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# PHYSIOLOGIC OBSERVATIONS ON RACE CAR DRIVERS

*by Vincent P. Collins*

*Prepared by*
BAYLOR UNIVERSITY
Houston, Texas
*for*

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# PHYSIOLOGIC OBSERVATIONS ON RACE CAR DRIVERS*

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## INTRODUCTION

Performance under conditions of stress is the essence of athletic activities. These are commonly carried out by individuals equipped with certain physical advantages augmented by special training of varying degrees of intensity. Athletic contests provide a testing ground where performance is an index of ability and preparation.

Many daily activities and occupations--driving on city streets and highways, piloting commercial planes or manning spacecraft--involve performance where error could endanger lives of individuals or projects of great national importance. In these activities, too, natural ability and preparation are elements in performance that become increasingly important where public welfare and safety are involved.

Educational and training programs for a wide variety of occupations provide reasonable competence to cope with normal circumstances. Emergency situations involving physical danger are more difficult to study because simulated testing necessarily lacks the ingredient of genuine danger and its effect upon emotion and performance.

One sports activity which provides an opportunity for a study of individual performance under conditions of non-simulated stress is auto racing. This is the subject of this investigation.

The underlying concept of the study is that environmental stimuli produce physiological and biochemical responses within the individual which govern the effectiveness and efficiency of his reaction. There are therefore two stages of investigation: the first is to identify and classify the range of physiological and biochemical responses; the second is to relate these to performance. This study deals with the first stage. Observations were carried out at four auto races which will be reported individually. This was augmented, when possible, by control studies in the radiology department at Baylor University College of Medicine.

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## GENERAL DESCRIPTION OF METHODS EMPLOYED

### Histories

A complete history of each driver was obtained. A copy of the history format is contained in the report. Information pertaining to the drivers' vital statistics, medical history, occupation, hobbies, and experience in racing was obtained. Additional information as to the amount of rest received prior to driving, food intake, or any other information giving a better understanding of factors which may influence performance, was recorded.

### In-Race Recording

Continuous monitoring of the transthoracic and sternal lead ECG, oral temperature, and respiration were recorded during the race. Four drivers were monitored simultaneously, two by telemetry and two by in-car tape recorders.

A lap-by-lap description of the race, giving position of monitored drivers and performance, was recorded on a tape recorder. Additional spotters were deployed at strategic positions around the track to assist in following the progress of the drivers. As each driver passed these observation points, the information was relayed to the monitoring station and pit area by radio communication.

### Tilt Table Test

Performance, and even existence, depend upon a precise maintenance of cerebral blood supply which hence becomes the first function of the circulatory system. The efficiency of the intricate reflexes involved is a likely index of the stress to which physiological mechanisms are subjected in maintaining an even blood flow to the brain while meeting the requirements of violent physical exertion, emotional responses, energy utilization, and heat exchange.

The tilt table test provides a simple record of cardiovascular reflex response to the altered dynamics of circulation required by a sudden change in the hydrostatic pressures in tilting the body from horizontal to vertical and back to horizontal.

In the recumbent position, the heart must circulate blood in a system less than 12 inches in vertical height against a resistance in the peripheral vascular bed of extremities and splanchnic areas that must be in balance with the resistance in the cerebral circulation on the same level. On tilting to the erect position, the heart must now pump blood to the brain at a height of 5 to 6 feet. There is a column of blood of this height offering a hydrostatic pressure which the dependent peripheral

vascular bed must resist to avoid dilatation and pooling. The correction must provide unfaltering cerebral blood flow while meeting and coordinating all circulatory demands for other parts and functions of the body.

In a rigid closed system, the demand upon the circulatory pump would be simplified by unchanging volume and distribution. In the elastic closed system of the human circulatory system, complex interlocking reflexes are involved.

There are two mechanisms for maintaining cerebral circulation on tilting up to the erect position: (1) The vasomotor reflex which increases peripheral resistance in order to maintain or increase the diastolic pressure (2) The cardiac output which increases in order to maintain systolic pressure by an increase in pulse rate and/or an increase in stroke volume.

One might expect stress to be reflected in alterations of either or both of these mechanisms.

The tilt table test utilizes a basket stretcher that can be tilted up to 70° with a smooth and rapid motion. The only support is the contour of the stretcher and the foot-plate; restraining bands are avoided because of possible influence on circulation. During the test a continuous recording is made of blood pressure, ECG, cardiotach, and pneumogram. There are three phases: (1) a pre-tilt recording in the recumbent position, (2) a recording with the subject in orthostatic position (70°) and (3) a post-tilt recording in the recumbent position. A minimum of 5 minutes of recording in each position is obtained unless the individual shows syncope in the erect position.

Systolic pressure is the result of ejection of blood by the heart. The factors will be the rate of ejection, the amount ejected, and the residual pressure in the great vessels. Heart rate and duration of systole are readily determined. In this study stroke volume can only be inferred.

In the erect position, diastolic pressure would fall if blood were to drain away or pool in dependent vascular beds under the effect of increased hydrostatic pressure. This is opposed by a nicely timed vasomotor reflex to contract the dependent capillary bed and increase the peripheral resistance by a precise degree which must vary as the hydrostatic pressure at different vertical heights within the circulatory tree. It is also opposed by an increased cardiac output to meet the demand of an increased blood flow through the dependent vascular bed. Diastolic pressure changes then are assumed to reflect the vasomotor response, primarily but not entirely.

Pulse pressure is the difference between systolic and diastolic pressure. This is primarily a function of cardiac output. If vasomotor protection of diastolic pressure is good, then cardiac output is principally utilized to maintain systolic pressure, and pulse pressure may be wide.

If vasomotor protection of diastolic pressure is poor, then an increased cardiac output may maintain diastolic pressure, at least temporarily. However, this is at the expense of maintenance of systolic pressure and pulse pressure which tend to fall. A narrow pulse pressure will generally indicate a diminished blood flow to all areas and, if it is not corrected, syncope may follow.

## Blood Volumes

Iodinated human serum albumin tagged with $I^{131}$ has been employed for several years in the determination of blood volumes. The basic principle of measurement is the introduction of a known quantity of a radioisotope tracer and measuring its distribution and dilution according to formula:

$$V_2 = \frac{C_1 V_1}{C_2}$$

where $C_1$ = concentration of isotope/ml injected.
$V_1$ = number ml injected.
$C_2$ = concentration of dilutent/ml.
$V_2$ = volume of dilutent/ml.

Standard technique of blood volume determination has a small technical error of ±2 percent S.E. and is suitable for clinical use. Certain modifications used by this institution further minimize this error.

Laboratory studies to determine blood volumes during the passive tilt study indicate changes in the estimated total volume of blood, plasma volume, red cell mass, and the ratio of red cell mass to total volume (hematocrit). This can be interpreted as a decrease in effective volume during the tilt. These changes become more pronounced after exercise.

The drivers were placed in a supine position in a Stokes stretcher and the physiograph electrode applied. At this time, 10 ml (approx. 20 μc) iodinated human serum albumin is injected. A sample of blood is withdrawn at the end of 10 minutes (equilibrium takes 6 to 8 minutes). The driver is immediately raised to 70° and retained in this position for 8 minutes, at which time another sample is obtained. He is then returned to the supine position.

Previous studies have indicated that during tilt there is a certain amount of pooling of whole blood and/or plasma in the extremities. The impression is that the effective volume is decreased by pooling whole blood in the vascular space and the total volume decreases by the loss of plasma water to the tissue space.

Returning to horizontal position, the vascular pooled blood returns to circulation rapidly but there is a delay in the return of the tissue pooled water. The more plasma water pooled in the tissue space the slower the return to normal.

An attempt was made to measure the plasma water loss during tilt, and the relation to cardiovascular response.

Results of the study are presented in tables 6 and 7. Because of some doubt as to the volume measured by iodinated human serum albumin dilution technique, the results are presented as normalized volumes related to the isotope concentration in the 10-minute whole blood sample. As equilibrium is reached by 6 to 8 minutes, the 10-minute whole blood determination represents a volume of 100. Therefore, the basic dilution equation is now written:

$$V_{B,t} = \frac{C_{B,10}\ (100)}{C_{B,t}} \tag{1}$$

where $V_{B,t}$ = volume of whole blood at time t.
$C_{B,10}$ = counts in whole blood at 10 min.
$C_{B,t}$ = counts in whole blood at time t.

The plasma volume at 10 minutes is:

$$V_{p,10} = \frac{C_{B,10}\ (100)}{C_{p,10}} \tag{2}$$

where $V_{p,10}$ = plasma volume at 10 min.
$C_{p,10}$ = counts in plasma at 10 min.

The plasma volume at time t is:

$$V_{p,t} = \frac{C_{B,t}}{C_{p,t}} \times V_{B,t} \tag{3}$$

where $C_{p,t}$ = counts in plasma at time t.

The red cell mass at any time can be determined by subtracting the plasma volume from the whole blood volume at that time. The hematocrit is the ratio of the red cell mass at any given time to the whole blood volume at the same time.

In tables 6 and 7, the changes from the value at 10 minutes are given in the column headed $\Delta V$, or for the hematocrit $\Delta$.

The predominant change is toward a decreased circulating whole blood volume while in tilt. Eight of the twelve drivers tested responded in this manner. Two were excluded from the analysis because of development of syncope. The other two drivers responded by an increased circulating blood volume. Analysis indicates this is due to an increase in red cells with little or no change in plasma. The decrease in whole blood is almost exclusively due to loss in plasma. Only one individual responded with a significant decrease in red cells. This was Driver L, whose hematocrit (53.9 percent) indicated marked dehydration. He is also the driver who lost the most weight and recorded the highest body temperature while driving.

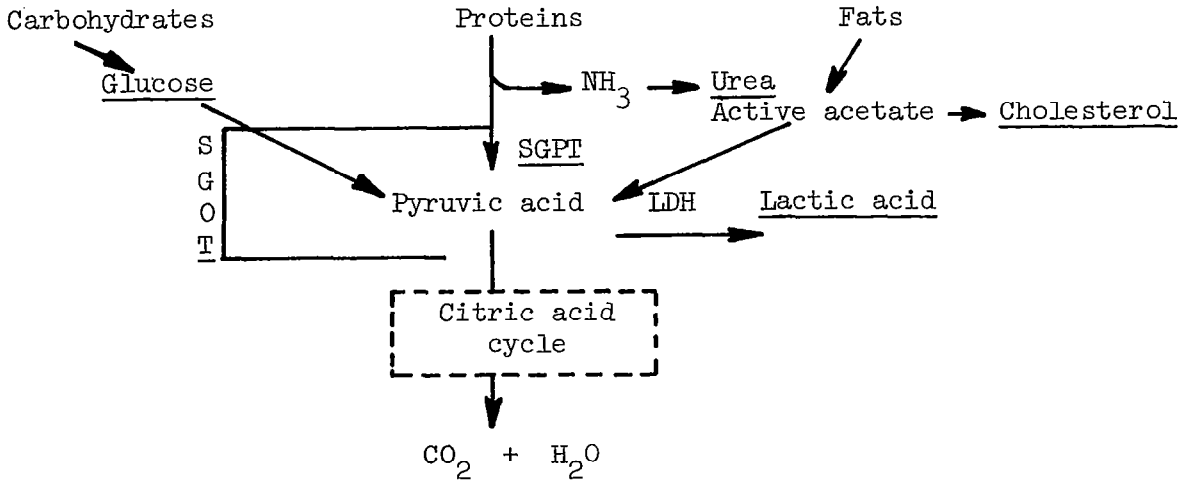
It is interesting to note that the change in whole blood volume is a manifestation of predominant changes in either plasma or red cells, but never both to the same degree. The direction of change in one is independent of the direction of change in the other. Regardless of which component of blood changed, the net result was an increase in hematocrit except in the one individual who showed indications of marked dehydration.

No relation between the increased whole blood volume and cardiovascular response in tilt could be drawn with this limited number of studies.

## Blood Chemistries

The notable changes in blood chemistries are in the values for glucose, $CO_2$ combining power, and lactic acid dehydrogenase. These and other metabolites and products recorded, fall within the following scheme for energy production from stores of carbohydrates, proteins, and fats.

During periods of high energy requirements, the metabolic activity associated with the conversion of stored metabolites to energy-yielding compounds and the relation to the biochemicals studied, can be summarized as:




(Lactic acid and $CO_2$ are indirectly measured by the $CO_2$ combining power.)

The following blood chemistries were obtained when possible: glucose, $CO_2$ combining power, cholesterol, SGOT, SGPT, LDH, BUN, uric acid, creatinine, and chlorides. These are normal constituents of the blood which may show changes in concentration during exercise, as each is involved in the various pathways of metabolic production of muscular energy. A discussion is given in the following paragraphs on each of the chemistries studied.

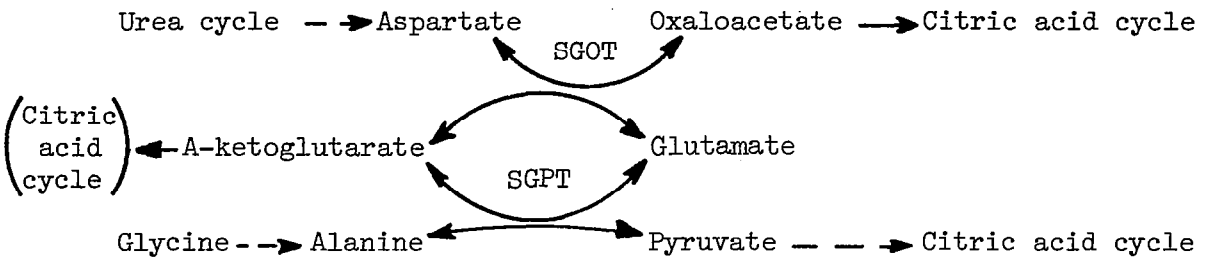
Glucose.- In short periods of exercise, the glucose concentration in the blood may reach 200 mg percent. This early rise in glucose may be a response to a release of adrenalin. Continuance of the exercise may use up the carbohydrate stores more rapidly than they are formed, resulting in a decrease in glucose concentration to subnormal levels. It has been suggested that lactic acid, formed during muscular activity, can stimulate the production of glucose by the liver from the metabolite stores. This may represent the stimulus for glucose production during prolonged exercise.

$CO_2$ combining power.- $CO_2$ combining power may be changed by the lactic acid liberated into the blood during muscular metabolism. In the blood, lactic acid is buffered by bicarbonate with the formation of carbonic acid. This bicarbonate, which is tied up with lactic acid, is removed from the role of a buffer for $CO_2$ in the blood. This loss of $CO_2$ buffering may result in a decreased transport of $CO_2$ from the tissue. Acidosis can ensue if this situation becomes critical. Measurement of the buffering capacity of the blood can be determined by the $CO_2$ combining power. In severe exercise, the amount of $CO_2$ which can combine with the serum should decrease.

Cholesterol.- Cholesterol, as mentioned, carbohydrates, fatty acids, and amino acids are utilized in the formation of glucose by the liver. Calorie-rich lipids, after having first been changed to the more active acetate in the liver, may be used to supply energy to areas of high metabolic activity, such as muscles during exercise. This active acetate is also an intermediate in the formation of cholesterol during periods of normal muscular activity. Cholesterol is not utilized in the formation of energy but is an alternate metabolic pathway for the high-energy compound active acetate. If the metabolic pathway for the active acetate is shunted toward meeting high energy requirements, then the rate of cholesterol synthesis should decrease.

Serum glutamic oxaloacetate transaminase (SGOT) and serum glutamic pyruvate transaminase (SGPT).- During tissue respiration certain protein and carbohydrate intermediates are rapidly converted to the keto-acid by transamination. Pyruvate, oxaloacetate, and alphaketoglutarate are

intermediates in the citric acid cycle. The interaction is illustrated in the following diagram.




These compounds, or the enzyme systems necessary for their metabolism, may change their concentration in the blood under the requirements of prolonged exercise. Changes in the glutamic-oxaloacetic and glutamic-pyruvate transaminase were followed during the driving stress.

Lactic dehydrogenase (LDH).- Lactic acid is formed by the muscular activity of contraction. The lactic acid thus formed is broken down to $CO_2$ and water in the presence of oxygen. Muscles do not require the immediate availability of oxygen to contract. Energy can be obtained from glycogen during the formation of lactic acid without oxygen. In the absence of oxygen, pyruvic acid is reduced to lactic acid and energy by the enzyme lactic dehydrogenase. During strenuous exercise, lactic acid may be formed more rapidly than the oxygen becomes available or the lactic acid can be converted back to the glycogen stores. This increase in lactic acid formation may result in an increased lactic dehydrogenase concentration in the blood.

Blood urea nitrogen (BUN).- Urea nitrogen proteins are not completely oxidized in the body. Partial oxidation occurs with about 40 percent of the available energy being excreted as urea. This energy does not become available because urea is not oxidized.

Ordinary exercise does not increase the excretion of nitrogen in the urine nor does it increase the concentration of non-protein nitrogen in the blood. As the work becomes strenuous and prolonged, an increased concentration of non-protein nitrogen is found in the blood, associated with an increased excretion in the urine. The major portion of the non-protein nitrogen of the blood is in the form of urea. Retention of urea should be further increased by the low urine flow on a hot day when water loss by perspiration is high.

Creatinine.- Creatine, or some complex body containing creatine, that is, creatine phosphate, is believed the precursor of the waste product creatinine which is the easiest of the nitrogenous waste products to be eliminated by the kidneys. Creatinine is excreted by the kidneys with no reabsorption; therefore, the plasma concentration is only a function of production and simultaneous filtration rate.

The reaction of phosphocreatine resulting in the transfer of phosphate to some acceptor does not yield creatinine directly. Therefore, the concentration of creatinine is not related to muscular acitivity but under

normal conditions it has been found to be more closely related to muscle mass. The concentration is possibly related to a constant synthesis of new creatine with degradation of that replaced to the waste product creatinine.

Since production of creatinine is not related to muscular activity, change in concentration with no change in muscle mass becomes a function of the filtration rate in the kidneys.

Any physiological event which causes a hindrance to the flow of urine may result in an increased creatinine concentration in the serum. Dehydration of the blood, caused by loss of body fluids, should be accompanied by an increase in blood creatinine. This is partly due to concentration per se, but primarily by impairment of renal circulation from a decrease in the effective renal plasma flow.

Phosphorus.- The physiological utilization of carbohydrates causes a decrease in serum phosphorus due to phosphorylation.

Urinary pH regularly decreases toward acid levels during and following exercise. This is a direct effort of the body to get rid of hydrogen ions, preserve its base as far as possible, and maintain the normal pH. An increased elimination of phosphate is one of the first events. Although the pH of urine changes only slightly during exercise, it decreases steeply to low levels within only a few minutes after completion of exercise.

Uric acid.- Uric acid is a weak dibasic acid which is an end product of purine metabolism. The sources of uric acid can be "endogenous," that derived from internal tissue metabolism, or "exogenous," which is due to ingestion of a purine diet.

Ingestion of large amounts of purine-yielding foods normally has very little effect upon the blood uric acid, as it is excreted rapidly, or up to 20 percent may be destroyed by the body rather than excreted. Of the nitrogenous compounds, uric acid is least readily excreted, in that any condition leading to suppression of urinary secretion would be reflected fast in an increased blood uric acid.

Therefore increases may be due to:

(1) Diminished excretion of uric acid.

(2) Increased production.

(3) Diminished destruction.

Violent exercise raises the concentration slightly, about 1 mg percent, probably due to diminished excretion. A decrease in the uric acid level of the blood is stated to almost never occur.

Chloride.- The chloride anions play an essential part in the buffering action of the plasma by means of the chloride shift. If the $CO_2$

tension is raised in the whole blood, the chloride of the plasma decreases, the chloride of the corpuscle increases, and the bicarbonate of the plasma increases. The chloride anions, which are released by the sodium during the formation of bicarbonate, migrate into the cells, causing a decrease in the chloride concentration of the plasma.

This chloride shift may be balanced or overcompensated for by a rapid decrease in sodium chloride excretion, which occurs during and after moderately severe and severe exercise. This decreased excretion returns to normal gradually after exercise, often requiring an hour or more. The excretion ratio of chlorides may decrease to 1/5 that of normal.

Loss of fluid and loss of salt generally accompany each other, also leading to a retention of chloride. If the loss of fluid is greater than the ability of the kidney to retain the chlorides, as in prolonged and profound sweating, weakness, fatigue, lack of appetite, nausea, a diminution of mental activity, and impairment of renal function can ensue.

## Renal Function

Many of the changes in plasma constituents center around kidney function either in part or entirety.

The effective renal plasma flow (ERPF) is normally decreased during and for about 1 hour after exercise. The degree and rate of decrease, as well as the duration, is possibly a function of the degree of exhaustion or taxation of the individual. If the exercise is exhausting, the ERPF may decrease to 1/3 the resting rate. Simultaneously, the glomerular filtration rate (GFR) decreases, but less than the ERPF. Consequently, the ratio of amount of plasma which is presented to the glomerular cells and that filtered per unit time rises.

The plasma concentration of a substance which is filtered and excreted becomes a function of rate of production, plasma flow, and glomerular filtration rate. For substances which may be reabsorbed, as in the case of glucose, reabsorption rate is another function to consider.

The maximum output of any material by the kidney can be defined as the point where the amount of material filtered minus the amount appearing in the urine per minute does not change with increasing concentration. A substance which is filtered and excreted with no reabsorption (such as creatinine) has a maximum output equal to the maximum filtration rate. The output at any time would be equal to the plasma concentration times the simultaneous filtration rate. The maximum output for other substances, such as glucose, appear to be more closely governed by the maximum reabsorption rate.

