i.e., the altitude chamber.

Increased stroke volume is the most efficient means by which the heart can improve its condition. Augmenting minute volume by increased stroke volume is more economical than by increased rate because a greater coronary flow per unit work is produced. Unfortunately, but few subjects can compensate by use of this means because of other physiological alterations during uncompensated pressure breathing. The author and Capt. Frank J. Leary, USAF, MC, have demonstrated that MC-1 performance can be improved by adding 1000 cc. of either Dextran or whole blood to the subject's circulating volume. This injection allows the individual to maintain a larger central blood volume, despite pooling and edema, and thus to operate at increased stroke volume. The fact that the Dextran acted primarily as a volume expander without appreciably aiding oxygen transport was considered significant. Following the injection, the subjects noted definite improvement in contrast to the previous runs. Objective measurements showed improvement in many factors, but the EKG changes still followed their familiar progressive path, though more slowly. This is not surprising because, whereas the fluid volume had been improved which indirectly aided the heart, the intrathoracic imbalances which are largely responsible for the myocardial hypoxia had not been altered.

Other methods failing, still the heart has alternative means of increasing coronary flow since resistance to coronary flow can be affected by certain neurological and hormonal influences. Excitement causes increased activity of the autonomic nervous system, a regulating system which controls the many automatic functions of the body, e.g., heart rate, blood flow through muscles, sweating; it also causes release of catecholamines, i.e., hormones, such as adrenalin or epinephrine and norepinephrine. Both epinephrine and norepinephrine cause the coronary arteries to dilate. But epinephrine also greatly increases heart rate, raises oxygen consumption of the heart, and more than negates the augmented coronary flow by an excessively increased

myocardial oxygen demand. The cardiovascular system becomes more efficient as the ratio of norepinephrine to epinephrine is increased.

Epinephrine is liberated from the medulla of the adrenal gland or the sympathetic nerve fibers and fed into the blood stream by direct neurosecretion. Norepinephrine is a nonmethylated precursor of epinephrine, and it is liberated from the same sources of the epinephrine, but mostly from the sympathetic fibers. Internally secreted or injected epinephrine elicits various degrees of cardiac acceleration, depending on the amount of reflectory vagal interference (ref. 40). Injected norepinephrine regularly slows the heart (refs. 41, 42, 43), probably as a result of vagal stimulation initiated by elevated blood pressures in the carotid sinus (refs. 41, 44). The carotid sinus is reported to be specially sensitized by norepinephrine (ref. 45) and possibly by reflexes originating in the walls of the heart (refs. 46, 47). Vagal section or the use of atrophine, a parasympathetic blocking agent, causes both epinephrine and norepinephrine to act solely as accelerators of heart rate (refs. 40, 46, 48). The effects of injected norepinephrine on the heart and vascular system are not the same as those resulting from endogenous production. This is because their normal secretion is usually the result of strong sympathetic discharges which tend to increase sympathetic tone, whereas exogenous injection leads to an increase in parasympathetic tone by stimulating the baroceptors of the carotid sinus, aortic arch, etc. Epinephrine or norepinephrine liberated endogenously elevate the heart output by increasing both the rate and stroke volume to varying degrees. However, evidence indicates that epinephrine so increases the oxygen consumption of the myocardium that the accompanying active dilation of the coronary arteries (ref. 49) and increased coronary blood flow (refs. 50, 58) are insufficient to offset the uneconomical use of oxygen, causing the myocardial oxygen demand to exceed the supply (refs. 50, 59, 61). As stated above, the condition is improved if more norepinephrine than epinephrine is liberated. Despite concomitant reduction in cardiac workload and simultaneously increased coronary artery flow, it has been demonstrated that myocardial hypoxia results from stimulation of the cardiac sympathetic nerves (ref. 59). Acetylcholine and vagal stimulation apparently exert antagonistic oxygen-conserving action (refs. 62, 65) on the myocardium, especially in offsetting epinephrine. The sympathetic-parasympathetic (vagal) interplay with its biochemical influence upon the heart is of obvious importance; the sympathetic and parasympathetic portions of the autonomic nervous system usually act antagonistically in that system's balance, i.e., one portion tends to dilate vessels in various areas while the other tends to constrict them. The exact mode of action of the catecholamines on the heart has not as yet been completely determined. However, in the case of any hypothetical individual involved, the heart is not operating as economically; and, in partial pressure suits at altitude, this condition, unfortunately, is worsened by the reduced amount of oxygen available in the blood. Hence, it is only of academic interest whether this decreased economy of heart operation may be attributed to a direct cellular metabolic change or to the heart's shifting to a "descending limb" on a "Starling" curve or to a less desirable curve in adjusting to the situation (see figure 8).

As the subject has been depleted of part of his effective circulating blood volume and the blood leaving the lungs contains less oxygen than it ordinarily would at ground level, the heart is virtually forced to increase its rate which is an uneconomical method of increasing cardiac output. At the same time, the pressure gradient across the myocardium has been lowered and coronary flow per unit has been reduced. As a result, the heart itself becomes hypoxic, regardless of whether or not adequate oxygen is reaching other tissues. In relation to myocardial hypoxia, decreased cardiac efficiency follows a decreased oxygen supply. Obviously, this type of vicious cycle can end in circulatory collapse.