Vasomotor disturbances accompanying excitement, digestion, and muscular activity may have marked effects on urine flow by the rate at which plasma is presented to the cell for filtration. Post-exercise urine flow returns to approximately normal about 8 minutes after stopping exercise.

There is contradictory evidence pointing toward the roles ERPF, GFR, and reabsorption have on the effectiveness of urinary output under stress. The major adjustment of renal function must be primarily by the effective renal plasma flow and tubular reabsorption.

Urine chemistries obtained included glucose, acetone, sodium, potassium, and osmolality when possible.

Glucose (urine).- For glucose, there appears to be a reabsorption maximum. At high plasma concentrations, the rate of reabsorption becomes fixed. Under normal filtration rates (100 to 125 ml/min), the threshold concentration in the blood is approximately 160 mg percent. Above this, the excess is "spilled" into the urine for elimination. The maximum reabsorption for glucose under these conditions appears to be about 160 to 200 mg/min. Individual variations may occur.

The filtration rate may decrease significantly under conditions of exhausting exercise. Assuming a decrease to 60 ml/min and no change in rate of reabsorption, the blood concentration of glucose would have to approach 270 mg percent before any sugars would be spilled into the urine.

Potassium (urine).- In mild exercise, potassium excretion either does not change or decreases slightly with rapid recovery afterward. A reasonable explanation of a decrease may be a diminished supply of sodium ions to the potassium-secreting segment of the nephrons. Potassium is only partially reabsorbed by the tubules; the rest is excreted. Increased urinary output may be related to impaired reabsorption of potassium due to a preference for the sodium ion. Additional increase may be due to the production of highly concentrated urine.

Sodium (urine).- A decrease in glomerular filtration rate is a sufficient cause for a decrease in sodium chloride excretion. This, along with an apparent increase in sodium reabsorption in an attempt to conserve the bases of the blood, would make any decrease more apparent. The excretion of sodium after exercise follows very closely that of chloride excretion previously mentioned in the discussion of the blood chemistries.

Osmolality (urine).- The measure of the osmolar concentration of a solute in a fluid is termed osmolality. The capacity of solutes to reduce the molar concentration of water is dependent on number, not kind, of solute particles. In effect, the solute particles displace or dilute the water molecules, reducing the escaping tendency of the water from solution. The capacity of solutes to influence this colligative property is expressed in osmols or milliosmols.

One mol of a substance that does not ionize or dissociate when dissolved in water is defined as an osmol. One osmol of an undissociated solute is therefore equal to its molecular weight. Where dissociation enters the picture, the osmol is something less than the molecular weight, depending on the number of particles into which the molecule is dissociated at the molar concentration.

The molar concentration of particles in solution can be calculated from the freezing point of solution using the equation:

$$t = K \times M$$

where t = freezing point of solution.

K = cryoscopic constant of water.

M = dissolved particle concentration.

The dissolved particle concentration is expressed as mols per kilogram of water (osmols). The normal range for plasma is 300 to 500 millimols per kg of water while urine may vary from 500 to 1350. Concentrated urine may reach values as high as 2700 millimols.

The higher the osmolality the more concentrated the solutes, which may be interpreted as meaning the more the body is conserving $H_2O$, or the more solutes being excreted.

## Vital Signs and Weights

Heart rate, blood pressure, respiration rate, temperature, and weight were taken, as feasible, before and after control studies and stress events, and, when possible, during the stress event. Manually counted radial pulse rate is of limited value since the change in pulse rate during the customary 15-second counting period may be great. Cardiotachometry is of much greater value, but of course requires application of electrodes and a paper or tape recorder. This is part of the tilt table test and of in-race recordings. Isolated blood pressure readings are also of limited value because of change and human error. The same is true for respiration rate manually or visually counted and the impedance pneumogram has been of little value during violent physical motions. Oral temperature readings are dependable if carefully taken; but, under conditions of stress, subjects may breathe with open mouth, drink cold fluids, or succumb to nausea and vomiting. The oral thermistor is satisfactory for constant recording with a trained subject, but some subjects lose the device or chew it destructively under excitement of the race.

These signs, therefore, are of limited value under field research conditions.

Weights were all recorded as stripped weight before and after active competition, with the minimum possible delay.

TABLE 1.- VOLUNTEER DRIVERS, DRIVING TIME, AND STUDIES[a] PERFORMED AT DAYTONA INTERNATIONAL SPEEDWAY

| Subject | Elapsed time |
|---|---|
| Challenge Cup Race | |
| Driver A | 2 hr 03 min |
| Driver B | 2 hr 15 min |
| Driver C | 2 hr 30 min |
| Driver D | 2 hr 30 min |
| Daytona Continental Race | |
| Driver E | 3 hr 51 min |
| Driver F | 10 hr 30 min |
| Driver G | 8 hr 25 min |
| Driver H | 8 hr 25 min |
| Driver I | 13 hr 00 min |
| Driver J | 10 hr 45 min |
| Driver K | 9 hr 45 min |
| Driver L | 9 hr 45 min |

[a]Studies performed:

Glucose, BUN, $CO_2$ combining power, cholesterol, SGOT, SGPT, LDH.
Steroid excretion in urine.
Blood volumes.
Passive tilt.
Body temperature, blood pressure, pulse rate, weight loss.
Telemetry on in-car recording of ECG, respiration, temperature.

TABLE 2.- VITAL STATISTICS OF SUBJECTS IN RACE AT DAYTONA

| Driver | Physical description | | | Occupation | Marital status and no. of children | Education | Racing experience | | Rest prior to race |
|---|---|---|---|---|---|---|---|---|---|
| | Weight | Height | Age | | | | Type | No. of years | |
| Driver A | 148 | 6 ft | 27 | Cattle rancher | No | 5 yr college | Sports | 7 | |
| Driver B | 215 | 6 ft 2 in. | 44 | Car dealer | Yes 2 children | | Sports | 8 | |
| Driver C | 166 | 6 ft | 37 | Lumberman | Yes 3 children | None after high school | Sports | 8 | 1 1/2 hr rest |
| Driver D | 194 | 6 ft | 34 | Radio announcer | No | Training | Sports<br>Drag | 3<br>14 | |
| Driver E | 166 | 5 ft 10 1/2 in. | 36 | Ship repair industrialist | No | Industrial training and economics | Sports | 14 | |
| Driver F | 175 | 6 ft 2 in. | 35 | Race driver | Yes 1 child | Degree in metal-lurgy and indus. design | Sports | 12 | |
| Driver G | 180 | 5 ft 10 1/2 in. | 38 | Auto salesman | Yes 4 children | | Sports | 8 | 7 hr sleep |
| Driver H | 230 | 6 ft 3 in. | 27 | Real estate broker | No | | Sports | 9 | 9 hr sleep |
| Driver I | 187 | 5 ft 9 in. | 36 | Industrial caterer | Yes 2 children | | Sports | 6 | 7 hr sleep |
| Driver J | 190 | 6 ft 2 in. | 32 | Race driver | Yes 4 children | 3 yr college | Sports<br>Stocks, Grand Prix, Ind. | 8<br>5 | 6 hr sleep |
| Driver K | 140 | 5 ft 10 1/2 in. | 26 | Race driver | Yes 2 children | | Sports | 4 | 8 hr sleep |
| Driver L | 145 | 5 ft 9 in. | 41 | Auto dealer | Yes 2 children | | Sports | 11 | 7 hr sleep |

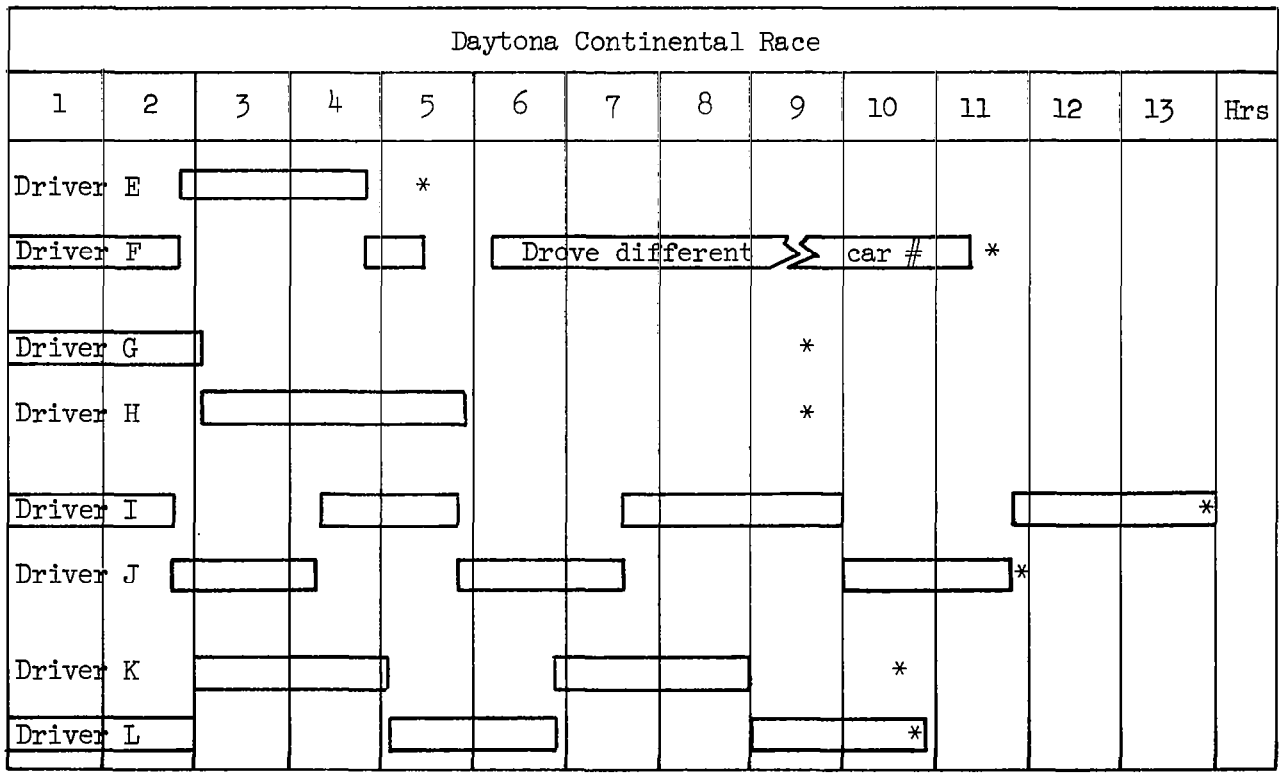



Figure 1.- Driving periods, Daytona International Speedway.

Pre- and post-race temperature, pulse, and blood pressure (tables 3 and 4).- These factors are expected to vary in response to environmental conditions, physical exertion, and emotional stress.

The values in these tables are obtained before and after each race and do not reveal in-race changes which were recorded by telemetry and in-car recorders. The ambient temperature during the first race on February 14, 1964, was 75°; during the second race on February 15, 1964, the ambient temperature varied from 65° to 78°. The temperature, pulse, and blood pressure of drivers have been of interest chiefly in mid-summer events when ambient temperatures of over 105°, cockpit temperatures of over 130°, closed cars, and flame-proof driving suits may contribute to stress. In the Daytona events, climatic conditions were ideal. No impressive changes occurred, but this event offers a baseline for comparison with later events under more severe conditions.

In Race 1, of 2 1/2 hours duration, the variations for all four drivers in temperature, pulse, and blood pressure are within limits for common occupational limits.

In Race 2, of 13 hours duration, with two drivers to relieve each other, Driver L showed a temperature rise from 98.4° to 103.4° F. This was in a closed car with an outside temperature of 75°. There was no associated malaise or evident impairment of performance. It raises a question for future consideration as to a possible variation in efficiency of body cooling as between individuals. The co-driver (Driver K) had a temperature rise from 98.8° to 101° F. One other driver (F), in an open car, showed a temperature rise from 97.8° to 101.0° F.

Blood pressures and pulse rates, pre- and post-race, are not informative for the participants in these events. Monitoring of blood pressure and pulse rate during tilt table testing is discussed separately. Monitoring of pulse and temperature during the race are on records in the possession of NASA personnel.

TABLE 3.- PRE- AND POST-RACE TEMPERATURE, PULSE, AND BLOOD PRESSURE READINGS OF DRIVERS IN THE AMERICAN CHALLENGE CUP RACE AT DAYTONA INTERNATIONAL SPEEDWAY

| Driver | Time | | Temperature, °F | | Blood pressure | | Pulse rate | |
|---|---|---|---|---|---|---|---|---|
| | Pre-race | Post-race | Pre-race | Post-race | Pre-race | Post-race | Pre-race | Post-race |
| Driver A | 11:30 | 3:40 | 99.8 | 100.2 | 108/70 | 120/70 | 112 | 108 |
| Driver B | 11:40 | 3:55 | 100.4 | 100.0 | 140/90 | 115/86 | 112 | 107 |
| Driver C | 11:20 | 4:20 | 99.7 | 100.0 | 110/80 | 115/85 | 112 | 112 |
| Driver D | 10:10 | 4:20 | 98.2 | - | 110/70 | 100/80 | 88 | 112 |
| | | | | | | [a]88/70 | | [a]102 |

[a]Blood pressure and pulse rate taken after development of syncope.

TABLE 4.- PRE- AND POST-RACE TEMPERATURE, PULSE, AND BLOOD PRESSURE READINGS OF DRIVERS IN THE DAYTONA CONTINENTAL RACE AT DAYTONA INTERNATIONAL SPEEDWAY

| Driver | Place (a) | Time | Temperature, °F | Blood pressure | Pulse rate | Weight |
|---|---|---|---|---|---|---|
| Driver E | H | 9:20 | 99.2 | 118/58 | 72 | 166 |
| | P | 1:50 | 100.5 | | | |
| | H | 2:35 | | | | 164 |
| | H | 3:15 | | 118/68 | 72 | |
| Driver F | H | 8:45 | 97.8 | 112/80 | 60 | 175 |
| | P | 11:50 | 100.2 | | | |
| | P | 2:30 | 101.0 | | | |
| | H | 2:35 | | 122/68 | 92 | 172.5 |
| Changed car | H | 8:30 | 98.0 | | | 172 |
| Driver G | H | 8:45 | 98.6 | 140/70 | 96 | 180 |
| | P | 12:16 | 99.2 | | | |
| | H | 6:35 | 99.2 | 110/72 | 84 | 179 |
| Driver H | H | 8:20 | 98.2 | 110/80 | 84 | 229.5 |
| | P | 2:50 | 98.0 | | | |
| | H | 6:40 | 98.4 | 118/60 | 96 | 228 |
| Driver I | H | 8:35 | 99.4 | 130/90 | 112 | 188 |
| | P | 11:40 | 100.2 | | | |
| | P | 2:54 | 99.6 | | | |
| | H | 11:00 | 99.2 | 113/66 | 82 | 187 |
| Driver J | H | 10:30 | 98.0 | 120/70 | 60 | 187.5 |
| | P | 1:24 | 98.5 | | | |
| | P | 4:38 | 98.0 | | | |
| | H | 8:55 | 97.8 | 130/85 | 88 | 184.5 |
| Driver K | H | 10:20 | 98.8 | 120/80 | 80 | 136.5 |
| | P | 2:15 | 101.0 | | | |
| | H | 7:45 | 99.1 | 110/85 | 100 | 134.5 |
| Driver L | H | 9:00 | 98.4 | 140/76 | 80 | 147 |
| | P | 12:00 | 101.7 | | | |
| | P | 3:50 | 103.4 | | | |
| | H | 7:45 | 97.8 | 135/70 | 100 | 141 |

[a] H - Hospital (testing area).

P - Pit area.

Weight-change studies conducted on drivers at Daytona International Speedway (table 5).- Table 5 indicates a maximum weight loss of 6 lb or 4.1 percent of initial body weight by one driver (L) in a closed car during Race 2. Two drivers in open cars during Race 1 lost 4.5 lb or 2.1 percent (B) and 2.3 percent (D). Remaining drivers lost 1 to 3 lb but, since fluid intake was not controlled, the values are not particularly informative. However, it does suggest that, under more stressful conditions, more impressive values might be encountered with commensurate changes in blood chemistries.

Blood volume and hematocrit (tables 6 and 7).- When blood volume and hematocrit are recorded as single numerical volumes, some reliance is merited for purposes of blood replacement in bed patients. However, the dynamics of circulation and fluid exchange across capillary membranes under conditions of physical activity and stress, transform blood volume and hematocrit into less tangible concepts. The variables that determine volume of the closed circulation at any time are contraction or expansion of capillary bed, the possibility of reservoirs not constantly in equilibrium with the general circulation, dilatation and pooling in a dependent capillary bed, movement of fluid in either direction across capillary walls, red cell reservoirs, and red cell sedimentation.

TABLE 5.- WEIGHT-CHANGE STUDIES CONDUCTED ON DRIVERS AT DAYTONA INTERNATIONAL SPEEDWAY

| Driver | Driving time, hr | Control weight, lb | Initial weight, lb | Final weight, lb | Weight change, lb |
|---|---|---|---|---|---|
| American Challenge Cup Race | | | | | |
| Driver A | 2 | 148 | 144 | 143 | -1 |
| Driver B | 2.3 | 215 | 216.5 | 212 | -4.5 |
| Driver C | 2.5 | 166 | 166 | 165 | -1 |
| Driver D | 2.5 | 194 | 197 | 192.5 | -4.5 |
| Daytona Continental Race | | | | | |
| Driver E | 2 | - | 166 | 164 | -2 |
| Driver F | 5 | - | 175 | 172 | -3 |
| Driver G | 2 | - | 180 | 179 | -1 |
| Driver H | 3 | - | 229.5 | 228 | -1.5 |
| Driver I | 7 | - | 188 | 187 | -1 |
| Driver J | 6 | - | 187.5 | 184.5 | -3 |
| Driver K | 4 | - | 136.5 | 134.5 | -2 |
| Driver L | 6 | - | 147 | 141 | -6 |

TABLE 6.- BLOOD-VOLUME STUDIES CONDUCTED ON DRIVERS IN THE AMERICAN CHALLENGE CUP RACE AT DAYTONA INTERNATIONAL SPEEDWAY

| Driver | Time | Blood volume | ΔV | Plasma volume | ΔV | Red cell mass | ΔV | Hct | Δ |
|---|---|---|---|---|---|---|---|---|---|
| Driver A | Control | | | | | | | | |
| | 10 min | 100 | | 53.8 | | 46.2 | | 46.2 | |
| | Tilt up | | | | | | | | |
| | 13 min | 99.3 | -0.7 | 54.5 | +0.7 | 44.8 | -1.4 | 45.1 | -1.1 |
| | 20 min | 96.9 | -3.1 | 51.0 | -2.8 | 45.9 | -0.3 | 47.4 | +1.2 |
| | Tilt down | | | | | | | | |
| | 23 min | 102.0 | +2.0 | 52.5 | -1.3 | 49.5 | +3.3 | 48.5 | +2.3 |
| | 31 min | 104.1 | +4.1 | 54.5 | +0.7 | 49.6 | +3.4 | 47.6 | +0.2 |
| | Post-race | | | | | | | | |
| | 10 min | 100 | | 53.9 | | 46.1 | | 46.1 | |
| | Tilt up | | | | | | | | |
| | 18 min | 97.4 | -2.6 | 51.5 | -2.4 | 45.9 | -0.2 | 47.1 | +1.0 |
| Driver B | Control | | | | | | | | |
| | 10 min | 100 | | 52.7 | | 47.3 | | 47.3 | |
| | Tilt up | | | | | | | | |
| | 20 min | 100.6 | +0.6 | 51.8 | -0.9 | 48.8 | +1.5 | 48.5 | +1.2 |
| | Tilt down | | | | | | | | |
| | 30 min | 102.1 | +2.1 | 54.8 | +2.1 | 47.3 | 0.0 | 46.3 | -1.0 |
| | Post-race | | | | | | | | |
| | 10 min | 100 | | 51.5 | | 48.5 | | 48.5 | |
| | Tilt up | | | | | | | | |
| | 18 min | 98.7 | -1.3 | 49.5 | -2.0 | 49.2 | +0.7 | 49.8 | +1.3 |
| Driver C | Post-race | | | | | | | | |
| | 10 min | 100 | | 54.4 | | 45.6 | | 45.6 | |
| | Tilt up (Syncope) | | | | | | | | |
| | Tilt down | | | | | | | | |
| | 13 min | 100.1 | +0.1 | 54.6 | +0.2 | 45.5 | -0.1 | 45.4 | -0.2 |
| Driver D | (Syncope- No results) | | | | | | | | |

TABLE 7.- BLOOD-VOLUME STUDIES CONDUCTED ON DRIVERS IN THE DAYTONA CONTINENTAL RACE AT DAYTONA INTERNATIONAL SPEEDWAY

| Driver | Time | Blood volume | ΔV | Plasma volume | ΔV | Red cell mass | ΔV | Hct | Δ |
|---|---|---|---|---|---|---|---|---|---|
| Driver E | Post-race 10 min | 100 | | 53.0 | | 47.0 | | 47.0 | |
| | Tilt up 18 min | 95.3 | -4.7 | 47.7 | -5.3 | 47.6 | -0.6 | 50.0 | +3.0 |
| Driver F | Post-race 10 min | 100 | | 55.9 | | 44.1 | | 44.1 | |
| | Tilt up 18 min | 96.3 | -3.7 | 52.4 | -3.5 | 43.9 | -0.2 | 45.6 | +1.5 |
| Driver G | Post-race 10 min | 100 | | 54.9 | | 45.1 | | 45.1 | |
| | Tilt up 18 min | 104.3 | +4.3 | 55.3 | -0.4 | 49.0 | +3.9 | 47.0 | +1.9 |
| Driver H | Post-race 10 min | 100 | | 51.6 | | 48.4 | | 48.4 | |
| | Tilt up 18 min | 96.5 | -3.5 | 48.1 | -3.5 | 48.4 | 0.0 | 50.1 | +1.7 |
| Driver I | Post-race 10 min | 100 | | 54.5 | | 45.5 | | 45.5 | |
| | Tilt up 18 min | 96.7 | -3.3 | 51.0 | -3.5 | 45.7 | +0.2 | 47.2 | +1.7 |
| Driver J | Post-race 10 min | 100 | | 52.0 | | 48.0 | | 48.0 | |
| | Tilt up 18 min | 97.4 | -2.6 | 50.3 | -1.7 | 47.1 | -0.9 | 48.3 | +0.3 |
| Driver K | Post-race 10 min | 100 | | 52.1 | | 47.9 | | 47.9 | |
| | Tilt up 18 min | 102.0 | +2.0 | 51.3 | -0.8 | 50.7 | +2.8 | 49.7 | +1.8 |
| Driver L | Post-race 10 min | 100 | | 46.1 | | 53.9 | | 53.9 | |
| | Tilt up 18 min | 96.9 | -3.1 | 46.2 | +0.1 | 50.7 | -3.2 | 52.3 | -1.6 |

TABLE 8.- BLOOD-CHEMISTRY STUDIES CONDUCTED ON DRIVERS AT DAYTONA INTERNATIONAL SPEEDWAY

| Driver | Time | Glucose | BUN | $CO_2$ combining power | Cholesterol | SGOT | SGPT | LDH |
|---|---|---|---|---|---|---|---|---|
| American Challenge Cup Race | | | | | | | | |
| Driver A | 2/3 | 76 | 15 | 25.5 | 189 | 6 | 4 | 400 |
| | Pre | 106 | 15 | 24.0 | 223 | 13 | 8 | 440 |
| | Post | 74 | 15 | 17.0 | 240 | 19 | 14 | 420 |
| Driver B | 2/4 | 98 | 13 | 25.0 | 215 | 4 | 6 | 450 |
| | Pre | 101 | 11 | 25.5 | 208 | 13 | 8 | 420 |
| | Post | 100 | 13 | 18.5 | 309 | 28 | 16 | 1410 |
| Driver C | Pre | 112 | 24 | 24.0 | 175 | 11 | 6 | 420 |
| | Post | 95 | 24 | 18.0 | 203 | 16 | 10 | 780 |
| Driver D | 2/2 | 87 | 13 | 24.0 | 223 | 10 | 8 | 920 |
| | Pre | 66 | 14 | 21.0 | 254 | 8 | 13 | 450 |
| | Post | 80 | 15 | 18.8 | 326 | 22 | 12 | 790 |
| Daytona Continental Race | | | | | | | | |
| Driver E | Pre | 122 | 18 | 24.0 | 240 | 14 | 10 | 500 |
| | Post | 116 | 17 | 23.0 | 215 | 11 | 12 | 420 |
| Driver F | Pre | 78 | 22 | 25.0 | 240 | 6 | 4 | 400 |
| | Post | 68 | 22 | 24.5 | 237 | 12 | 4 | 450 |
| Driver G | Pre | 103 | 17 | 23.5 | 200 | 6 | 7 | 440 |
| | Post | 58 | 15 | 16.0 | 200 | 15 | 9 | 640 |
| Driver H | Pre | 105 | 12 | 24.0 | 170 | 7 | 8 | 550 |
| | Post | 70 | 13 | 22.0 | 180 | 13 | 9 | 640 |
| Driver I | Pre | 70 | 14 | 25.5 | 249 | 19 | 23 | 510 |
| | Post | 62 | 17 | 18.0 | 212 | 22 | 10 | 680 |
| Driver J | Pre | 108 | 17 | 26.5 | 185 | 12 | 6 | 440 |
| | Post | 60 | 15 | 21.5 | 212 | 16 | 8 | 620 |
| Driver K | Pre | 114 | 12 | 25.5 | 187 | 12 | 8 | 450 |
| | Post | 50 | 17 | 21.0 | 185 | 15 | 8 | 640 |
| Driver L | Pre | 115 | 25 | 26.5 | 258 | 14 | 8 | 440 |
| | Post | 40 | 30 | 18.0 | 270 | 17 | 16 | 550 |
| Normal | | 60-95 mg percent | 5-20 mg percent | 24-30 meq/l | 180-300 units | 8-40 units | 5-35 units | 100-350 units |

Figure 3(a) and (b): Three drivers in the challenge cup race had elevated glucose in the control study while six of the eight drivers studied in the endurance race had elevated values in the control samples. This may be due to the samples not being drawn during a fasting period. Although some drivers had little change in glucose concentration post-race, the general trend was toward a decrease with the average being in the low normal value.

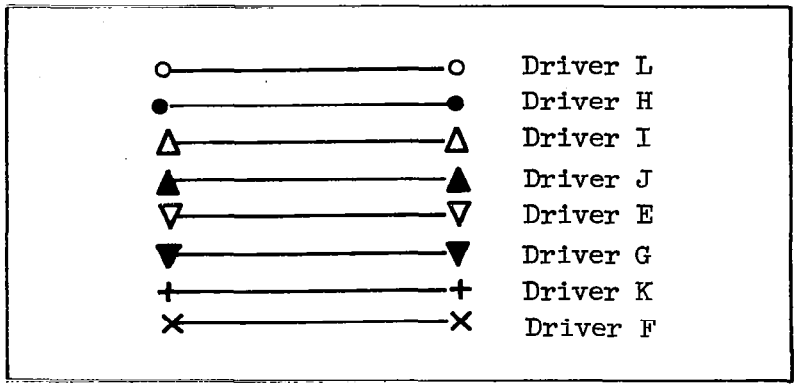
Figure 4(a) and (b): The $CO_2$ combining power began at low normal and during the race decreased about 25 percent with the average post-race value well below normal.

Figure 5(a) and (b): The LDH had the greatest change. Initial values were elevated in all individuals before the race and rose to a height of 1410 units post-race in one driver. Two drivers had a decrease in LDH after racing while others driving for approximately the same period of time had increases. During such short periods of driving, driving time may not be an adequate estimate of physical exertion. Other factors, such as handling of car, congestion of track and other factors, which may influence the amount of work performed, need be considered. The high LDH values and the low $CO_2$ combining power indicate a tendency toward subclinical acidosis. The degree seems to be greater in some than others. In general, the values would correspond to that seen in moderate acidosis. No correlation can be drawn to the amount of driving nor the interval between active driving and follow-up. There would be an expected return to normal values within 3 to 6 hours after racing. Comparison of the results obtained on the two drivers who had a delayed follow-up and those obtained within a short period, indicate recovery within the parameters studied may be more prolonged than expected.

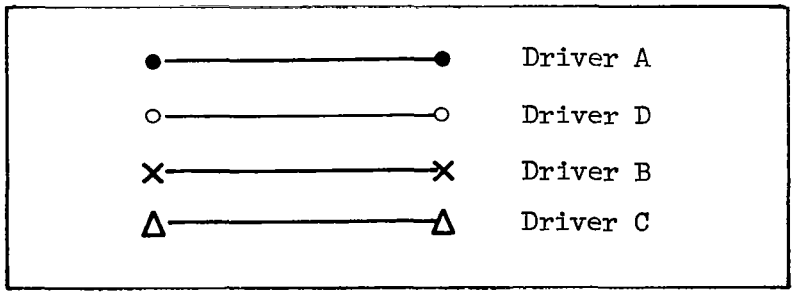
Figure 6(a) and (b): The urea nitrogen showed no significant changes except in one individual. In this case the urea nitrogen rose from 25 mg percent, which is slightly above normal, to 35 mg percent.

Figure 7(a) and (b): Cholesterol concentration does not seem to be a good indicator of fat metabolism. This is probably due to the large amount of cholesterol present in tissues which may be liberated into the circulation. No correlation seems to exist between driving time of any of the drivers and the direction of change in cholesterol concentration.

Figure 8(a), (b), (c), (d): SGOT and SGPT were within normal or slightly below normal in all drivers before the race. The general pattern was toward an increase but stayed within the low normal range.

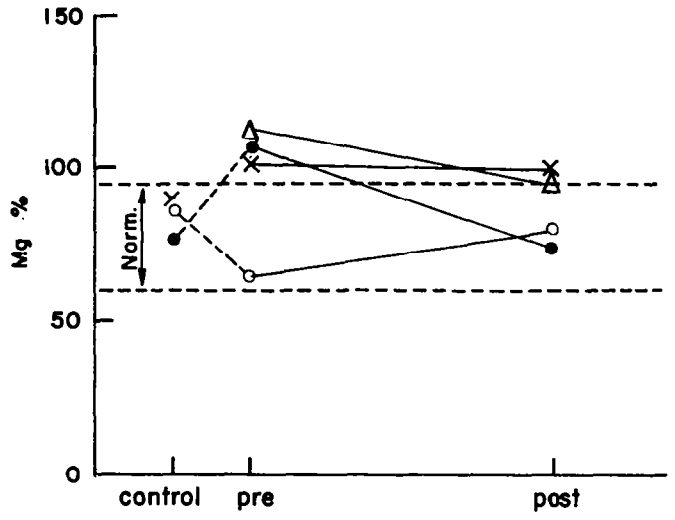



(a) Daytona Continental Race

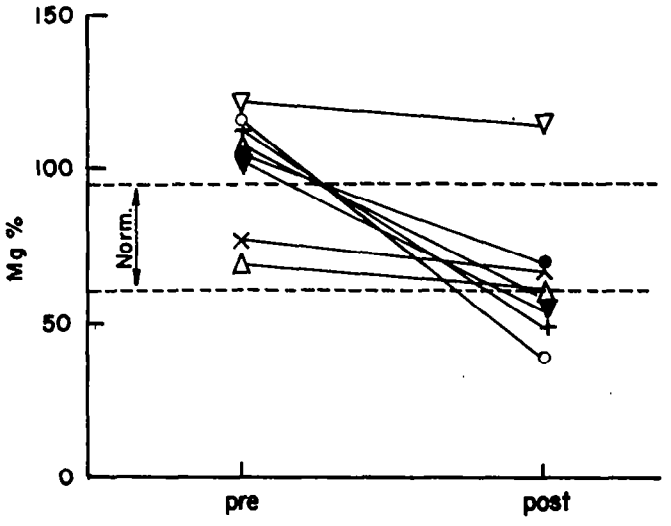



(b) American Challenge Cup Race

Figure 2.- Legends for blood chemistry figures.

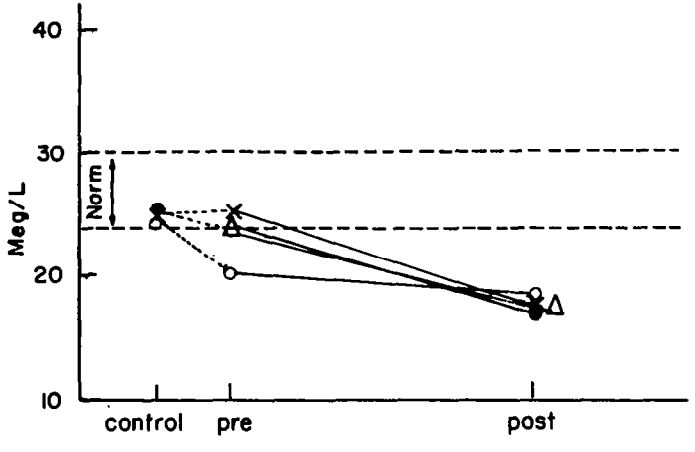



(a) American Challenge Cup Race.

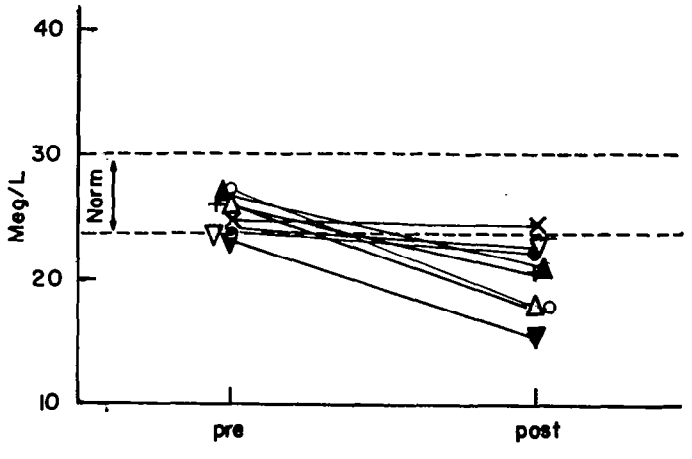



(b) Daytona Continental Race.

Figure 3. - Blood chemistry, glucose.

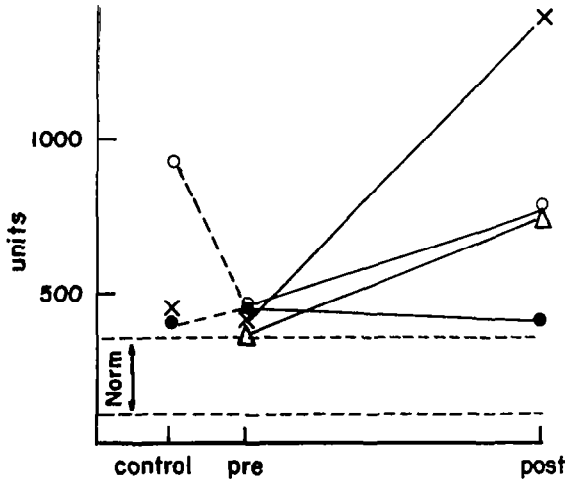



(a) American Challenge Cup Race.

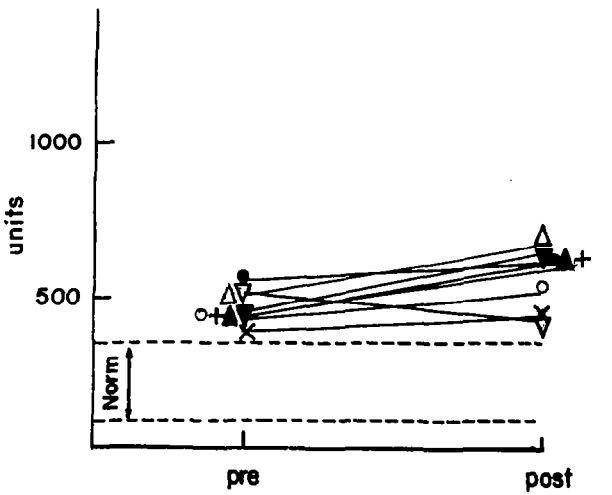



(b) Daytona Continental Race.

Figure 4. - Blood chemistry, $CO_2$ combining power.

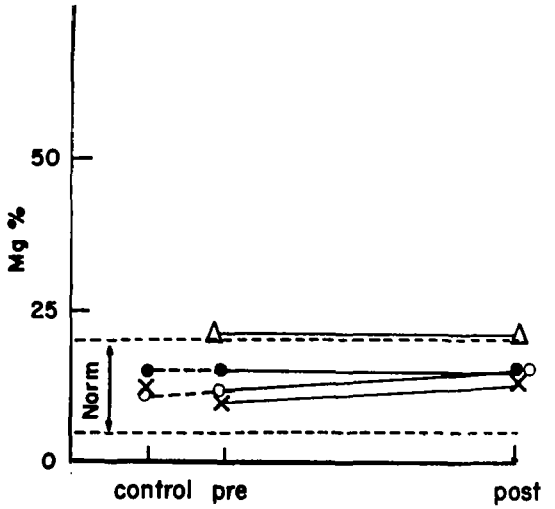



(a) American Challenge Cup Race.

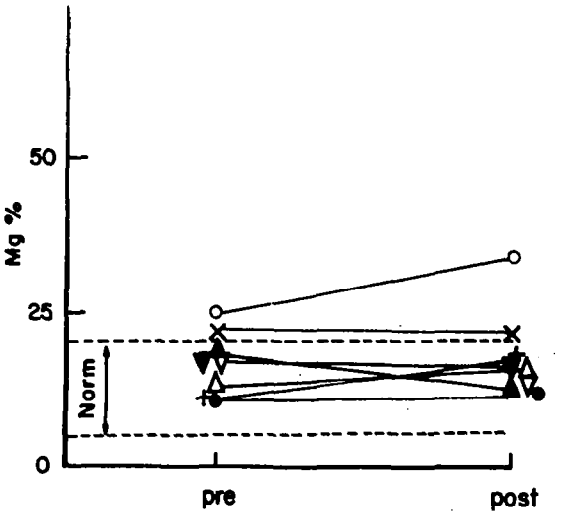



(b) Daytona Continental Race.

Figure 5. - Blood chemistry, lactic dehydrogenase.

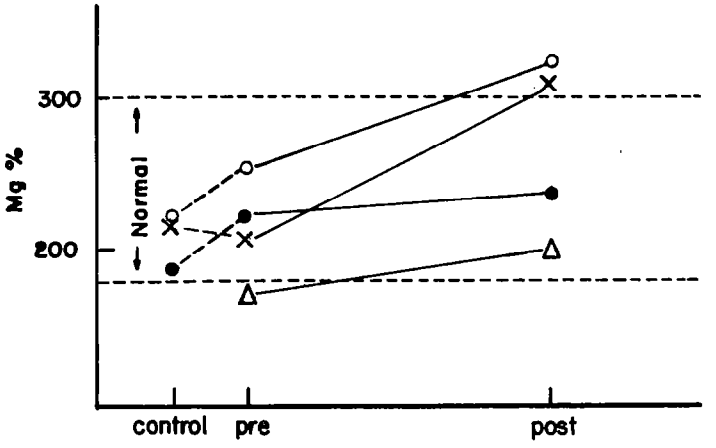



(a) American Challenge Cup Race.

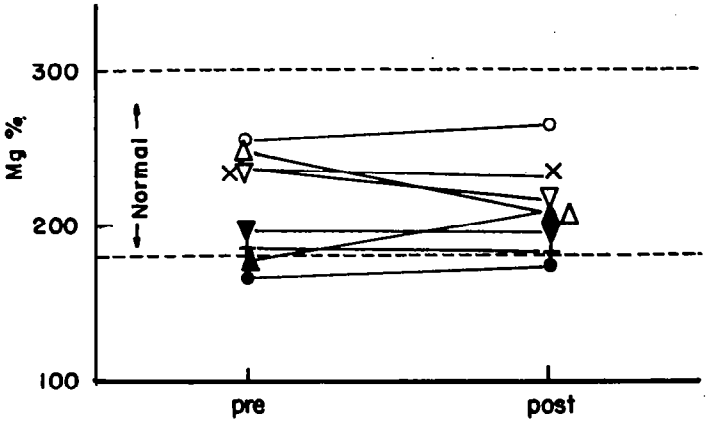



(b) Daytona Continental Race.

Figure 6. - Blood chemistry, blood urea nitrogen.

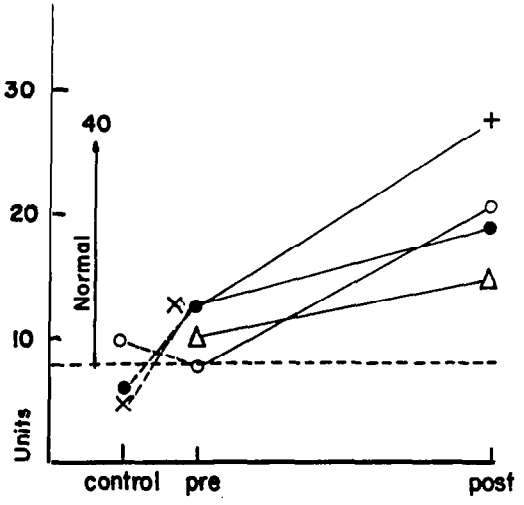



(a) American Challenge Cup Race.

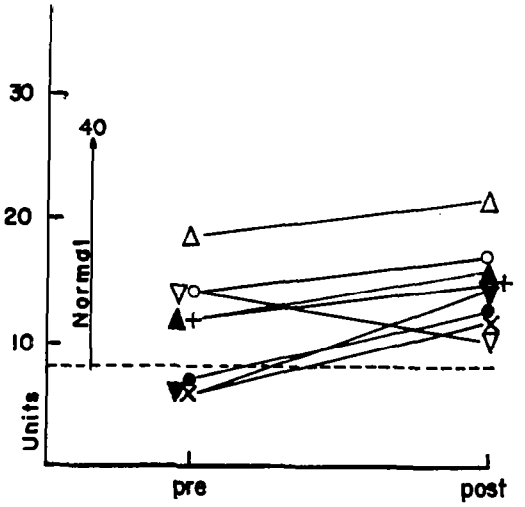



(b) Daytona Continental Race.

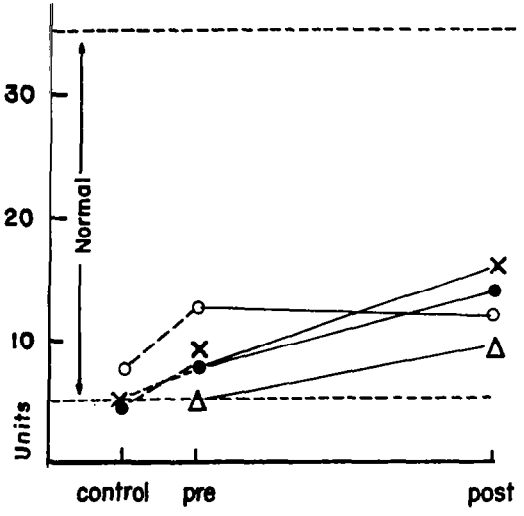
Figure 7. - Blood chemistry, cholesterol.