As the heart has been penalized and stressed, it attempts to increase or at least maintain its output. But the output falls significantly in most individuals. If the cardiac output falls, this affects how much oxygenated blood reaches the brain and other

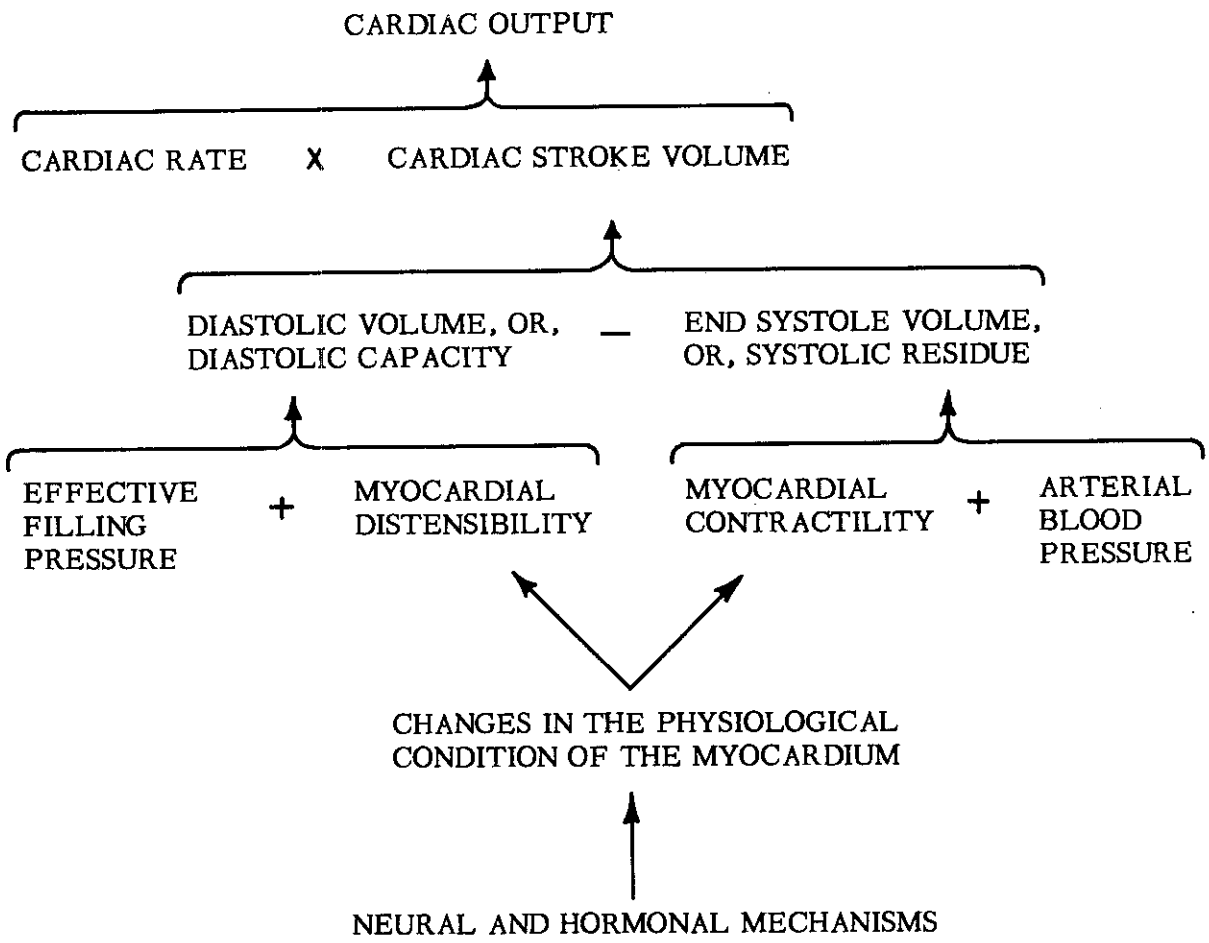


Figure 8. The Determinants of Cardiac Output

tissues. As the myocardium becomes more hypoxic, it functions less well and the output falls further with circulatory collapse ensuing.

Patients with advanced pulmonary fibrosis, such as stenosis of the pulmonary valve of the heart, or some other condition which greatly raises the load on the right side of the heart are observed occasionally in clinical medicine. Yet, many of these people compensate excellently for years for an increase in resistance which exceeds that imposed by the MC-1 tests. There are several reasons to explain this compensation.

First, the right side of the heart has had ample time to change anatomically from the low-pressure type of pump described above to the high-pressure type, similar to the left ventricle of the heart.

Secondly and more important, in the situations described above, the venous return is unimpaired and blood volume at the inlet of the pump (right ventricle), if there is any change, increases. Since the availability of blood volume at the inlet of the pump (central venous volume) considerably affects the pump's ability to compensate for resistance at the outlet, the importance of this increase of blood volume is obvious. The theory of the "family of Starling curves" (refs. 11, 12), which is becoming increasingly more accepted, further elucidates and strengthens this point. While breathing with

unbalanced pressures, as in the MC-1 ensemble, the central venous volume is reduced. Also, blood does not "pile up" at the inlet of the pump (right ventricle) during pressure breathing because the inflow threshold is at the inlets to the thorax and not at the heart itself. There is a definite relationship between central blood volume and ventricular filling pressure on one hand and ventricular work on the other (refs. 13-15). This can be plotted as either a "ventricular function" or "Starling" curve. The height and shape of this curve can be greatly altered by many factors, such as reduced coronary flow (ref. 37), hormones (ref. 38), etc. Rather than "absolute" ventricular function curve, there is a family of curves. Basically, reduced oxygen to the myocardium depresses the ventricular function curve (ref. 37) which leads to a descending spiral involving reduced cardiac output, reduced coronary profusion pressures, and higher arterial pressures. The author and Capt. Frank J. Leary, USAF, MC, initiated a carefully controlled cardiac catheterization study, utilizing the catheterization experience of a local medical facility (ref. 39). This study added supportive information to this problem area.

Thirdly, due to the fact that the cardiac reserve has been compromised by the 40,000-foot equivalent and the imbalance in the suit, tolerance to physical exertion is markedly reduced in the inflated MC-1 ensemble. The cardiac output findings acquired in the MC-1 pressurization tests have shown a significant drop in cardiac output. The condition is similar in some aspects to that of the individual having a congestive heart failure whose resting cardiac output may be abnormally low even though the resting oxygen uptake may be unaltered. But, in congestive heart failure, the venous reserve is utilized in greater proportion and oxygen extraction increases in all tissues except the myocardium where, as indicated before, oxygen extraction always approaches a maximum. Even cerebral blood flow is reduced in a person having this failure, and, when he attempts to exert himself even mildly, the unpleasant symptoms resulting from lack of reserve tend to discourage the effort. Although physical reserve is definitely reduced in the MC-1 ensemble, the exercise limitations are, of course, not as severe. Human variability, discussed later, is an important factor in this consideration.

d. Reflex Activity

The stimulation of sensitive nerve centers by the MC-1 ensemble adversely affects the time-altitude capability. The area of the carotid sinus pressoreceptor (pressure-sensitive) centers responds to the stretching of its walls resulting from increased internal pressure or from pressure applied externally. The centers lie bilaterally (either side) in the neck a short distance below the angle of the jaw where these two main arteries (the carotid arteries, one on each side) to the head divide into two branches each. When the reflex is activated, it usually causes the heart to slow (bradycardia), cardiac output to fall, blood pressure to fall, reduction in the force of myocardial contraction, dilation of peripheral vasculature, etc. Also, a local reflex acting only on the brain can sometimes cause collapse by affecting cerebral (brain) blood flow (ref. 21). Efforts are made in the practice of judo to stimulate directly these areas. Stimulation of the carotid sinus can also cause on occasion significant reflex EKG change (ref. 123), including cardiac standstill (ref. 112). Thus, the carotid sinus mechanisms can cause syncope by cerebral, cardioinhibitory, or vasodepressor mechanisms (ref. 132). Unfortunately, the neckseals of the K-1 or MB-5 helmets can rest across these sensitive areas, causing continuous external pressure. Also, as shown by recent investigations, nerve endings with functions similar to that of the carotid sinus extend down the common carotid artery toward the base of the neck. With the MC-1 ensemble in place, a wide gap along the neck without the effect of counterpressure extends down from the neckseal of the helmet to the top of the suit which reaches only to the base of the neck. Although the pressure inside the carotid artery in the area of the gap may remain the same, the reduced ambient pressures of high altitude would cause a pressure differential and subsequent stimulation of the vessel. Further, the

seal often rode over the laryngeal prominence, another pressure-sensitive area. The riding can disable by either fracturing the larynx which leads to laryngeal spasm and strangulation by resultant gross edema of the laryngeal passages or causing sudden intense reflex activity that can cause slowing or even stoppage of the heart. Unfortunately, the K-1 or MB-5 neckseal often compromised three of the seven most pressure-sensitive areas above the shoulders.

Actually, the whole respiratory tract is quite active reflexively. Stimulation of the pharynx, hilus or "root" of the lung, etc., in surgery accounts for a significant percentage of cardiac standstills (ref. 101). Considering the stimulating effect, it is noted that the syncope of a paroxysm of coughing is more easily produced in the MC-1 situation. Not only is the reflex itself more sensitive and the situation potentiated by an already existent relative pressure increase in the thorax and by reduced oxygen tensions, but also the dry oxygen can, after a time, cause sufficient irritation of the upper respiratory tract to precipitate a coughing paroxysm in some individuals. As evidenced by X-ray study, there is also a significant distention of the upper respiratory tract above the shoulders because of the lack of external balancing pressures. The following areas are extremely active reflexively: the upper respiratory tract (refs. 66, 68), the entire respiratory tract (refs. 113, 114, 122), the pulmonary vascular bed, and the great veins of the thorax (refs. 115-118, 126). To be added to the above are sources of stimulation, such as subclinical "bends" and gaseous distension of the intestine, resulting from reduced atmospheric pressures.

In completing this brief discussion of reflex activity, it should be noted that (a) since these separate sources of stimulation have the same final pathway of effect, they can summate or strengthen the effects of each other centrally and (b) their effect is potentiated further by blood loss (pooling) and by reduced blood oxygen saturation when adjusting to the 40,000-foot equivalent.