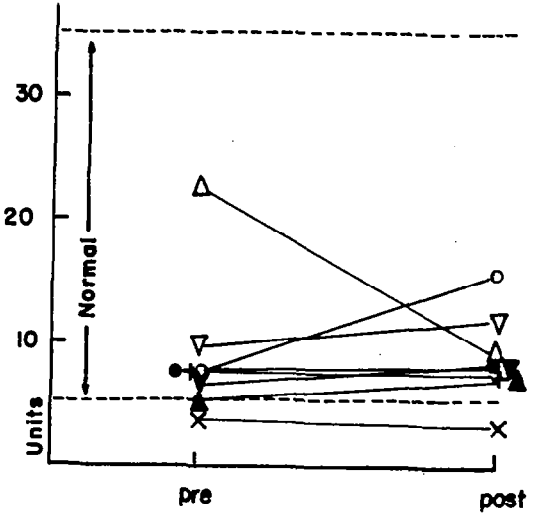

(a) SGOT, American Challenge Cup Race.




(b) SGOT, Daytona Continental Race.




(c) SGPT, American Challenge Cup Race.




(d) SGPT, Daytona Continental Race.

Figure 8. - Blood chemistries.

Comments on tilt table tests, Daytona race (figs. 9 through 22).- Two of the Daytona drivers have pre-race control studies and post-race studies suitable for direct comparison. A third driver showed syncope on tilting in his control and is unsuitable for comparison.

In the control studies both Drivers B and A have a cardiac output that is adequate to maintain an almost level systolic pressure on tilting up. However Driver B maintains systolic pressure without an appreciable increase in pulse rate and therefore must accomplish this by an increase in stroke volume. Driver A maintains systolic pressure by a rise in pulse rate from 115 to 160 and therefore is assumed to rely upon a lesser stroke volume with this increase in pulse rate.

Post-race, both drivers show an immediate rise in pulse rate which is maintained throughout the tilt at approximately 136 by Driver B and approximately 150 by Driver A. Driver B shows a momentary fall of systolic pressure and pulse pressure, with the recovery apparently by increasing stroke volume. Driver A shows a slow fall in systolic pressure and pulse pressure with a slow recovery, also apparently by increasing stroke volumes.

The remaining nine drivers were not available for control studies and only post-race tilt table studies were made. Diastolic pressure is maintained or rises in seven of the nine, indicating effect of vasomotor responses following the stress of driving. Two of the nine who showed a fall in diastolic pressure, indicating a failure of vasomotor reflexes, were under the greatest handicap. Driver C had worked on his race car all the preceding night and had 1 1/2 hours of sleep. He maintained diastolic pressure for approximately 1 minute when tilted up, then diastolic pressure and vasomotor control failed resulting in syncope. Driver I had driven 7 1/2 hours with a 3 1/2 hour drive in darkness just before testing. He maintained diastolic pressure for approximately 4 1/2 minutes in tilt and then lost diastolic pressure and vasomotor control. The tilt was ended to prevent syncope.

Both initially maintained systolic pressure and then showed a sudden loss in both systolic pressure and pulse pressure. Driver C had maintained his systolic pressure at 100 mm of mercury for approximately 1 minute, by rise in pulse rate from 100 to 150. The pulse rate of approximately 150 is maintained even after systolic pressure begins to fall, indicating a diminution in stroke volume.

Driver I maintains systolic pressure at approximately 100 mm of mercury by rise in pulse rate from approximately 75 to 112. A fall in pulse rate to 54 preceded the fall in systolic pressure with no corrective response by way of an increase in pulse rate to offset the fall in systolic, diastolic, and pulse pressures.

Three drivers (F, H, and G) show a notably stable pattern through the tilt table test with a rise in diastolic pressure indicating a strong vasomotor response, a moderate increase in systolic pressure by means of a moderate increase in pulse rate, and, by inference, an increase in stroke volume.

Four drivers (J, L, K, and E) have a comparable response pattern to tilt except for a more marked increase in pulse rate to maintain the systolic pressure and, inferentially, a smaller stroke volume.

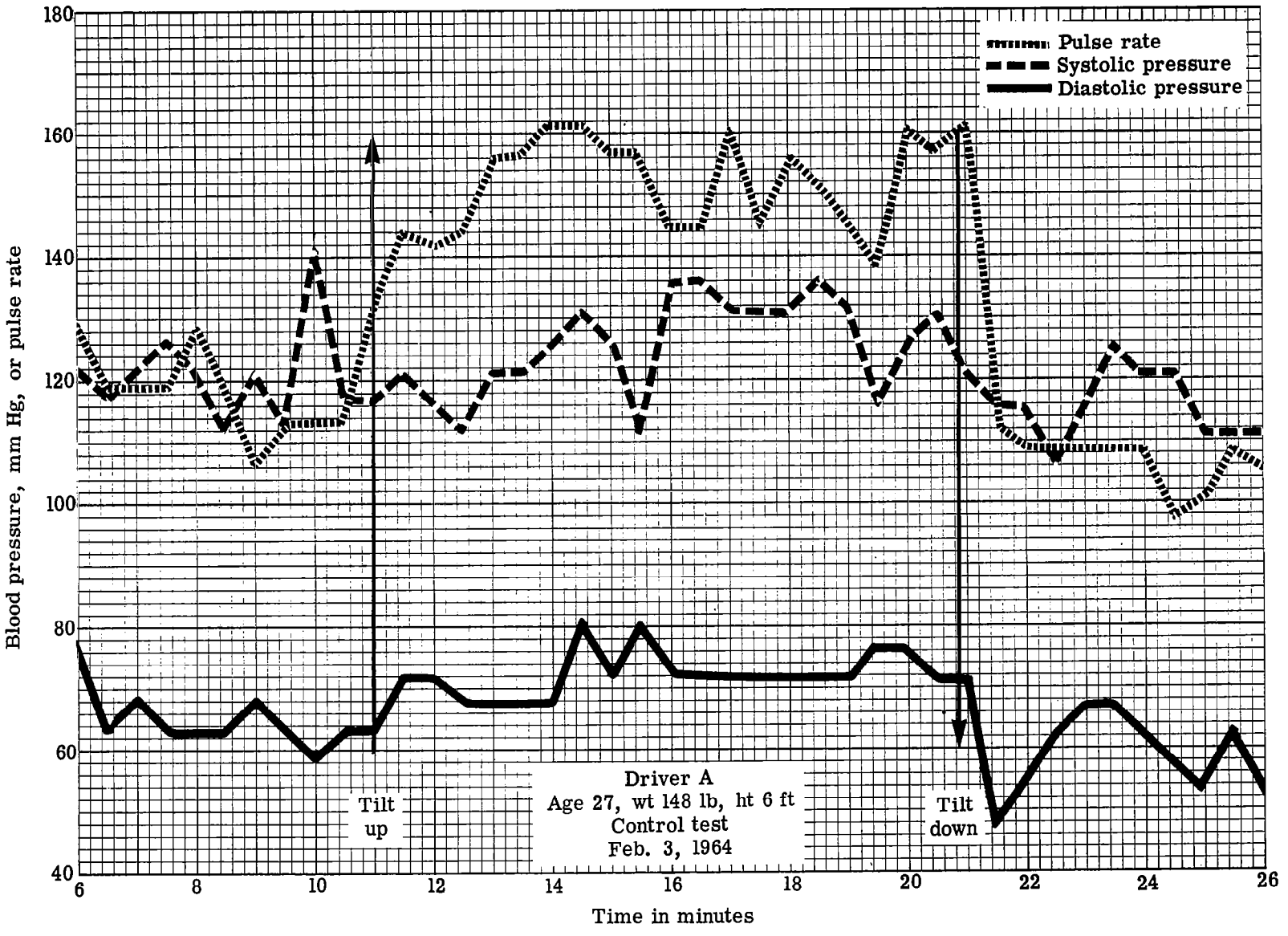



Figure 9. - Pulse rate and blood pressure during tilt-table testing.

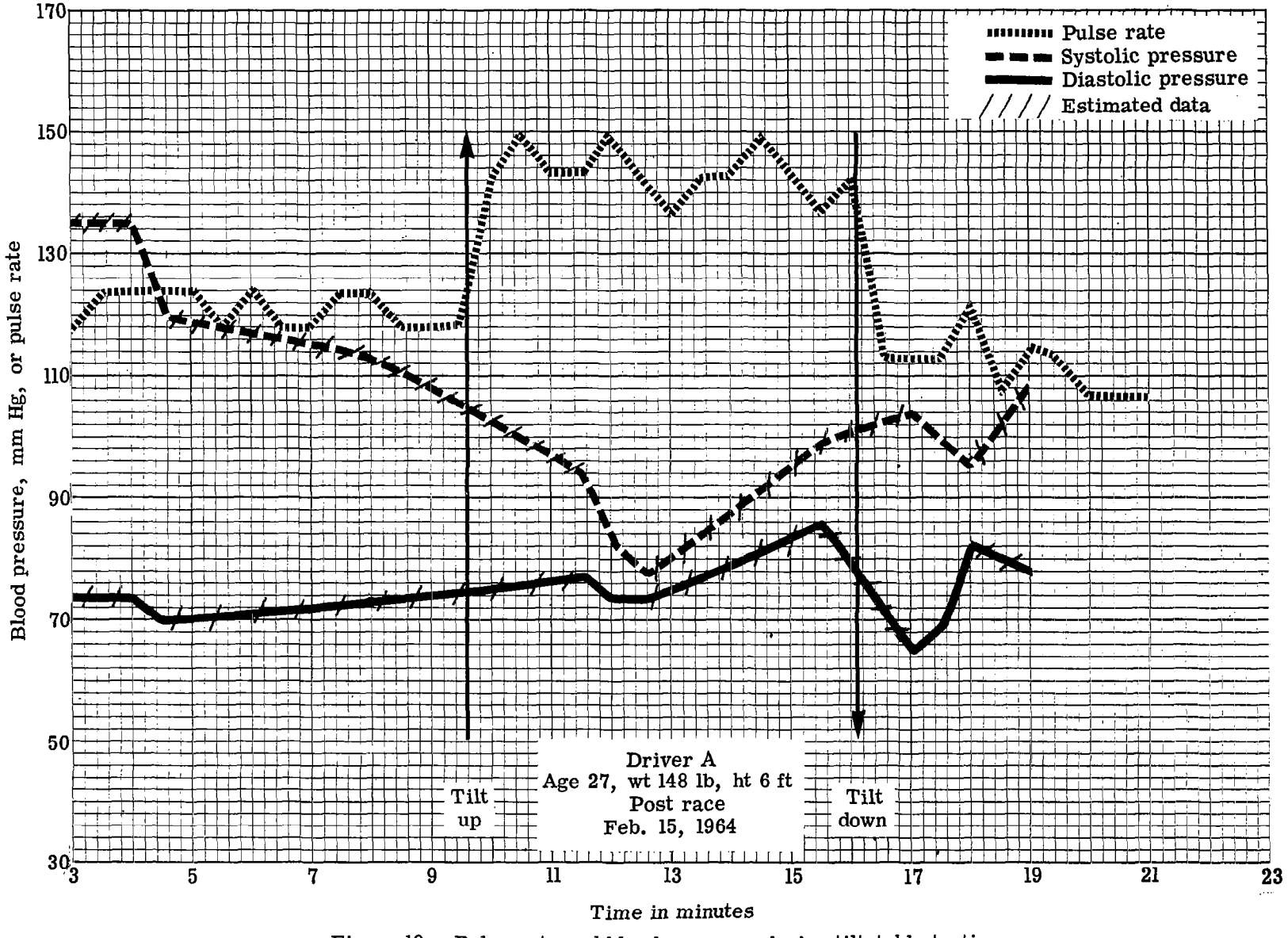



Figure 10. - Pulse rate and blood pressure during tilt-table testing.

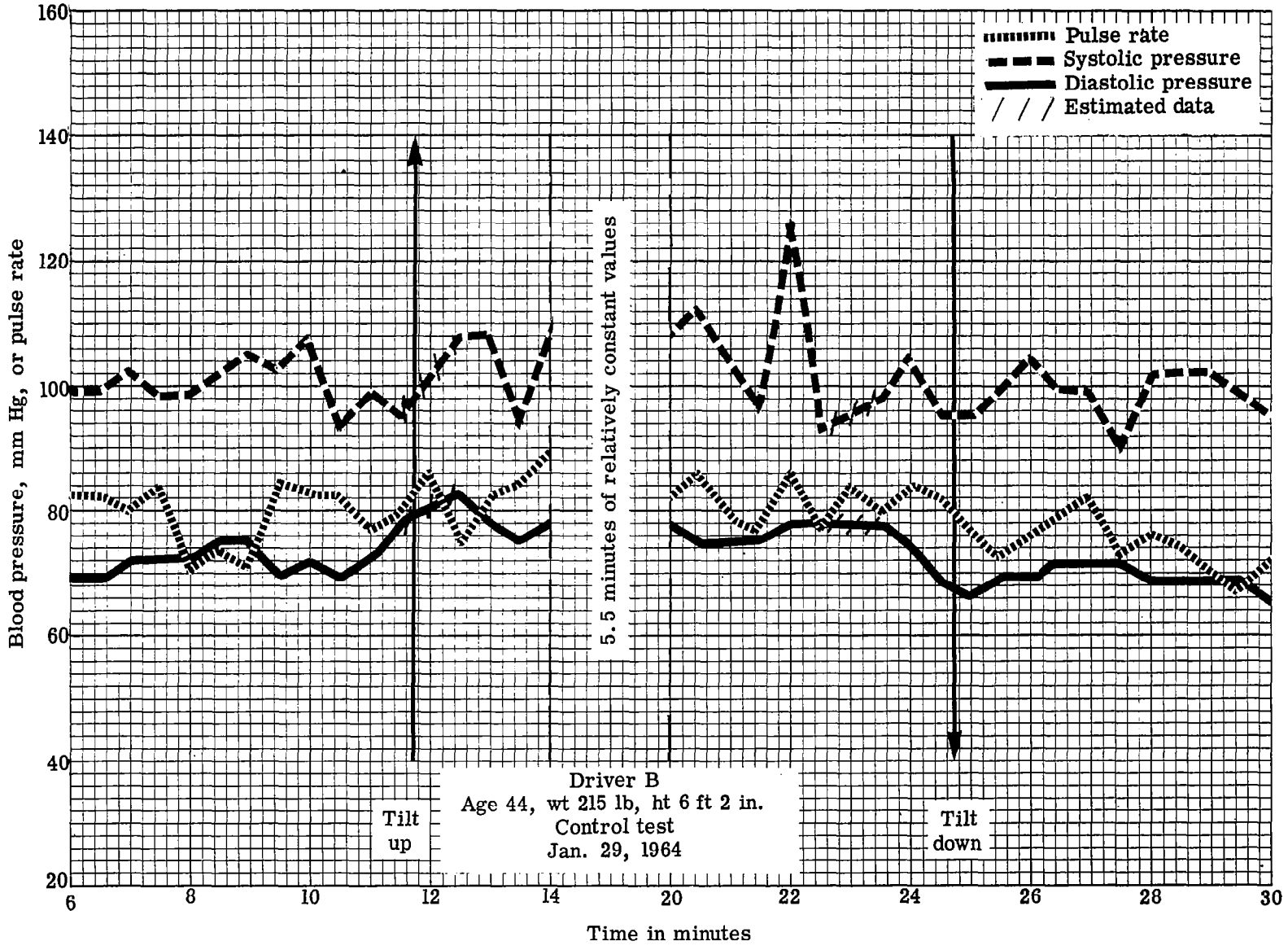



Figure 11. - Pulse rate and blood pressure during tilt-table testing.

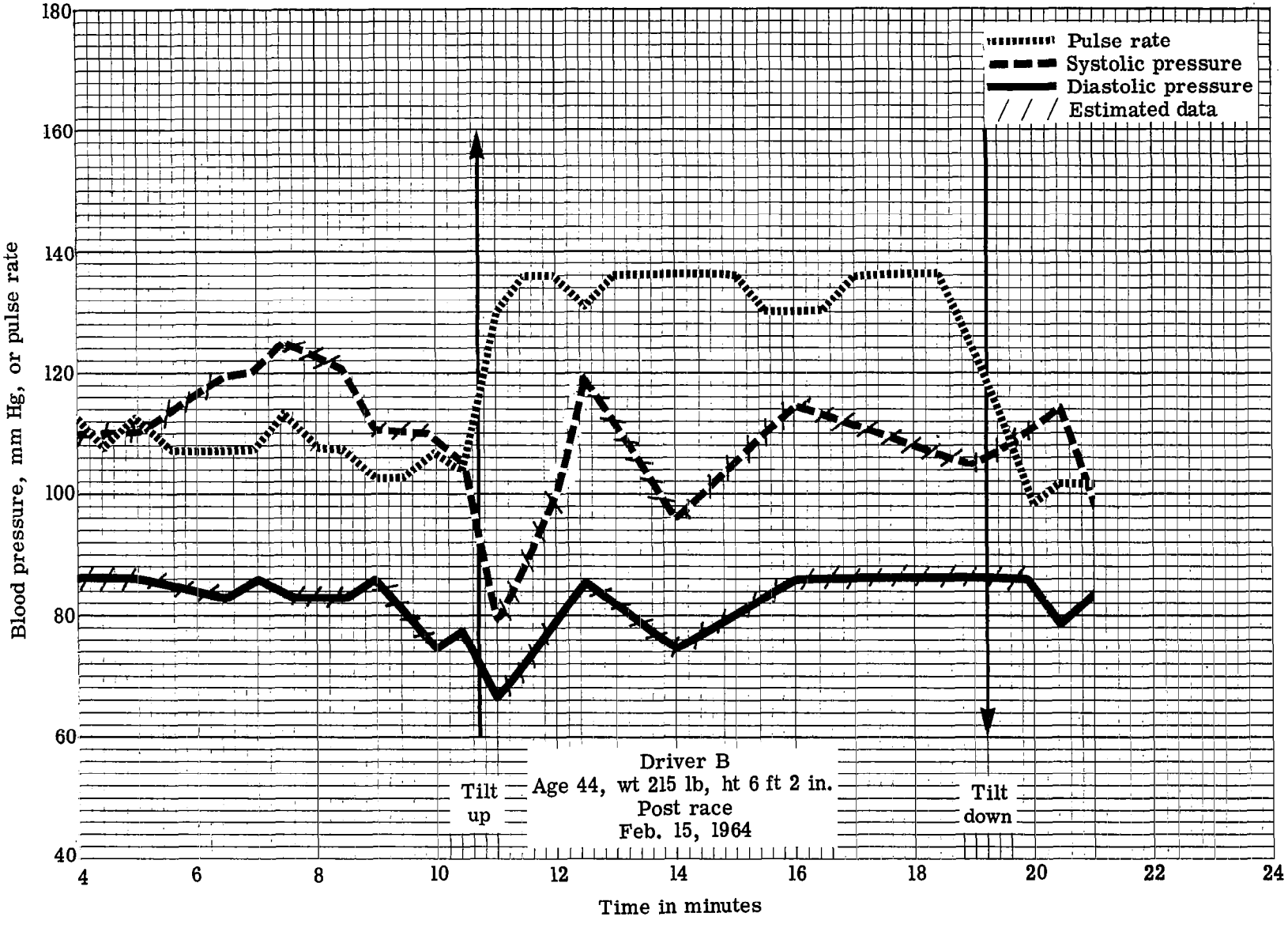



Figure 12. - Pulse rate and blood pressure during tilt-table testing.

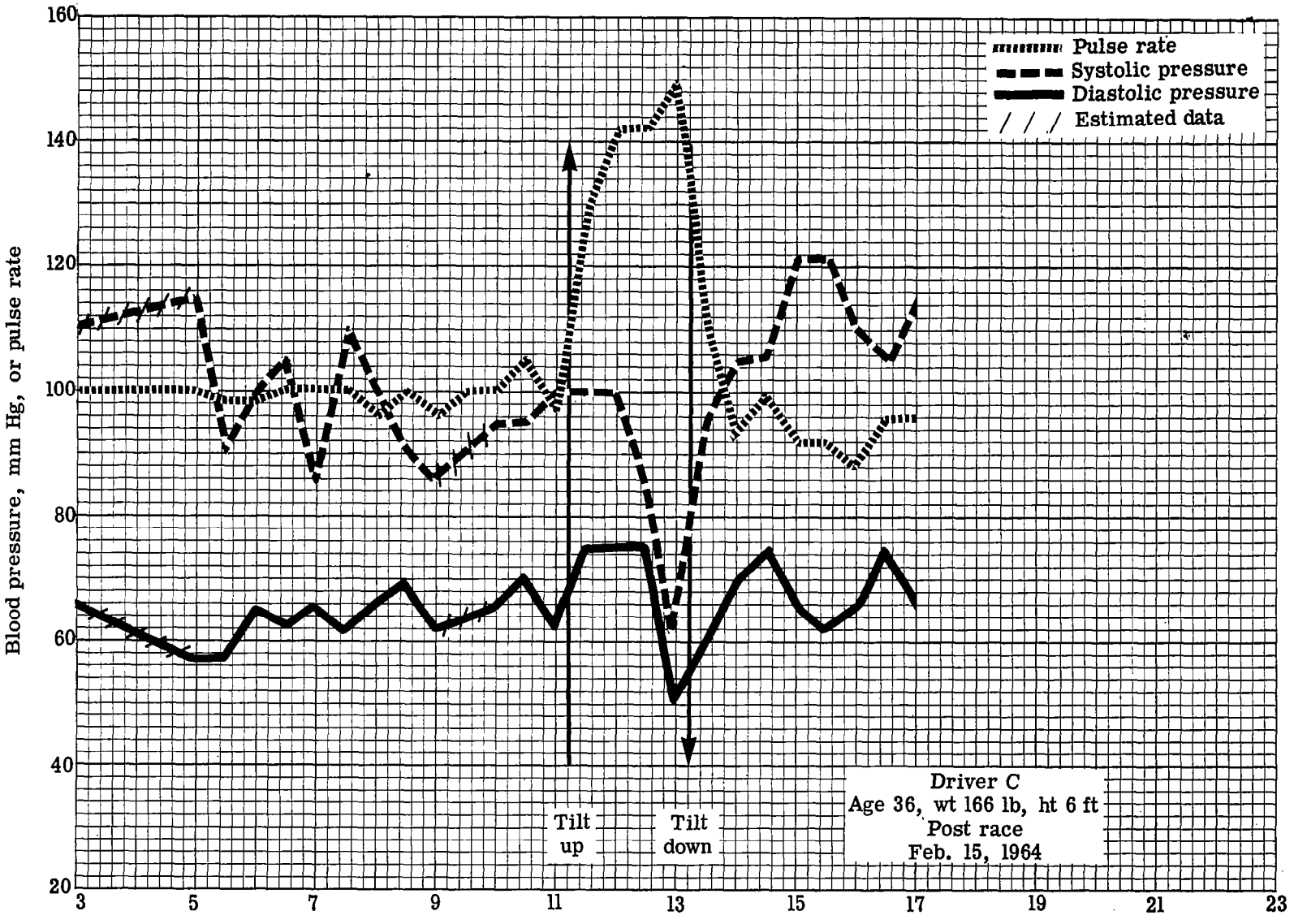



Figure 13. - Pulse rate and blood pressure during tilt-table testing.