4. Significance of Anxiety, Individual Variability, Physical Fitness

a. Anxiety

The importance of anxiety has been noted frequently (refs. 124, 125, 133-135). As severe anxiety lowers the tolerance to physical stress, its effect on cardiac function is relatively unchallenged. Some subjects evidencing apprehension during the MC-1 tests were definitely aided by distraction. Severe anxiety can produce cardiac embarrassment or even sudden death in "susceptible" individuals (refs. 69-74). An extensive report (ref. 75) on sudden death in young soldiers contained a group of at least 140 carefully investigated sudden deaths, with essentially normal post-mortems. It has been estimated that at least 10 percent of all sudden, nontraumatic deaths are in apparently healthy young men and are assumed to be largely the result of reflex cardiac standstill or arrhythmia. Aside from such unequivocally negative autopsy and toxicological reports, while there are many more which noted abnormalities, it is questionable whether these abnormalities were important in contributing to the sudden death. In the same study, another group of about 300 cases showed some coronary sclerosis at autopsy but 55 percent of these showed no evidence of occlusion or thrombosis. It is of interest to note that the average Strategic Air Command pilot is in a "precoronary" age group and statistics would imply that the majority of these pilots have some degree of coronary sclerosis. Two extensive and separate studies of sudden death, one in Europe and one in this country (ref. 76), found coronary sclerosis, with and without occlusion or thrombosis, in 66.3 and 67.7 percent, respectively, of those examined. However, there is serious doubt of the importance of slight or moderate sclerotic changes in the coronaries as the ultimate cause of death. Beck (ref. 77) believes it is likely that sudden death in the apparently healthy is induced by influx of excess amounts of the adrenosympathogenic catecholamines into the myocardium which work via their "hypoxiating" effect with

local coronary sclerosis to produce areas of greater myocardial hypoxia. Then, these irregular areas of hypoxia render the heart electrically unstable and initiate fatal ventricular fibrillation.

The following treats of the relationship of anxiety to angina pectoris (heart pain) in those with impaired myocardial circulation. Physicians report that innumerable patients relate that their cardiac pains occur only, or especially, under the aggravating influence of mental tension, frustration, anxiety, anger, or simple awareness of purpose. Conversely, without such influence, they can perform much heavier physical labor without suffering the pain. Angina under purely emotional stimulus is quite commonly evidenced (ref. 69) in those with impaired myocardial circulation. Similarly, EKG changes are observed in those of presumably normal cardiovascular status (refs. 124-128). However, it is difficult to quantitate the stimulus with the symptoms. Emotional stress causes an increased cardiac output and accelerated circulation (refs. 78-83) which are similar, in most aspects, to observed reactions to small doses of epinephrine (refs. 79, 83). Also, EKG changes seen in anxiety show changes similar to those induced by small amounts of catecholamines (refs. 84-88). Good examples of EKG abnormalities attributed to anxiety were observed in subjects resting quietly in partial pressure suits on 100 percent oxygen prior to the simulation of reduced ambient pressures in the altitude chamber. The incongruities between the degree of cardiac work and the occurrence or nonoccurrence of cardiac pain and/or EKG changes are explained largely on a neurohormonal basis. The crux of the entire situation is that anxiety can prejudice the cardiac function to varying degrees: although motivation also plays an obvious part, it is not synonymous with anxiety. As observed during the altitude chamber runs, anxiety can compound the cardiac condition of a subject under stress in an MC-1 ensemble and greatly influence his time-altitude potential. Although stress experienced in newer equipment is much reduced, it would be hazardous for an aircrewman of questionable cardiovascular status to ascend to altitudes where the effects of reduced oxygen supply, anxiety, and some degree of cardiovascular stress could summate. Although an adequate cardiovascular system and a perfectly functioning respiratory system are physiologically intertwined, the former is much more important to the potential combat aircrewman. Figure 9 exemplifies how the efficiency of the heart can be grossly affected by neurohumoral mechanisms.

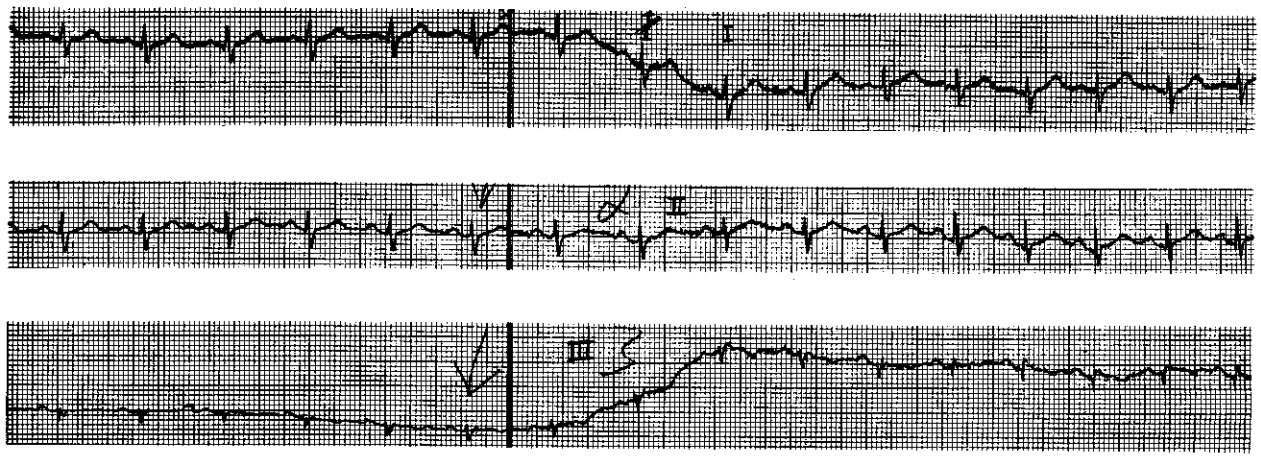


Figure 9. EKG Changes During Decompression with Subject Wearing MC-3 Ensemble

One-second decompression from 38,000 ft to 65,000 ft in MC-3 ensemble. Note absence of EKG axis change see with MC-1 ensemble where torso counter pressure is inadequate.

The use of tranquilizing drugs to offset anxiety in the high altitude situation can be only decried. The author and Capt. Frank J. Leary have demonstrated that the reduction of the ability to compensate for the stresses experienced at altitude in the pressure suit more than offsets the calming effect of such drugs. Because of the subduing effect that the drugs have on autonomic reactivity, they reduce tolerance to everything from oxygen reduction to pressure breathing. Although the subjects with severe anxiety changes had better ground level readings and were more subjectively comfortable at altitude while on tranquilizers, their physiological deterioration rate was greatly accelerated at altitude.

b. Individual Variability

The ability of aircrewmen to remain at 65,000 feet in an MC-1 ensemble ranged from 2 minutes to more than 2 hours. While in the horizontal position (refs. 103, 104), some men became syncopic after only a 4 percent blood loss, whereas others were not disturbed up to a 25 percent loss. Whereas several minutes of the tilt-table stimulation exceeds the apparent capacity of one man, another may be unaffected after several hours of such stimulation (ref. 105). There are many examples of such variability (refs. 106-108, 110). Although two F-104 aircraft perform essentially the same, the stress capacities of their pilots may differ considerably.

Purely on the basis of a careful physical examination and personality evaluation, the author could predict with a high degree of accuracy a short, medium, or long time-altitude ability of each subject in an MC-1 ensemble. The assessment was a "clinical judgement" which was based primarily on an evaluation of autonomic nervous system activity and included the following observations: general muscle tone, pupillary response during ophthalmoscopic examination, how easily the subject blushed, how much the subject sweated or flushed when performing a Harvard step test evaluation for physical fitness, and personality type. The last aspect was considered since there is a recognized relationship between personality and emotional activity on one side and autonomic activity and the ratio of certain key hormones on the other. It became evident that it was extremely difficult to convey this subjective approach using clinical judgement to another investigator without his having acquired the actual experience. It would be most desirable to have a simple objective test or test series which would be of proven significance in indicating premium reaction types and could be easily administered anywhere by relatively unskilled personnel. In general, the aggressive independent individual performs much better than the more submissive type.

c. Physical Fitness

Although correlation of performance with five subgroupings of physical fitness in an unselected flying population was rather poor, most of the few truly superlative performers did rank rather high in exercise tolerance and "physical fitness." Age and gross anthropological subdivision, with one known exception in the latter, did not correlate significantly with performance due to the overriding importance of other factors. Preliminary results of a study to determine the importance of physical fitness indicated significant improvement in general stress tolerance with increased physical fitness.

Improvement in physical fitness should make some distinct contribution to the increase in stress tolerance for a number of reasons: (1) improved total circulating blood volume, (2) increased hemoglobin and myoglobin, (3) vastly increased (estimated tenfold) capillary beds for more efficient metabolic exchange, (4) better oxygen utilization index, (5) lower oxygen debt incurred per unit work, but greater maximal oxygen debt possible, (6) less rise in blood lactate per unit work, but higher maximal blood lactate tolerated, (7) increased tolerance to CO_2 alterations, (8) more efficient

blood-buffering system, (9) increased vital capacity, (10) greater maximal sustained pulmonary ventilation, (11) more efficient handling of body temperature problems, (12) much greater body glycogen stores, (13) better coordination and more efficient muscular movement, (14) advantageous alteration of autonomic nervous system tone, and (15) greater cardiac efficiency.

The athlete has superior qualities of efficiency, endurance, and metabolic safety. Top athletes may have as much as 160 percent of the blood of the average man (ref. 102). Though basically vagotonic, the athlete maintains simultaneously a potent sympathetic neurosecretory functional reserve upon which to draw when necessary. During exercise the athlete's heart increases output by markedly increased stroke volume, thus requiring less oxygen than an equal output produced by acceleration. Cardiac reaction to catecholamines is altered in that the athlete's acquired vagal predominance yields to adrenergic effects during exercise in such a way that the adrenosympathogenic myocardial hypoxia is prevented and optimal oxygen economy is guaranteed. Physical improvement would be highly desirable for the average combat aircrewman. This should not imply that each aircrewman should become an outstanding athlete since the expenditure in time and effort would not warrant it. Yet, regular exercise, not of an extreme nature in severity or time involvement, can increase blood volume by 25 percent (ref. 87) which is important to partial pressure suit duration as previously discussed.