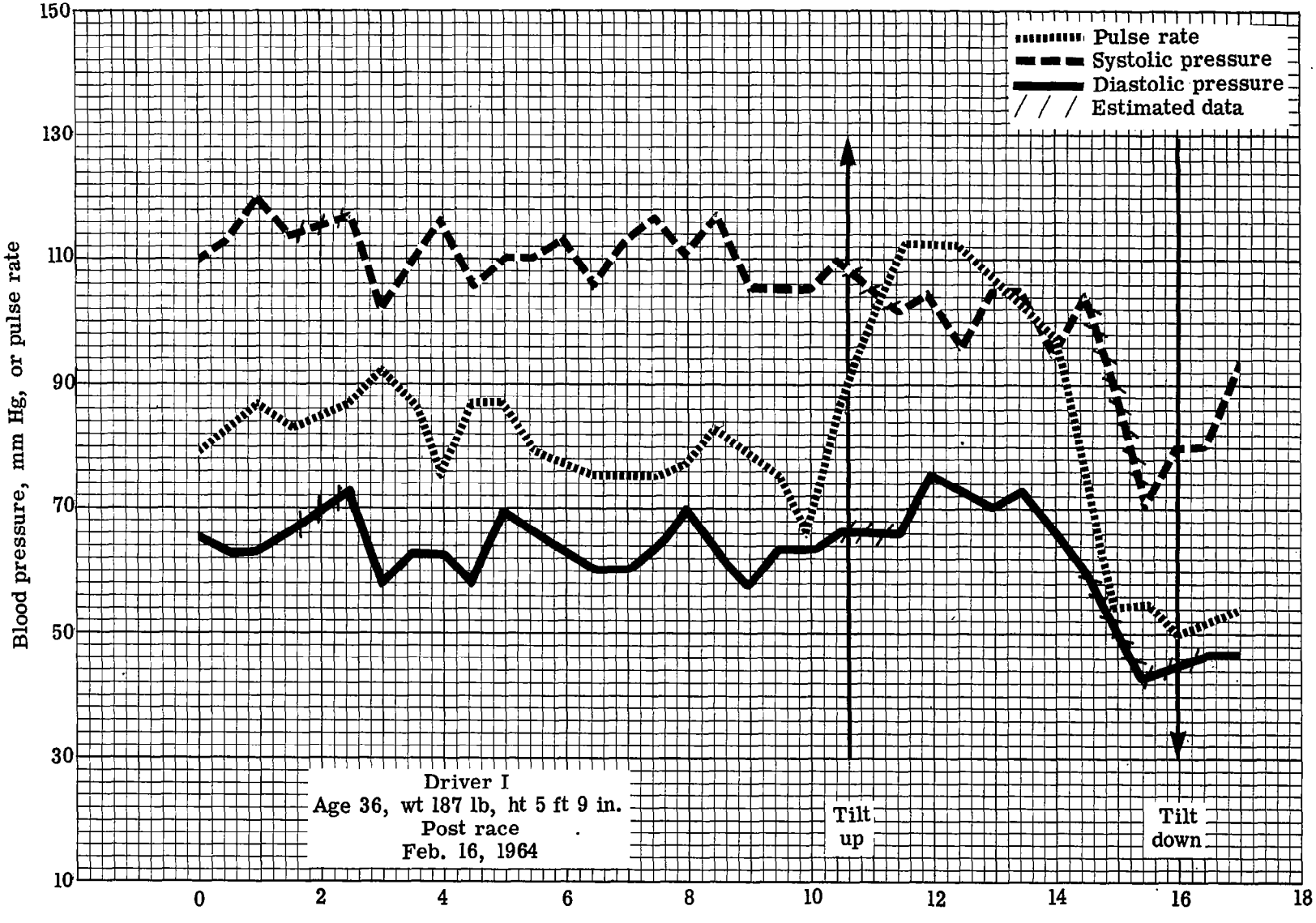



Figure 14. - Pulse rate and blood pressure during tilt-table testing.

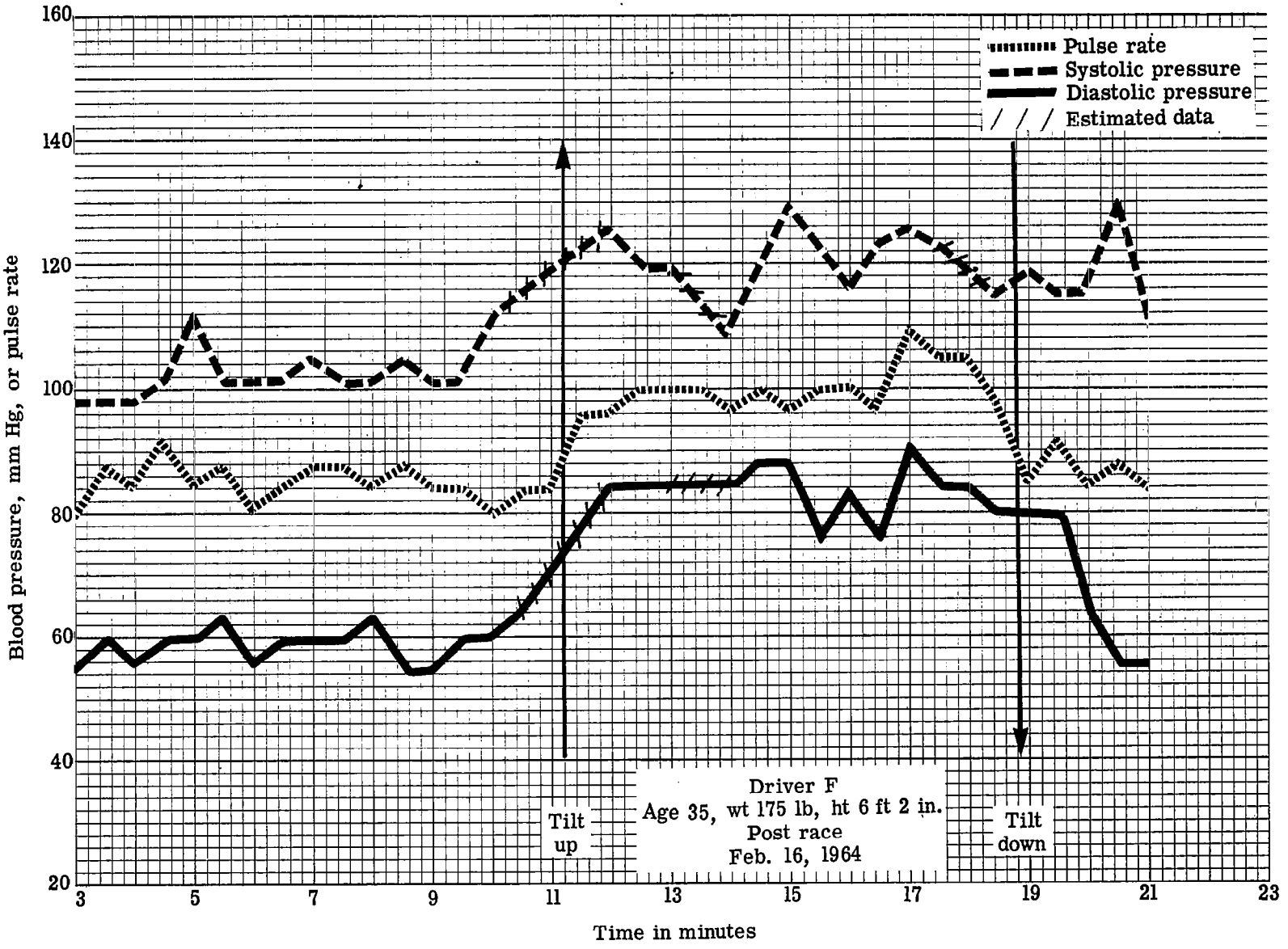



Figure 15. - Pulse rate and blood pressure during tilt-table testing.

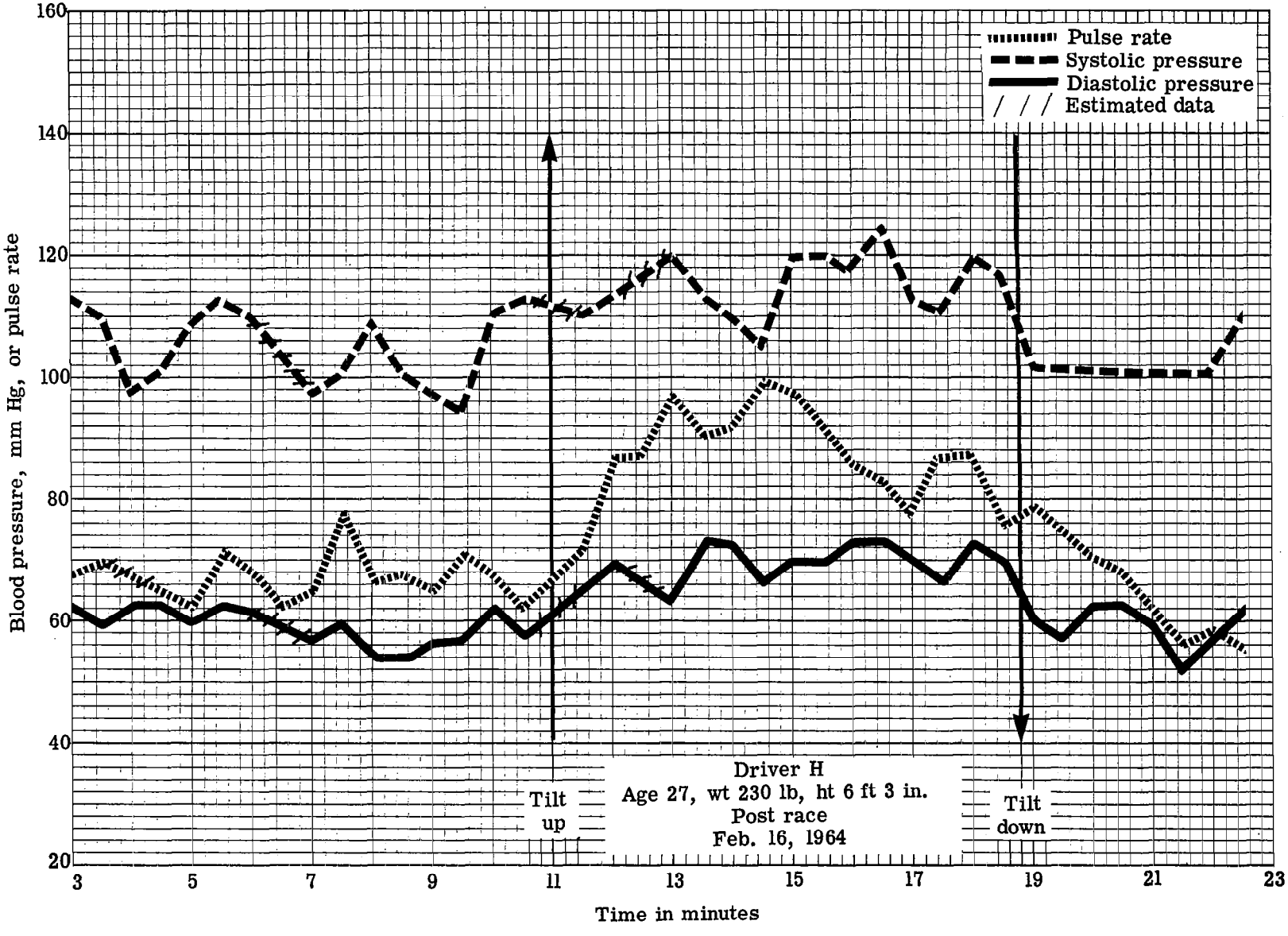



Figure 16. - Pulse rate and blood pressure during tilt-table testing.

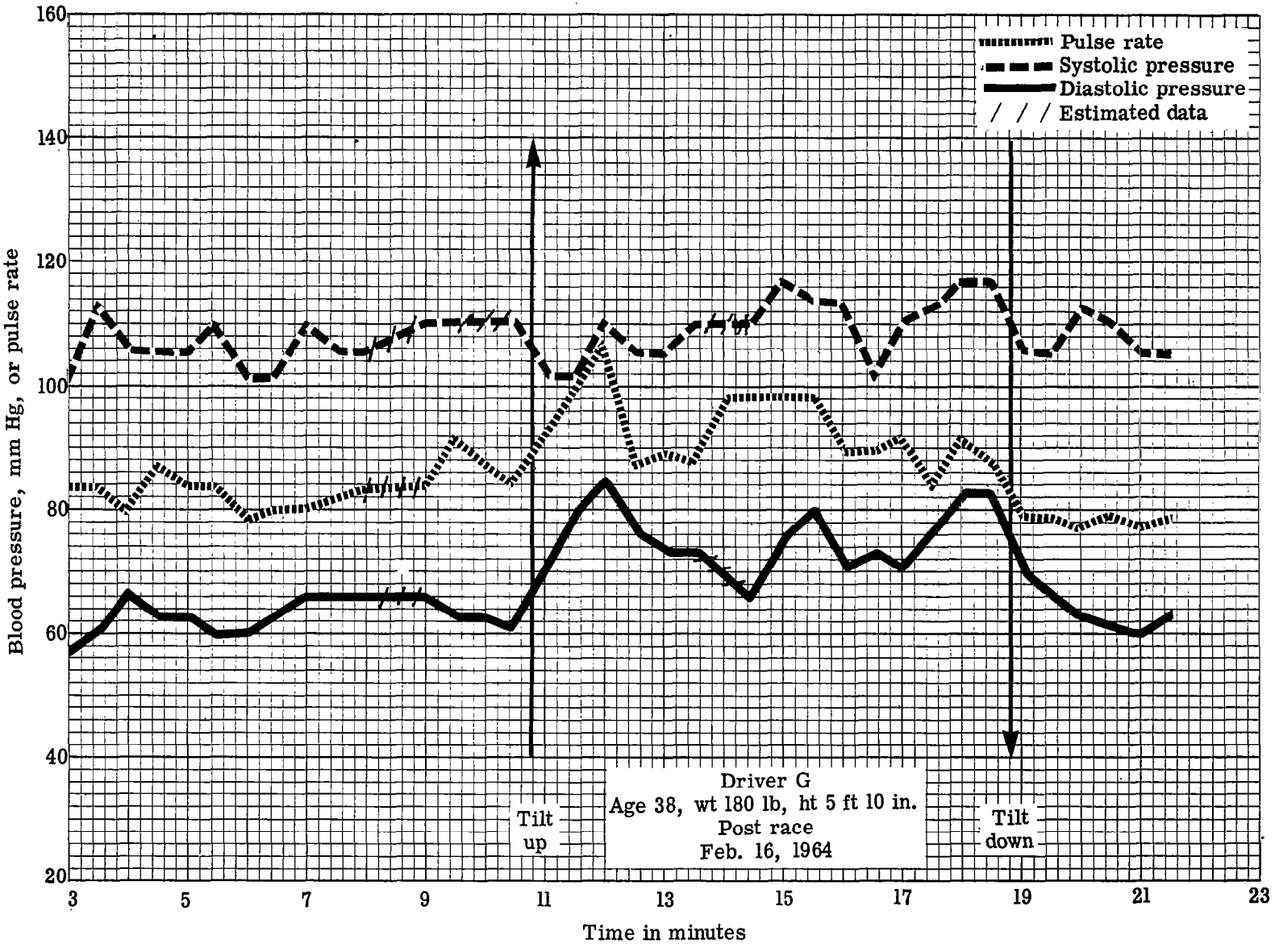



Figure 17. - Pulse rate and blood pressure during tilt-table testing.

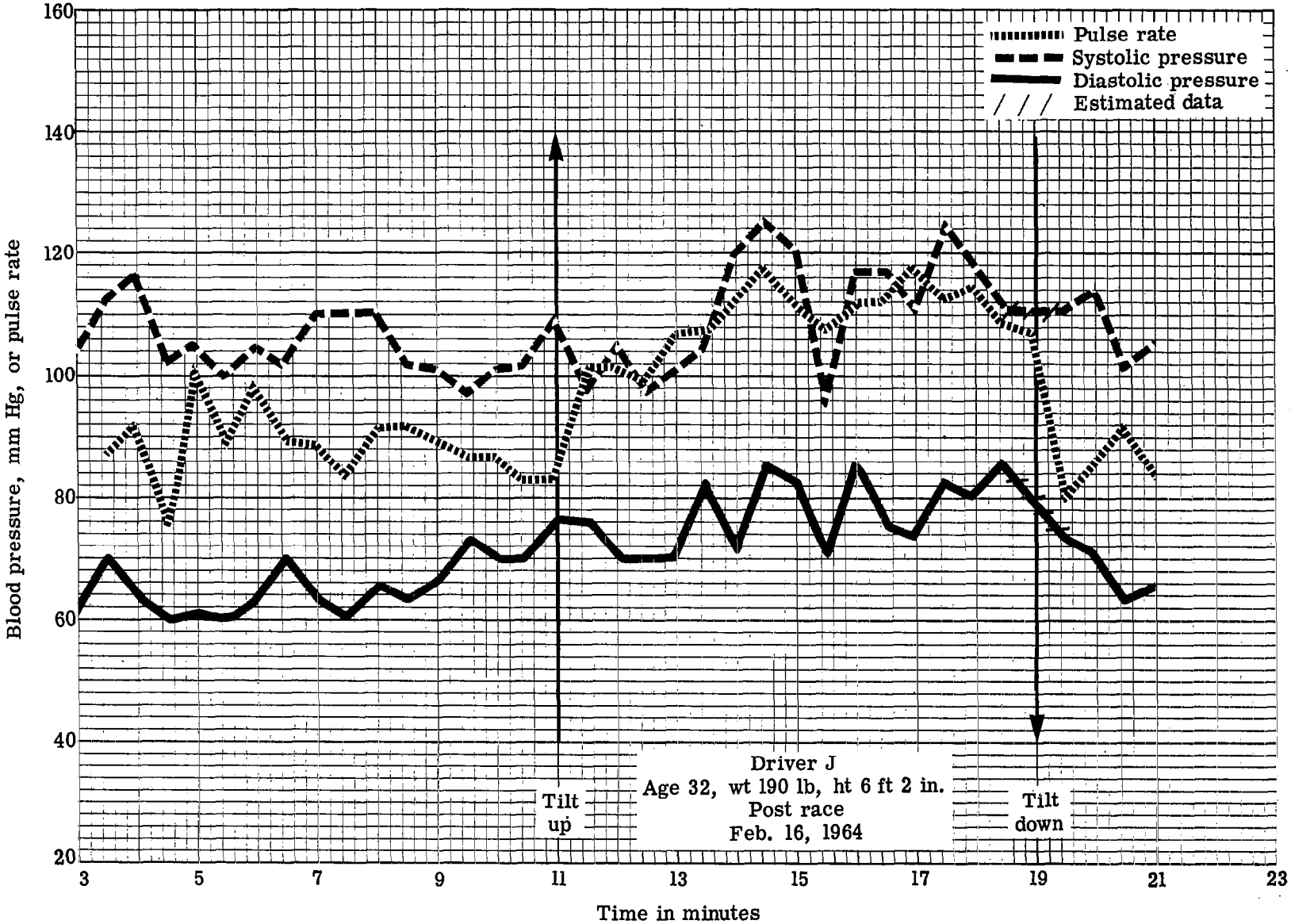



Figure 18. - Pulse rate and blood pressure during tilt-table testing.

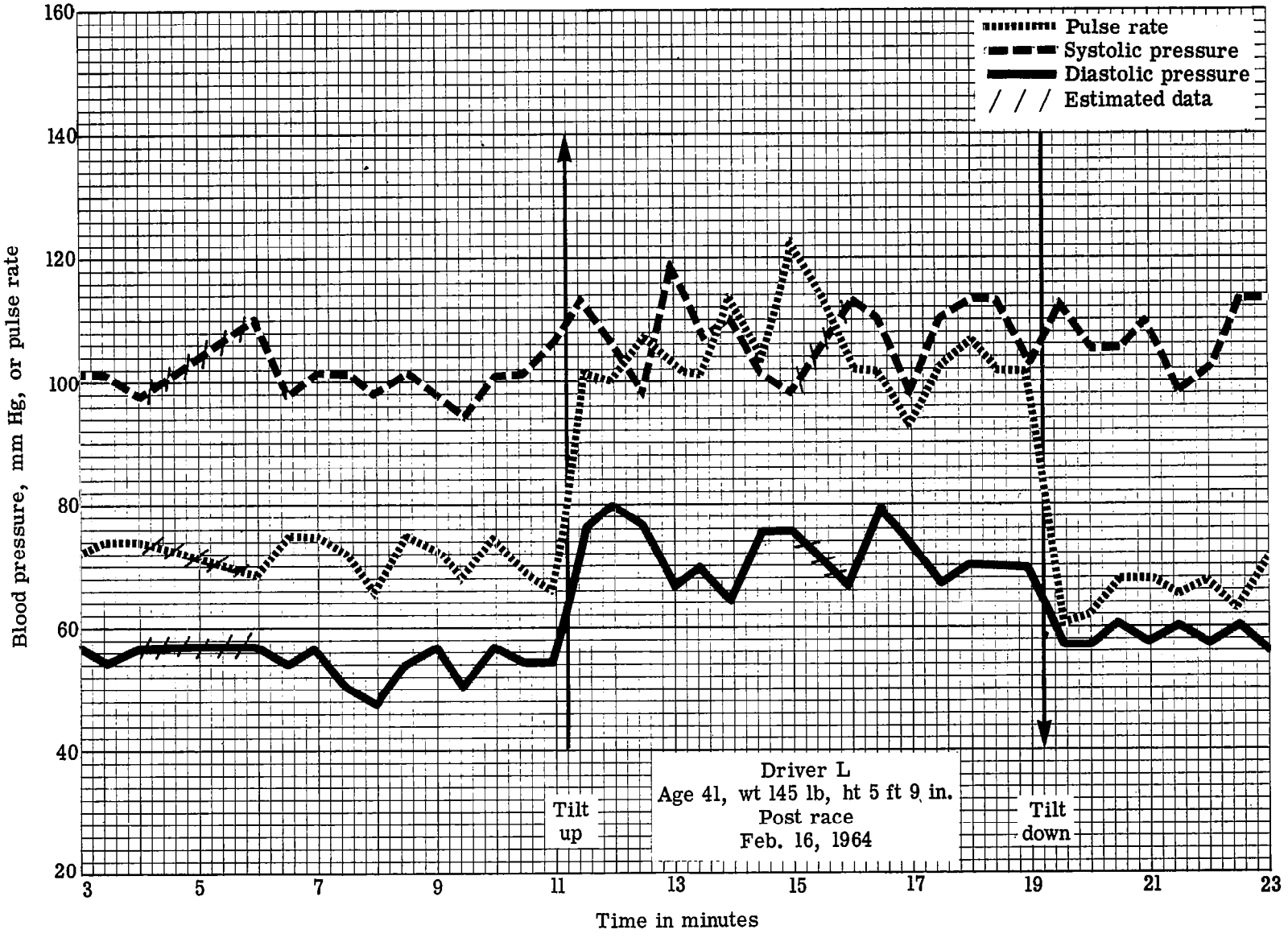



Figure 19. - Pulse rate and blood pressure during tilt-table testing.

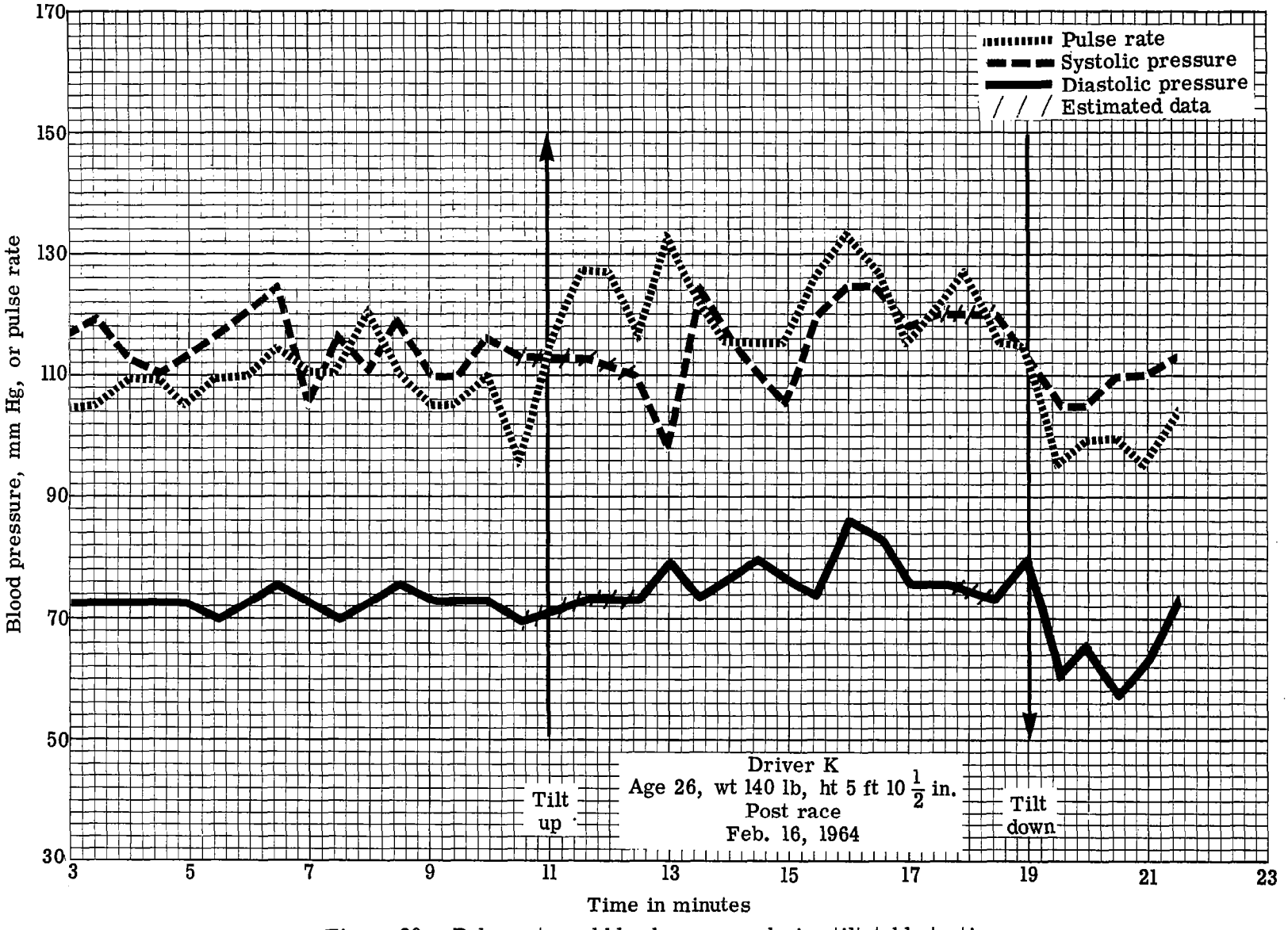



Figure 20. - Pulse rate and blood pressure during tilt-table testing.

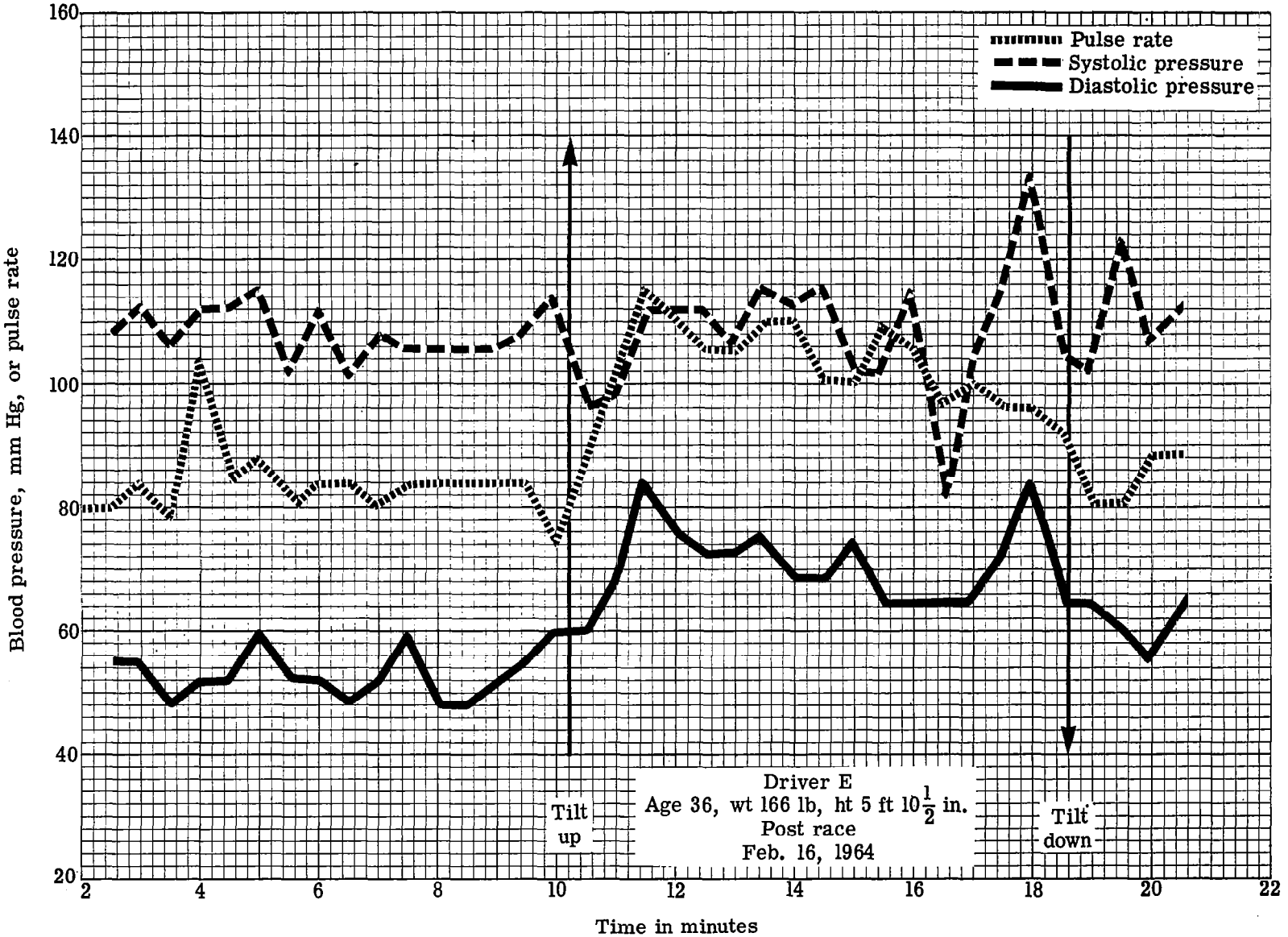



Figure 21. - Pulse rate and blood pressure during tilt-table testing.

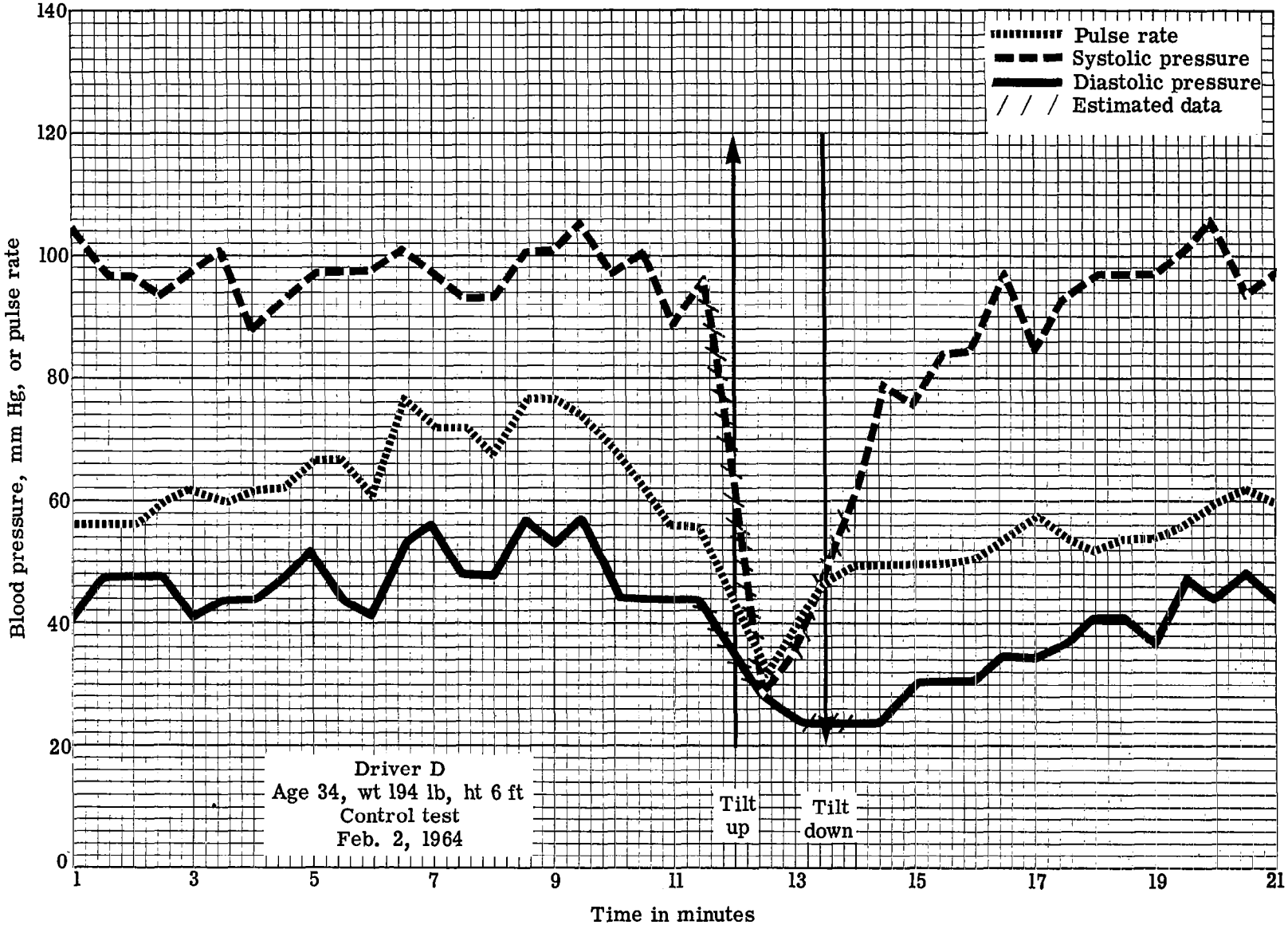



Figure 22. - Pulse rate and blood pressure during tilt-table testing.

Comment on figure 24(a) and 24(b).-

Glucose: The pre- and post-race serum glucose concentrations showed a consistent increase in the seven studies obtained. The maximum increase the first day was 87 percent with the lowest being 30 percent. Two of the same drivers were studied the second day and indicated a more profound increase in glucose concentration. The maximum increase for the second day was 303 percent with the lowest being 24 percent.

Comment on figure 25(a) and 25(b).-

Cholesterol: The pre- and post-race cholesterol values again indicate variable response. It is interesting to note that a decrease in cholesterol level was noted both days on the same driver. Others consistently had increases. Whether this is an individual variation can only be speculated. Only one driver had a significant elevation and this was in his pre-race sample. It was not possible to obtain a follow-up on this driver.

Comment on figure 26(a) and 26(b).-

Uric acid: Pre- and post-race uric acid concentrations showed variable response. Three of the seven studies performed had essentially no change while two had increases and two decreased. Two of the drivers who had no change the first day were followed the second day with significant decreases in uric acid concentration post-race.

Comment on figure 27(a) and 27(b).-

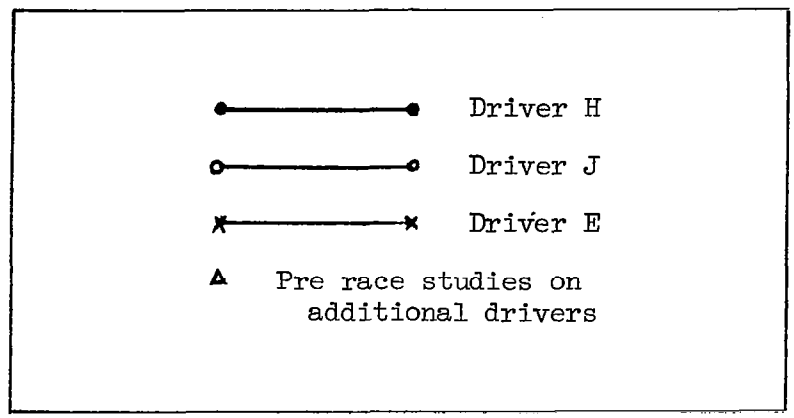
Creatinine: The predominant direction of change in the creatinine concentration was toward an increase. Six of the seven studies indicated an increase while one had a decrease on the first day but on the next day he had a significant increase. Although the normal values stated are lower than the pre-race control values, the serum creatinine may have values up to 1.8 mg percent under normal daily activity. The activities of these drivers up to the time the control samples were drawn cannot be classified as normal daily activity; therefore, the high pre-race values are not significant.

Comment on figure 28(a) and 28(b).-

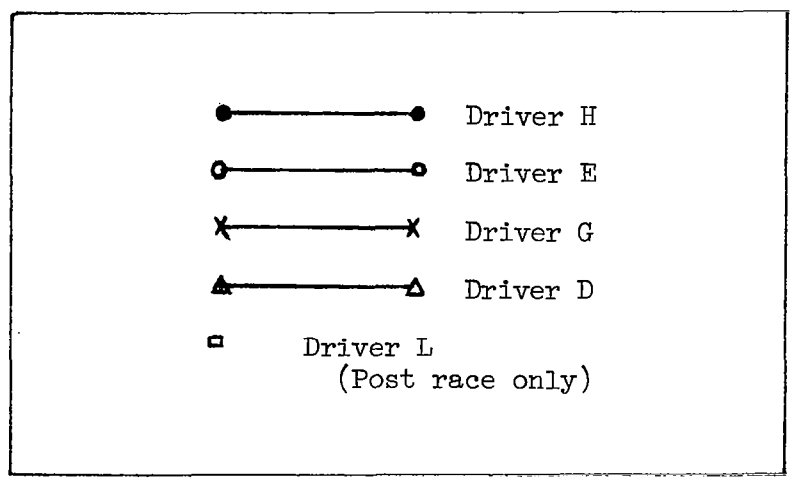
Chloride: The principle change was toward an increased chloride concentration probably associated with increased retention. Two drivers showed decreases. This may be associated with high fluid loss (perspiration) without retention as both had pre-race levels above normal.

Comment on figure 29(a) and 29(b).-

Phosphorus: The most consistent change observed was a decrease in inorganic phosphorus. This is probably a result of an increase in carbohydrate metabolism and increased excretion of acid phosphates by the kidneys.




(a) July 4, 1964




(b) July 5, 1964

Figure 23.- Legends for blood chemistries,
San Jacinto Regional Race,
Galveston, Texas

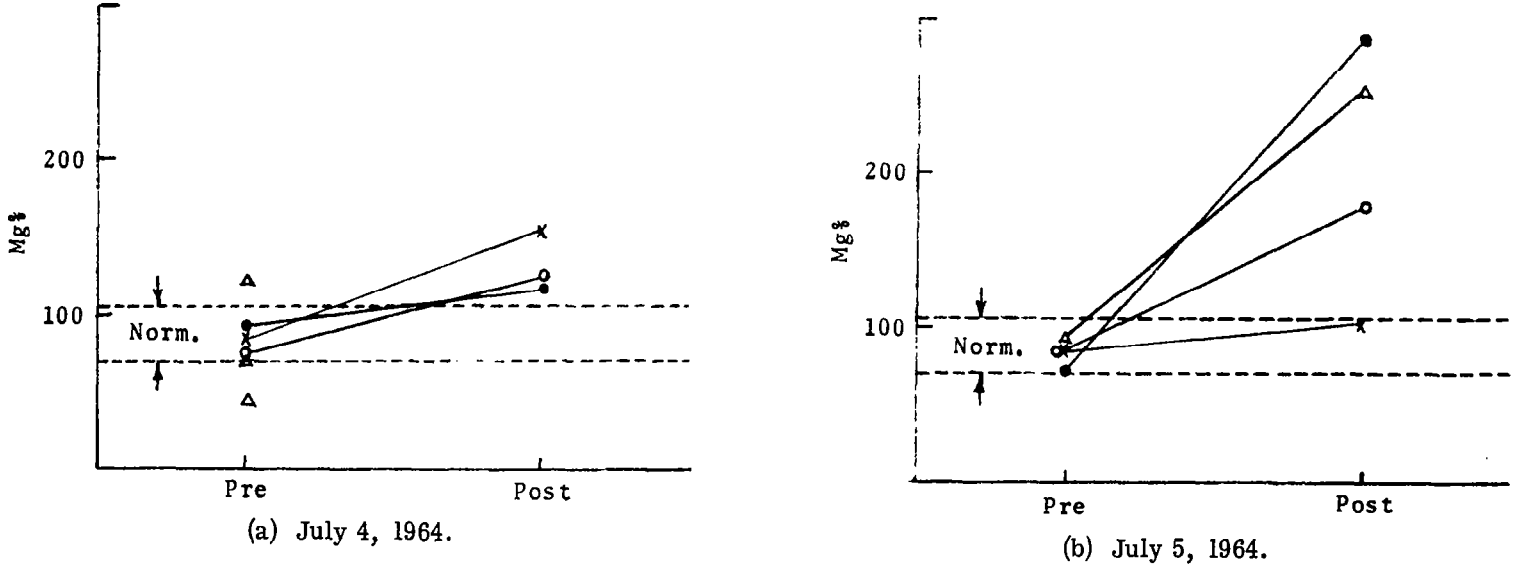



Figure 24. - Blood chemistry, glucose, San Jacinto Regional Race.