Observations of physiological compensation have revealed that degrees of improvement vary widely among different individuals. Whereas some people cannot adapt adequately, even in a year's time, to the physiological stimulation of living at an altitude approaching 15,000 feet (ref. 95), other of similar physical stature compensate without apparent difficulty. Similarly, some people of apparently fine physical fitness who attempt mountain climbing cannot adjust physiologically to the high altitudes, even after weeks expended in compensating at lower altitudes. Such climbers become incapacitated at camp sites as low as 18,000 feet. Yet, others can camp as high as 23,000 feet for weeks and climb as high as 28,000 feet without supplemental oxygen (ref. 136).

D. MC-3 Partial Pressure Suit and the MA-2 Helmet

1. Development and Description

More effective counterpressure and balance than that provided by the MC-1 were mandatory if suit efficiency was to be improved. With the MC-1 and an adjustment to the 40,000-foot equivalent, the aircrewman would have a counterpressure of only 3 psi in contrast to the normal 14.7 psi at sea level. To achieve the desired balance, the author directed construction of a suit which contained a double-layered airtight lining (figure 10) extending from neck to midthigh. This encompassed the torso with a "balloon" whose outward expansion was resisted by the retaining effect of an inelastic outer shell. All edges of the bladder at exits where the arms, legs, and neck protruded were tapered. The front opening was tapered and overlapped to prevent blunt edges. To produce a situation as much like ground level as possible, the torso bladder was hooked into the breathing pressure line by a "T" connection, allowing a pressure equal to the breathing pressure to surround the torso.

Next, to increase protection around the neck and reduce reflex stimulation from this area, a biblike extension of the neck seal was fabricated. This seal, which was based on a hand and shoulders cast of a fellow officer, extended down under the edge of the suit bladder system and then inverted to cover the neck.

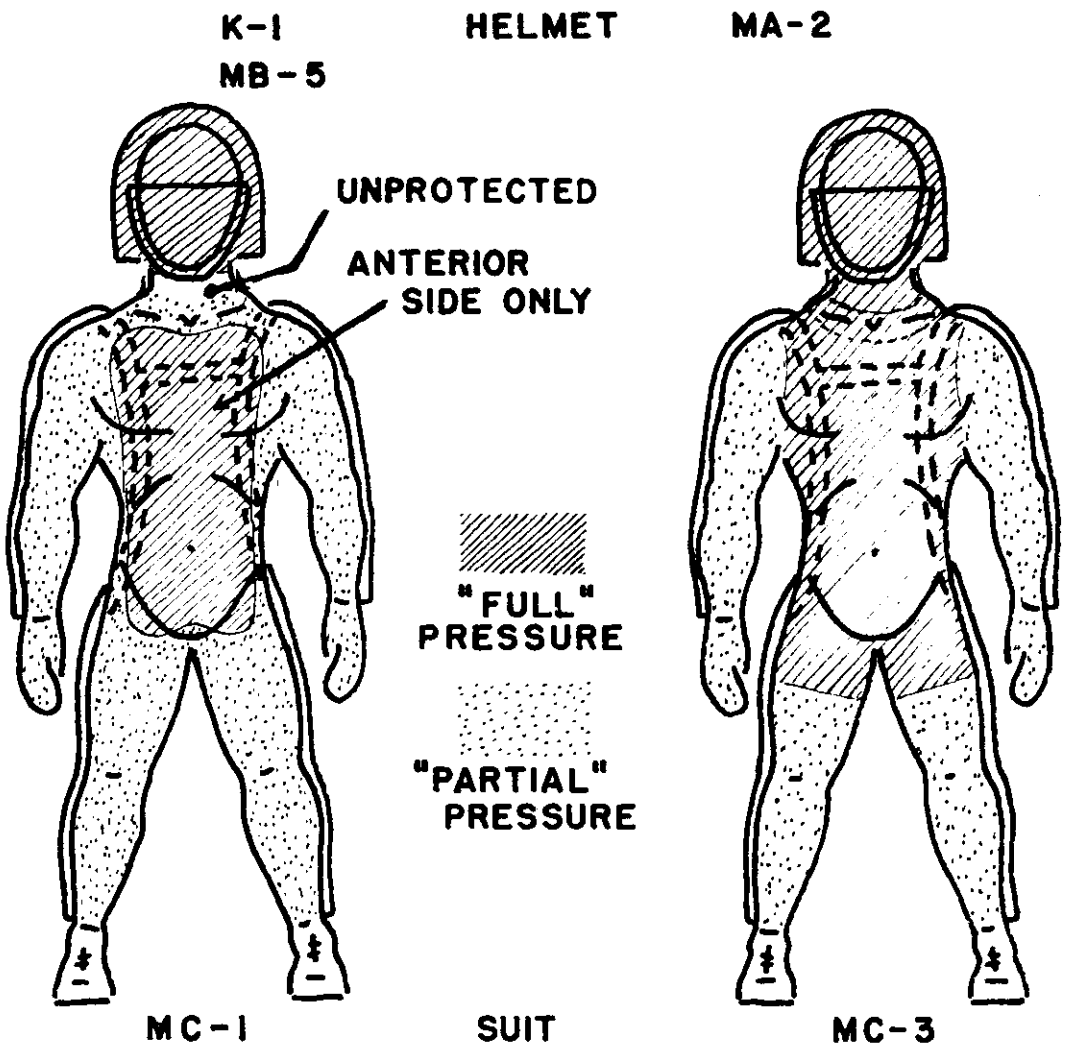


Figure 10. Comparison of MC-1 and MC-3 Pressurized Areas

The combination of the "bladder suit," later termed MC-3 (MC-4 contains a G-suit), and the long neck seal addition to the K-1 helmet, now designated as the MA-2 helmet, gives "full pressure" coverage from top of the head to mid-thigh, with only the extremities under "partial pressure" (see figure 10). As the extremities are of tubular form, they are well counterpressurized by utilizing the capstan system.