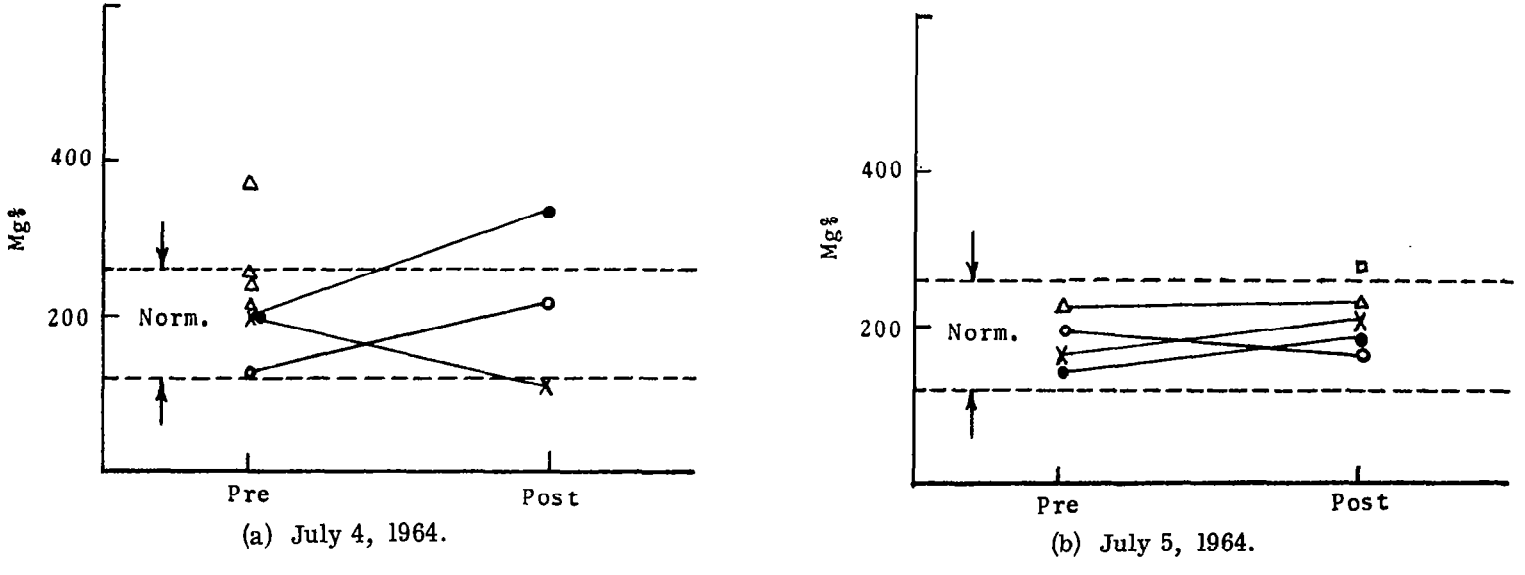
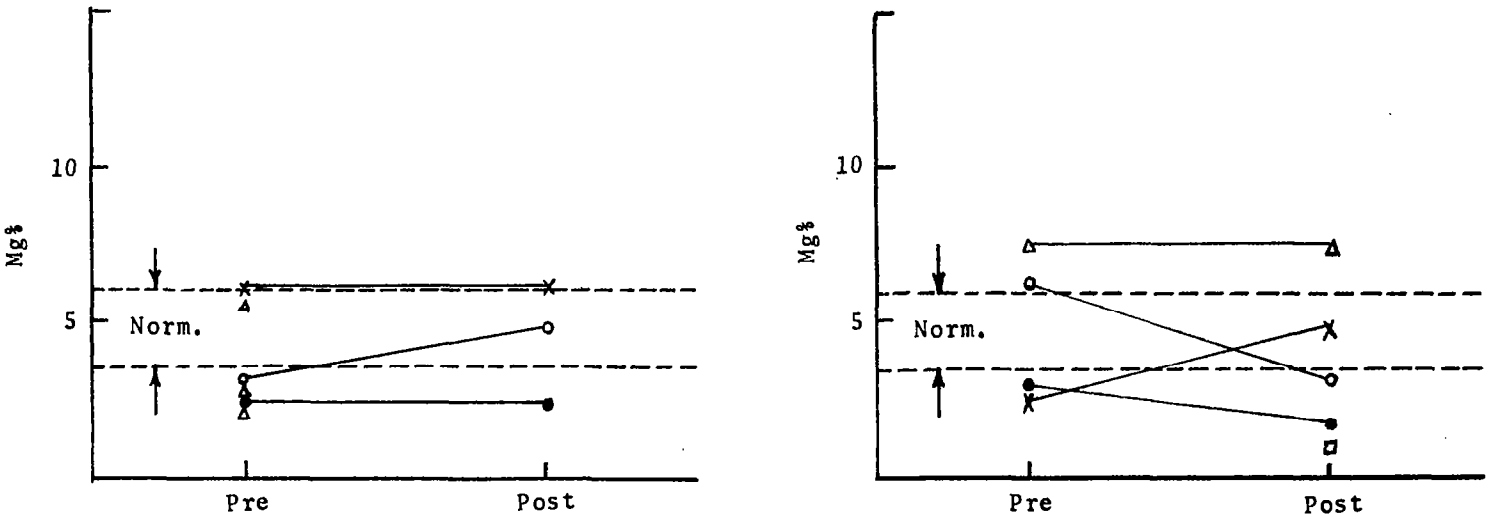
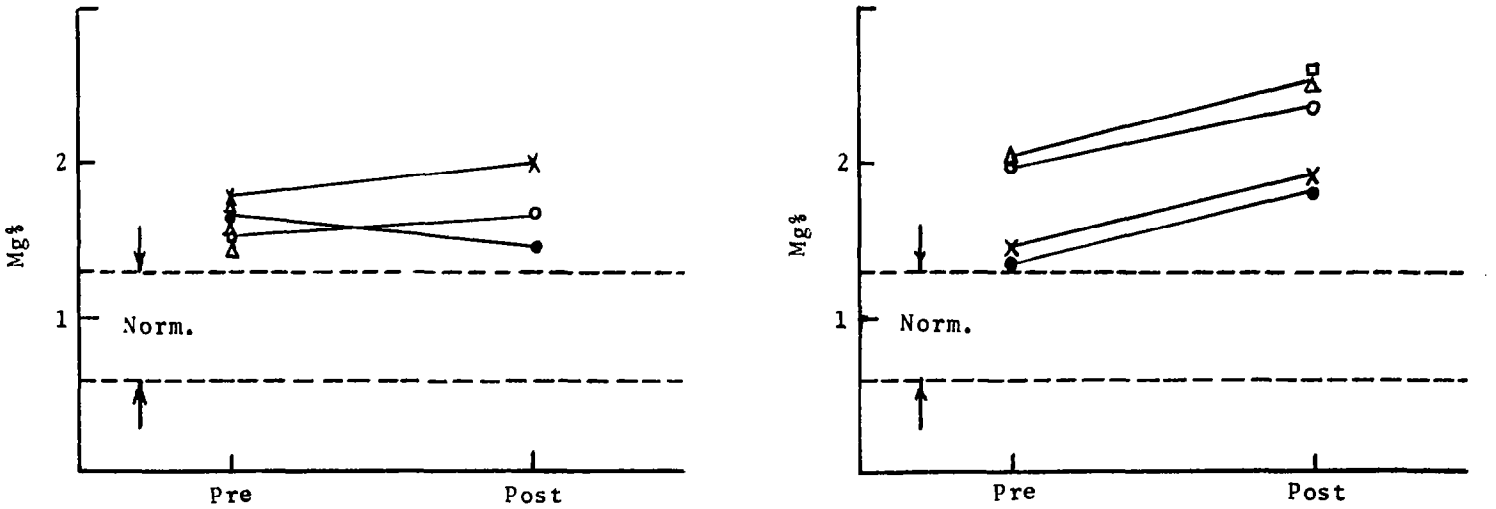



Figure 25. - Blood chemistry, cholesterol, San Jacinto Regional Race.



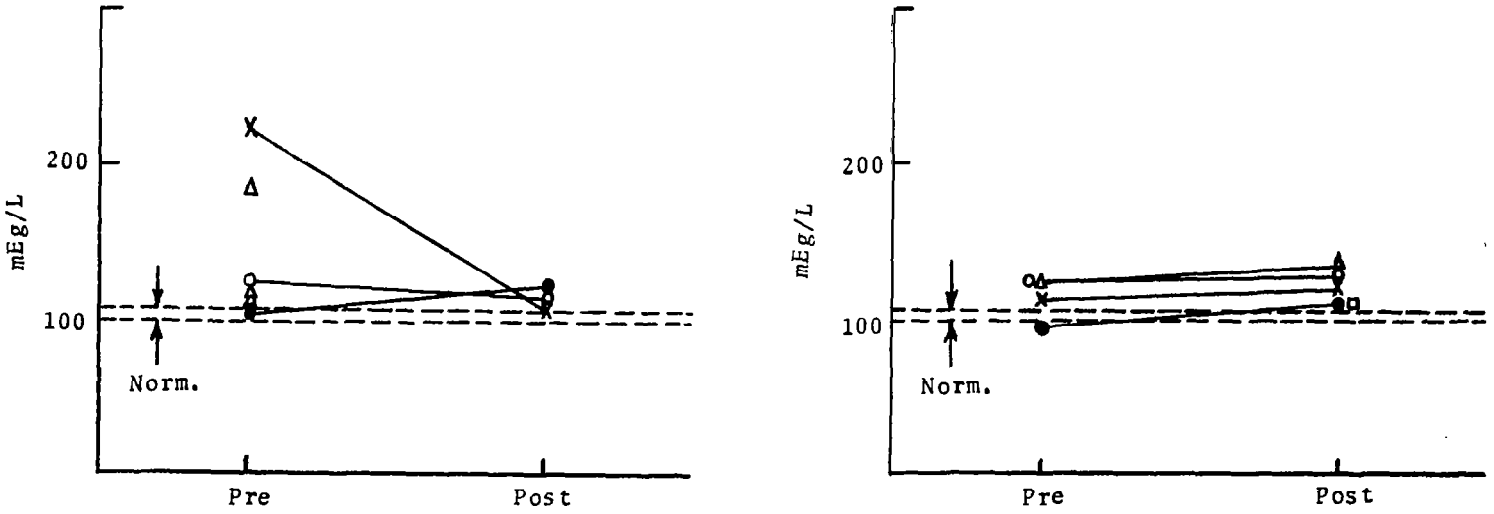

(a) July 4, 1964. (b) July 5, 1964.

Figure 26. - Blood chemistry, uric acid, San Jacinto Regional Race.



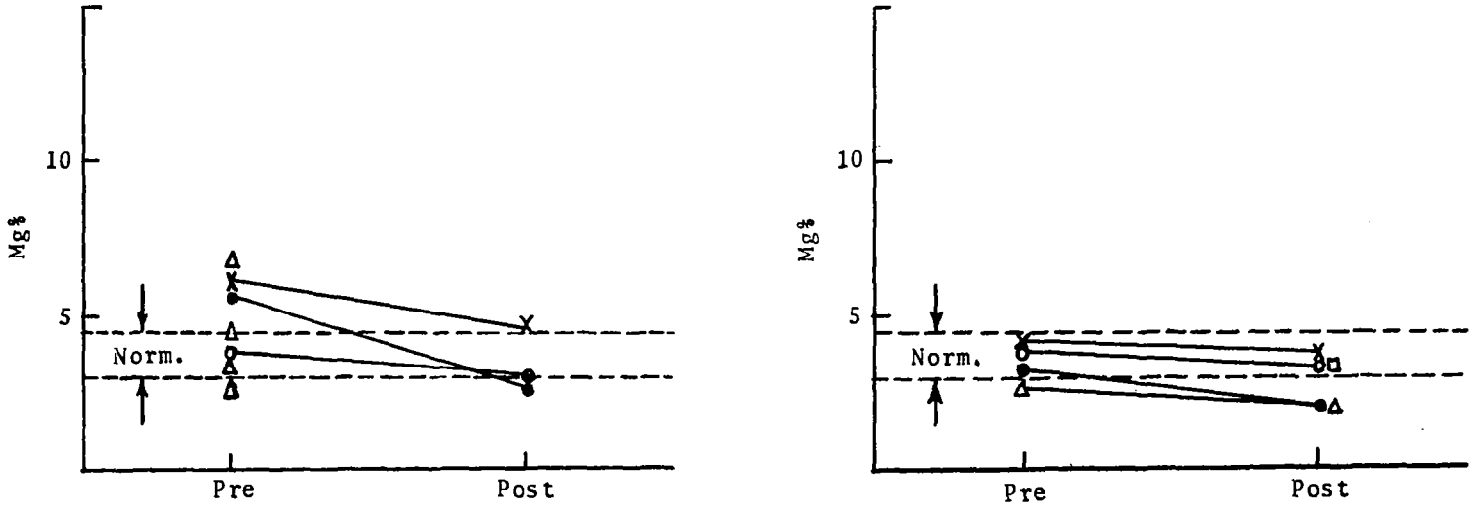

(a) July 4, 1964. (b) July 5, 1964.

Figure 27. - Blood chemistry, creatinine, San Jacinto Regional Race.




(a) July 4, 1964. (b) July 5, 1964.

Figure 28. - Blood chemistry, chlorides, San Jacinto Regional Race.




(a) July 4, 1964 (b) July 5, 1964.

Figure 29. - Blood chemistry, phosphorus, San Jacinto Regional Race.

TABLE 9.- VITAL STATISTICS OF SUBJECTS IN RACES AT GALVESTON, TEXAS, IN 1964

| Driver | Physical description | | | Occupation | Marital status and no. of children | Education | Racing experience | | Rest prior to race |
|---|---|---|---|---|---|---|---|---|---|
| | Weight | Height | Age | | | | Type | No. of years | |
| Driver A | 156 | 5 ft 9 1/2 in | 37 | Photographer (commercial) | Yes 2 children | M.S. in photography | Sports | 5 | 6 hr sleep |
| Driver B | 200 | 6 ft 2 in | 34 | Business executive | Yes 1 child | 4 yr college | Sports | 8 | 7 hr sleep |
| Driver C | 170 | 5 ft 9 in | 51 | M.D. (Radiologist) | Yes 3 children | M.D. L.L.B. | Sports | 3 | 7 hr sleep |
| Driver D | 180 | 5 ft 10 in | 47 | Retired Col. | Yes 2 children | West Point graduate | Sports | 1 | 9 hr sleep |
| Driver E | 190 | 6 ft 2 in | 28 | Mechanic and sales | Yes 2 children | 1 yr college G.M. Train. Center | Sports | 3 | |
| Driver F | 173 | 5 ft 11 in | 35 | Electronics business | Yes 4 children | | Sports | 6 | 5 hr sleep |
| Driver G | 193 | 6 ft 2 in | 39 | Airline pilot | Yes 2 children | Business math. major | Sports | 12 | 8 hr sleep |
| Driver H | 165 | 5 ft 10 in | 33 | Chemical operator | Yes 4 children | 1 1/2 yr Jr. College | Sports | 3 | No sleep before race working and drive |
| Driver I | | 5 ft 10 in | 51 | Auto mechanic | Yes 1 child | | Sports | 4 | 7 hr sleep |
| Driver J | 180 | 5 ft 9 in | 32 | M.D. | Yes 2 children | M.D. | Sports | 3 | 5 hr sleep |
| Driver K | 200 | 6 ft 2 in | 43 | Personnel Director | Yes 1 child | High school | | 1 | 5 1/2 hr sleep |
| Driver L | 160 | 5 ft 8 in | 43 | Investor | Yes 3 children | 2 yr college | | 10 | 7 hr sleep |

TABLE 10.- PRE- AND POST-RACE TEMPERATURE, PULSE, AND BLOOD PRESSURE READINGS OF DRIVERS IN THE RACES AT GALVESTON, TEXAS

| Name | Date | Time | | Temperature, °F | | Blood pressure | |
|---|---|---|---|---|---|---|---|
| | | Pre-race | Post-race | Pre-race | Post-race | Pre-race | Post-race |
| Driver A | 7-4-64 | 4:35 | | 99.0 | 100.2 | 110/70 | |
| Driver B | 7-5-64 | 11:50 | | 99.0 | 99.2 | 128/80 | 138/110 |
| Driver D | 7-5-64 | 11:30 | 3:30 | 99.4 | 99.8 | 138/90 | 150/100 |
| Driver E | 7-4-64 | 4:45 | 5:40 | 99.6 | 101.0 | 130/80 | |
| Driver E | 7-5-64 | 11:00 | 3:45 | 100.0 | 100.8 | 130/80 | |
| Driver F | 7-5-64 | 11:55 | 2:30 | 99.0 | 99.2 | 118/78 | |
| Driver G | 7-5-64 | 3:00 | | 97.2 | 101.0 | 130/70 | 140/80 |
| Driver H | 7-4-64 | 1:45 | 3:25 | 99.8 | 100.4 | | 130/90 |
| Driver H | 7-5-64 | 9:45 | 12:40 | 99.2 | 100.2 | 104/78 | |
| Driver I | 7-4-64 | 2:23 | 4:30 | 98.6 | | | |
| Driver J | 7-4-64 | 4:10 | 5:10 | 98.6 | 99.4 | | |
| Driver K | 7-4-64 | 2:10 | 4:00 | 99.0 | 99.2 | | |

Comment: The maximum ambient temperature was 102° F with intermittent cloudiness and light breeze so that this day was not oppressively warm. Of 11 drivers only 4 showed a rise in body temperature of more than 1° during a race. The maximum body temperature recorded was 101.0° F but this represented a rise of 3.8° for one driver.

Three drivers with blood pressures before and after racing all showed a slight increase in both systolic and diastolic pressure with pulse pressure remaining constant in two and narrowing slightly in one driver.

TABLE 11.- WEIGHT-CHANGE STUDIES CONDUCTED ON DRIVERS IN THE GALVESTON RACES IN 1964

| Driver | Date | Pre-race weight | Post-race weight | Weight loss, lb | Percent |
|---|---|---|---|---|---|
| Driver A | 7-4-64 | 153 | --- | --- | --- |
| Driver B | 7-5-64 | --- | --- | --- | --- |
| Driver D | 7-5-64 | 180 | 180 | 0 | |
| Driver E | 7-4-64 | 186 | --- | --- | --- |
| Driver E | 7-5-64 | 183 | 182 | 1 | .5 |
| Driver F | 7-5-64 | --- | 173 | --- | --- |
| Driver G | 7-5-64 | 196 | 193 | 3 | 1.5 |
| Driver H | 7-4-64 | 160 | 159 | 1 | .6 |
| Driver H | 7-5-64 | 160 | 157 | 3 | 1.9 |
| Driver I | 7-4-64 | 155 | 154 | 1 | .6 |
| Driver J | 7-4-64 | 177 | 177 | 0 | |
| Driver K | 7-4-64 | 198 | 195 | 3 | 1.5 |

Comment: Three drivers each showed a weight loss of 3 lb. The percentage losses of body weight paired with temperature changes were: 1.9 percent and +1.0° F, 1.5 percent and +0.2° F, and 1.5 percent and +3.8° F.

No correlation can be attempted but future observations with larger numbers of subjects and closer attention to details of cockpit temperature, car construction, and clothing may be profitable.

TABLE 12.- BLOOD-CHEMISTRY STUDIES CONDUCTED ON DRIVERS

IN THE RACES AT GALVESTON, TEXAS

| Driver | Time | Glucose | Cholesterol | Uric acid | Creatinine | Chloride | Phosphorus |
|---|---|---|---|---|---|---|---|
| July 4, 1964 | | | | | | | |
| Driver A | Pre-race | | 250 | 2.8 | 1.47 | 184 | 6.9 |
| | Post-race | | | | | | |
| Driver C | Pre-race | 42 | 208 | 5.5 | 1.68 | 117 | 4.5 |
| | Post-race | | | | | | |
| Driver E | Pre-race | 82 | 208 | 6.0 | 1.79 | 223 | 6.1 |
| | Post-race | 153 | 116 | 6.1 | 2.00 | 110 | 4.7 |
| Driver H | Pre-race | 91 | 208 | 2.5 | 1.68 | 104 | 5.6 |
| | Post-race | 118 | 332 | 2.4 | 1.47 | 120 | 2.7 |
| Driver I | Pre-race | 120 | 258 | 2.3 | 1.58 | 111 | 2.7 |
| | Post-race | | | | | | |
| Driver J | Pre-race | 72 | 124 | 2.9 | 1.52 | 123 | 3.7 |
| | Post-race | 122 | 214 | 4.8 | 1.68 | 113 | 2.8 |
| Driver K | Pre-race | 70 | 378 | 26.0 | 1.58 | 116 | 3.6 |
| | Post-race | | | | | | |
| July 5, 1964 | | | | | | | |
| Driver D | Pre-race | 92 | 227 | 7.7 | 2.02 | 125 | 2.7 |
| | Post-race | 253 | 236 | 7.5 | 2.53 | 137 | 2.2 |
| Driver E | Pre-race | 84 | 191 | 6.3 | 2.00 | 125 | 4.0 |
| | Post-race | 179 | 173 | 3.3 | 2.36 | 130 | 3.4 |
| Driver G | Pre-race | 84 | 160 | 2.7 | 1.47 | 117 | 4.1 |
| | Post-race | 104 | 218 | 4.9 | 1.92 | 124 | 3.8 |
| Driver H | Pre-race | 71 | 145 | 2.8 | 1.37 | 98 | 3.1 |
| | Post-race | 286 | 182 | 1.9 | 1.81 | 109 | 2.2 |
| Driver L | Pre-race | | | | | | |
| | Post-race | | 282 | 1.3 | 2.63 | 112 | 3.5 |
| Normal | | 70-105 mg percent | 120-160 mg percent | 3.5-6.0 mg percent | 0.6-1.3 mg percent | 100-106 meq/l | 3.0-4.5 mg percent |

Comment on table 13.-

Sugar: Pre-race urine samples all had sugar present except that from one driver. The qualitative amount ranged from a trace up to 2+. Post-race samples indicated either no change in excretion of sugars or increases up to 3+. One driver had a decreased excretion during competition, falling from 2+ to trace amounts. His pre-race serum concentration was 70 mg percent at the same time his urine sugar was 2+.