The capstans, although overridden by the bladder system, were purposely retained in position on the torso. If the torso bladder were pierced, it could be closed off by a simple disconnect valve and the back capstans would then function, providing the equivalent of a T-1 type suit which would suffice as a "get-me-down" garment. Also, the capstans would help to "contain" the torso bladder.

2. Advantages

The advantages of the MC-3 partial pressure suit ensemble are summarized as follows:

a. The problem of reduced effective circulating volume was minimized

considerably because the bladder fitted more closely into the numerous concavities and produced essentially balanced pressures over the trunk (see figure 10);

b. Also because of the torso pressure balance, the cardiac strain was greatly mitigated;

c. The problem of stimulation or oversensitization of reflex mechanisms was greatly reduced;

d. As respiration could be performed in a near-normal pattern so that breathing was no longer a task, there was the psychological gain of allaying the apprehension of persons in the pressurized state;

e. Even though the time extent at altitude was much prolonged, the incidence of bends was reduced by a third which was probably due to two factors: improvement of the general circulation which lessened the opportunity for the accumulation of gas bubbles, and raising of the threshold of sensitivity since the subject operated under less continued stress which reduces the threshold to subsequent added stresses;

f. Mobility was moderately improved which was due partially to the slightly looser torso fitting allowed by the bladder system and to tailoring improvements made by the cooperative efforts of the contractors and the contract monitors;

g. Time duration at altitude was greatly improved.

3. Capabilities

Volunteers have been at altitudes as high as 198,770 feet which is almost a perfect vacuum with only one three-thousandths of an atmosphere. Selected subjects of extremely poor time-altitude capability in the MC-1 ensemble have remained at about 100,000 feet for several hours. With only one exception to this latter group, descent from altitude was not prompted because of physical necessity.

4. Limitations

The limits of this ensemble are discussed in WADC TR 57-536, Part II, which includes the time-altitude capabilities of the MC-1 as well as the MC-3 suit ensembles.

A few subjects in the MC-3 ensembles developed mild EKG changes, such as can be produced in the susceptible at 10,000 to 12,000 feet on free air. There were also some subjects who had the EKG changes attributed to anxiety. Although the time duration has been greatly increased, the responsible physician should, nevertheless, carefully screen the personnel with particular attention given to the cardiovascular status. The routine Master's EKG is recommended as an integral part of all pre-run physicals.

A particular disadvantage of this ensemble is the potential temperature problem arising from the impermeability of the bladder coverage. While 60 percent of the body bulk is covered, about 60 percent of the skin area, the potential sweating and heat loss area, remains outside of the bladder section and the axillae are open to the porous material. Studies indicate that the average subject under high heat and humidity loads suffers a significant time-decrement in performance and is ultimately forced to terminate his subjection to these loads. Since it is not likely that temperatures over 100° F would be experienced while in flight with current aircraft, the heat problem would arise only with ground conditions of high ambient temperature and during waiting periods, such as briefings, preflight checks, and alert standbys. The situation can be improved for the present by wearing the suit unzipped while prebreathing (denitrogenation), by providing

air-conditioned ready rooms or trucks, by cooling the aircraft with truck-borne air conditioners during long preflight checks, or by having someone other than the pilot perform the preflight operation.

E. Drinking Fluids While Pressurized

Because of the fluid balance problem, it seems desirable to allow a man to drink while at altitude in the partial pressure suit. Further, it is dangerous in the partial pressure situation to permit dryness of throat to develop to an extent which may precipitate paroxysm of coughing, as occasionally happens on long-term runs at high altitude. A coughing "fit," especially under the conditions imposed by partial pressure suits, can initiate a reflex chain that can cause fainting (refs. 22, 23, 122).

A pressurized "port" through the facepiece of the helmet and implement for opening cans and sucking their contents, termed the "canmaster," provided the means of drinking. Initially, the "Canmaster" proved impractical at altitudes above 40,000 feet, for, as the helmet pressure increased, the pressure was transmitted from the helmet to the can which forced the contents of the can to escape outside via the air vent in the "Canmaster." However, by automatically backloading the "Canmaster" air vent from the helmet when the drinking tube entered the helmet, the subject could drink with relative ease at 80,000 feet.

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APPENDIX I

PHYSICAL PROFILE

HISTORY AND PHYSICAL

MEN TO BE SUBJECTED TO SIMULATED HIGH ALTITUDES

NAME:	SERIAL NUMBER:
RANK:	DATE:

I. GENERAL HISTORY

1. Occupation (Duties):

2. Theatres In Which Subject Has Served:

3. Flight Personnel: ( ) Yes ( ) No

4. Has Subject Been Above 30M Unpressurized For Significant Length of Time ( ) Yes ( ) No

Past Experiences at Altitude: ( ) Neg ( ) See Below
- sinus
- ears
- chest pain
- abdominal discomfort
- joint pains
- paresthesias

5. Past History: ( ) Neg ( ) See Below
- medical
- surgical
- injuries

6. Diseases of Specific Interest: ( ) Neg ( ) See Below
- rheumatic fever
- rubeola (measles)
- hepatitis
- scarlet fever
- strep throat
- viral pneumonia
- recurrent pneumonia
- influenza
- tuberculosis
- asthma
- spontaneous pneumothorax
- sinus difficulties
- venereal disease
- epilepsy
- encephalitis
- septicemia
- subacute bacterial endocarditis
- diabetes

PHYSICAL PROFILE (CONT'D)

7. Family History (as far back as grandparents): ( ) Neg ( ) See Below
   - high blood pressure
   - stroke
   - heart disease
   - rheumatic fever
   - tuberculosis
   - other pulmonary disease
   - neurological or mental disorder
   - kidney disorder
   - epilepsy
   - diabetes

II. SYSTEMIC REVIEW

1. General: ( ) Neg ( ) See Below
   - syncope
   - unexplained fevers
   - night sweats
   - jaundice
   - tremor
   - weight gain or loss
   - diplopia, scotomata, photophobia
   - allergies

2. Musculo-Skeletal: ( ) Neg ( ) See Below
   - muscle weakness or pain
   - joint pain
   - varicosities
   - skin ulcers

3. Nervous: ( ) Neg ( ) See Below
   - headaches
   - convulsions
   - paresthesia
   - paralysis
   - emotional outlook toward test

4. Cardio-Respiratory: ( ) Neg ( ) See Below
   - chest pain
   - cough
   - sputum
   - hemoptysis
   - dyspnea
   - fatigue
   - palpitation
   - tachycardia
   - vertigo
   - edema
   - orthopnea
   - asthma
   - fever and/or chills
   - elevated blood pressure
   - cyanosis
   - pleurisy

PHYSICAL PROFILE (CONT'D)

5. Gastro-Intestinal: ( ) Neg ( ) See Below
   - appetite
   - abdominal pain or distress
   - discomfort relieved or aggravated by food
   - colic
   - nausea
   - vomiting
   - hematemesis or melena
   - belching or flatulence
   - constipation or diarrhea
   - hernia
   - hemorrhoids
   - stool abnormality

6. Genito-Urinary: ( ) Neg ( ) See Below
   - discharge
   - sores
   - frequency
   - nocturia
   - hematuria
   - stones
   - dysuria
   - urgency
   - retention
   - abnormal color

III. PHYSICAL EXAM

1. Chest X-Ray, ECG (Master's), CBC, UA: ( ) Neg ( ) See Note

2. General:
   - age
   - sex
   - nutrition
   - height
   - weight
   - temperature
   - pulse
   - respirations
   - blood pressure
   - deformity
   - physical type
     - ( ) endomorph
     - ( ) mesomorph
     - ( ) ectomorph
   - race

3. Head: ( ) Neg ( ) See Below
   - cranium and scalp
   - ears
   - eyes, incl. fundiscopic exam
   - nose
   - mouth, incl. teeth and pharynx

PHYSICAL PROFILE (CONT'D)		
4. Neck: bruits thrills sinuses scars masses pulsations distended veins carotid sinus reflex	( ) Neg	( ) See Below
5. Thorax:	( ) Neg	( ) See Note
6. Lungs: palpitation percussion auscultation	( ) Neg	( ) See Below
7. Heart: size murmurs thrills rhythm	( ) Neg	( ) See Below
8. Abdomen: scars tenderness herniae masses liver, kidney, spleen flank tenderness	( ) Neg	( ) See Below
9. Back: scoliosis kyphosis tenderness	( ) Neg	( ) See Below
10. Extremities:	( ) Neg	( ) See Note
11. Blood Vessels:	( ) Neg	( ) See Note
12. Skin:	( ) Neg	( ) See Note
13. Nervous System: pupils ocular movements visual fields and acuity, gen'l hearing sensory & motor to face swallowing, gag reflex tongue movements neck movements arm and leg ataxia Achilles Babinski & Oppenheim	( ) Neg	( ) See Below

PHYSICAL PROFILE (CONT'D)

13. Nervous System: (continued)
ankle & patellar clonus
patellar
biceps
triceps
vibratory sense
Kernig or Brudzinski
abdominal
Romberg
gait

14. Lymphatic Glands: ( ) Neg ( ) See Note

15. Genital: ( ) Neg ( ) See Note

16. Rectal: ( ) Neg ( ) See Below
prostate and seminal vesicles
tenderness
masses
hemorrhoids or fissures
sphincter tone

17. Physical Fitness (Harvard Step Test):
platform 20 inches high
30 step-ups/min ---- as long as subject can, up to 5 min
pulse rates taken from 1 to 1 1/2 min, 2 to 2 1/2 min, 4 to 4 1/2 min
index of fitness = $\frac{\text{time of stepping in seconds x 100}}{\text{2 (sum of the 3 pulse counts)}}$

below 55	( ) poor physical condition
55-64	( ) low average
65-79	( ) high average
80-89	( ) good
above 90	( ) excellent

DATE OF PHYSICAL:
DATE OF TEST:

TEST DESCRIPTION:

MEDICAL OFFICER'S SIGNATURE