This pattern may be expected in non-fasting individuals if at one time he had a high intake of glucose with no voiding until the time the samples were taken.

Sodium: All drivers from whom we were able to obtain pre- and post-race urine samples during the second day of racing had a decreased sodium concentration. The percent decrease ranged from 5 to 35 percent. There is close correlation between the percent decrease in sodium excretion and the percent increase in chloride concentration in the blood.

Potassium: An increased potassium concentration was observed in three of the four drivers studied. The fourth had no change. Driver F had mechanical trouble while on the track and there was a long delay before return to the testing area. This may account for a return to normal potassium concentration in the urine. Apart from this individual, the magnitude of increase appears to be associated with the magnitude of decrease in sodium concentration.

Osmolality: The total concentration of solutes in the urine appears to be quite variable with maximum changes toward a decreased concentration. This correlates with the decrease in sodium concentration in the urine being greater than the increase in potassium. A decreased excretion of other materials may account in part for some of the observed increases in plasma concentration.

Additional information on creatinine excretion and uric acid excretion may be useful. Creatinine is not reabsorbed and the concentration in the urine may indicate the water concentration.

TABLE 13.- PRE- AND POST-RACE URINALYSES OF DRIVERS

IN THE RACES AT GALVESTON, TEXAS

| Name | Time | Sugar | Na, meq/l | Na, meq/l | Osmolality |
|---|---|---|---|---|---|
| July 4, 1964 | | | | | |
| Driver A | Pre-race | Trace | --- | --- | --- |
| | Post-race | --- | --- | --- | --- |
| Driver E | Pre-race | Trace | --- | --- | --- |
| | Post-race | Trace | --- | --- | --- |
| Driver H | Pre-race | Trace | --- | --- | --- |
| | Post-race | 1+ | --- | --- | --- |
| Driver I | Pre-race | 1+ | --- | --- | --- |
| | Post-race | 3+ | --- | --- | --- |
| Driver J | Pre-race | Negative | --- | --- | --- |
| | Post-race | Trace | --- | --- | --- |
| Driver K | Pre-race | 2+ | --- | --- | --- |
| | Post-race | Trace | --- | --- | --- |
| July 5, 1964 | | | | | |
| Driver B | Pre-race | 1+ | --- | --- | --- |
| | Post-race | 2+ | 132 | 66 | 869.2 |
| Driver E | Pre-race | Trace | 124 | 112 | 830.5 |
| | Post-race | Trace | 118 | 116 | 854.3 |
| Driver F | Pre-race | Trace | 184 | 72 | 960.6 |
| | Post-race | Trace | 120 | 72 | 830.0 |
| Driver G | Pre-race | Trace | 116 | 80 | 846.7 |
| | Post-race | 1+ | 88 | 98 | 922.6 |
| Driver H | Pre-race | Trace | 176 | 76 | 1110.3 |
| | Post-race | 2+ | 116 | 100 | 922.6 |
| Driver L | Post-race | Trace | 108 | 82 | 992.4 |

Comment on tilt table tests - Galveston, Texas (figs. 30 through 38).-

Driver I: In the control test there is delay and temporary fall in systolic pressure which in general is maintained by only a slight rise in pulse rate, indicating an appropriate stroke volume response. The diastolic pressure rises slightly, indicating a satisfactory vasomotor response.

The post-race tilt table test shows a dramatic change. The pulse pressure is very narrow due principally to a drop in systolic pressure despite a rise in pulse rate. This must mean a poor stroke volume response. Diastolic pressure falls only slightly which may be taken to indicate a fairly satisfactory vasomotor response.

This distinctive pattern is the only one of its type that we have seen. This subject, age 51, has been quite successful in competitive driving.

Driver H has two tracings, both following races, one of which he won. There was no control test. In general this man maintains his systolic pressure well with only a very slight rise in pulse rate, indicating a good stroke volume. He maintains a good diastolic pressure, indicating satisfactory vasomotor response. This 33-year-old man shows the response that is expected of a man in good condition whose occupation is an airline pilot.

The graphs of Driver E and B are both post-race. No controls are available, but they show a sharp contrast. The older individual, Driver B, age 35, shows good maintenance of systolic pressure with only a small rise in pulse rate. The pulse pressure is well maintained. Diastolic pressure rises slightly during the tilt. Driver E, age 27, the younger individual by 8 years, shows a very narrow pulse pressure with a falling diastolic pressure despite a rise in pulse rate, indicating a poor stroke volume response. His diastolic pressure is well maintained, indicating a satisfactory vasomotor response.

Driver J, a 32-year-old physician, shows a temporary fall in systolic pressure with only a slight rise in pulse rate, indicating perhaps an inadequate stroke volume response. The diastolic pressure rises sharply producing momentarily a very narrow pulse pressure.

Driver F, age 35, shows a small fall in systolic pressure and a slight rise in pulse rate, indicating a reasonably satisfactory combined response of pulse rate and stroke volume. A level diastolic pressure is maintained, indicating a satisfactory vasomotor response. This man is a very competitive driver.

Driver D, age 47, shows well maintained systolic and diastolic pressure with only a small increase in pulse rate, indicating a good stroke volume response and a good vasomotor response although the pulse pressure narrows appreciably during the tilt. This man is believed to have diabetes and hypertension under medical treatment.

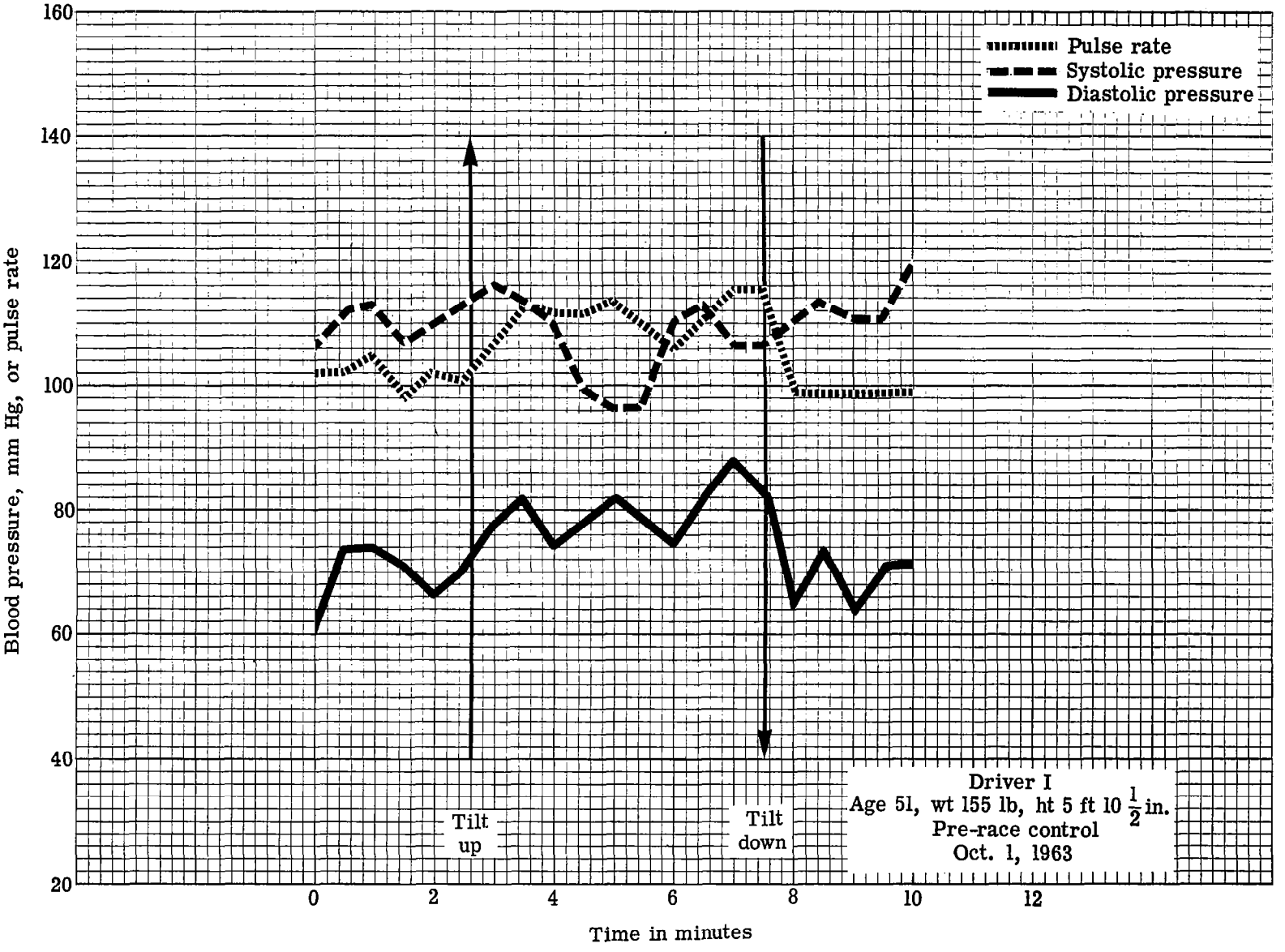



Figure 30. - Pulse rate and blood pressure during tilt-table testing.

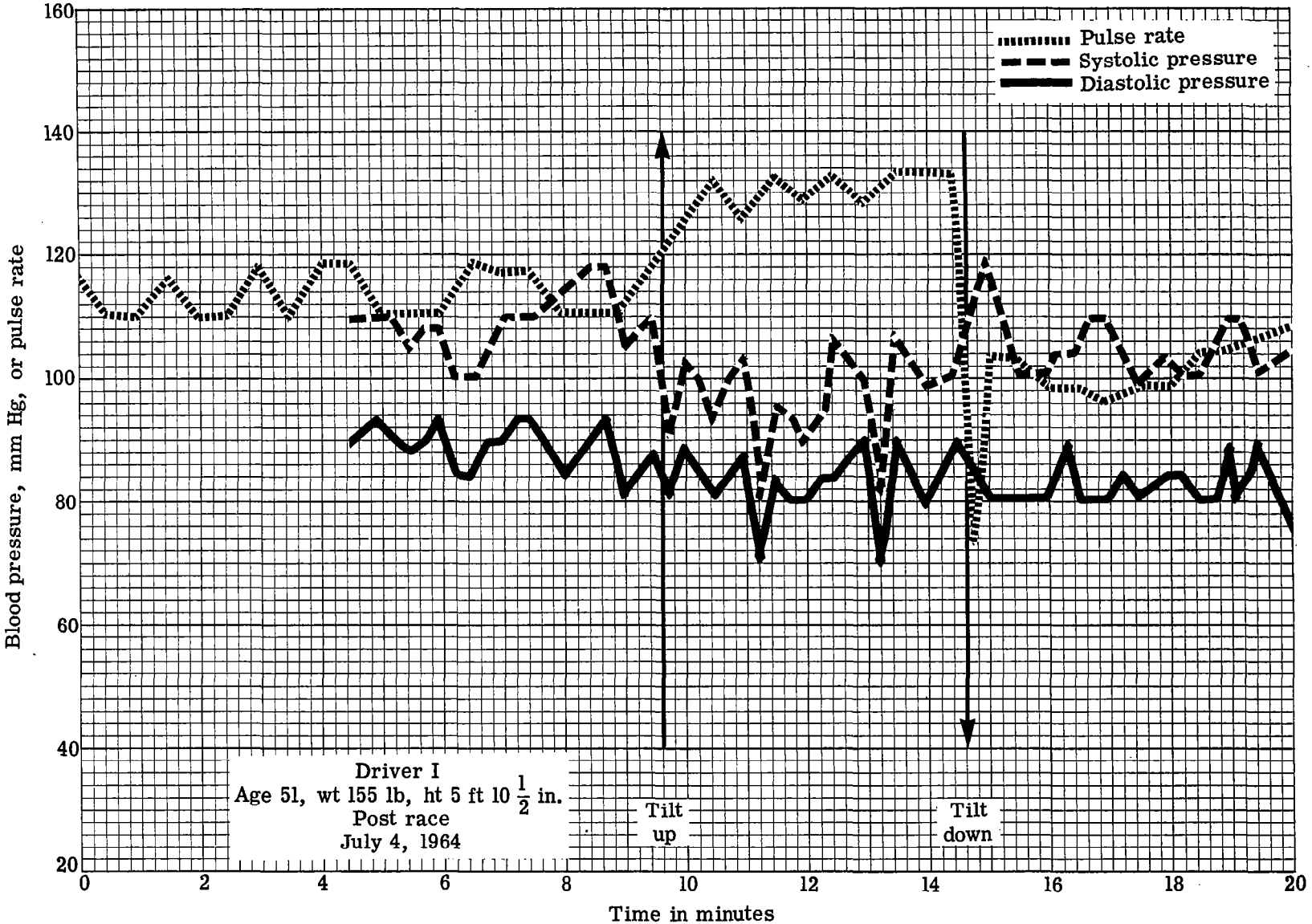



Figure 31. - Pulse rate and blood pressure during tilt-table testing.

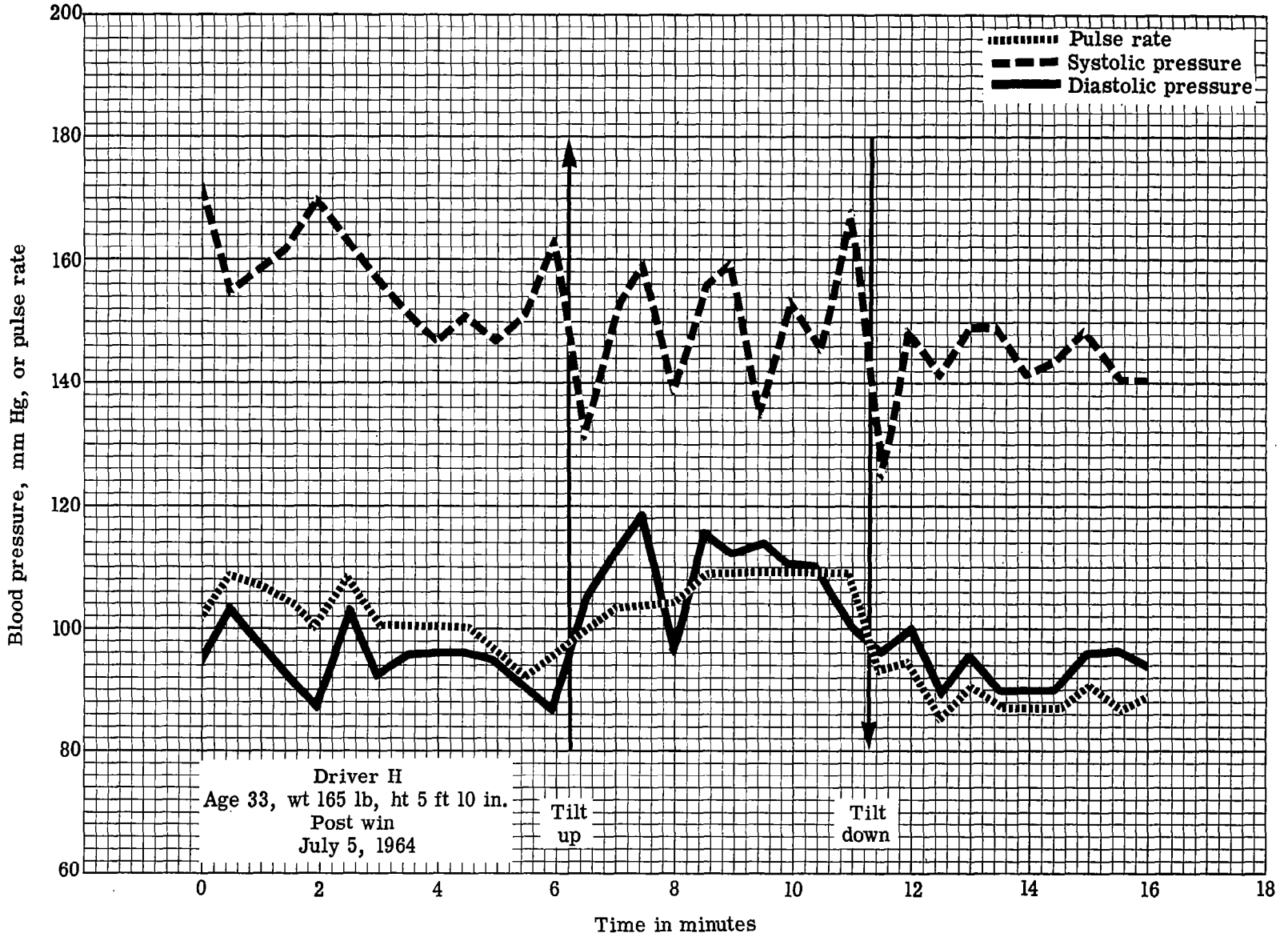



Figure 32. - Pulse rate and blood pressure during tilt-table testing.

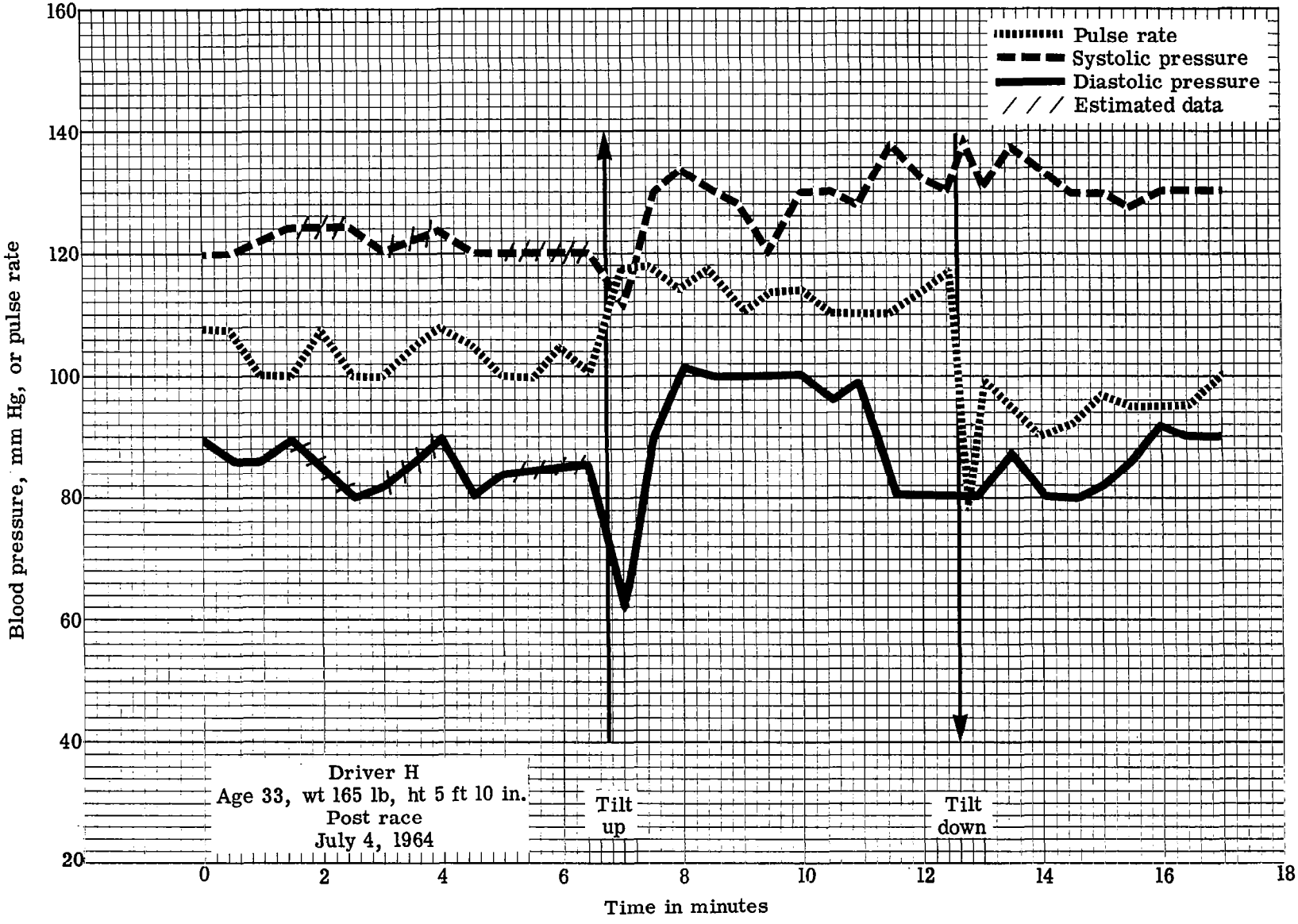



Figure 33. - Pulse rate and blood pressure during tilt-table testing.

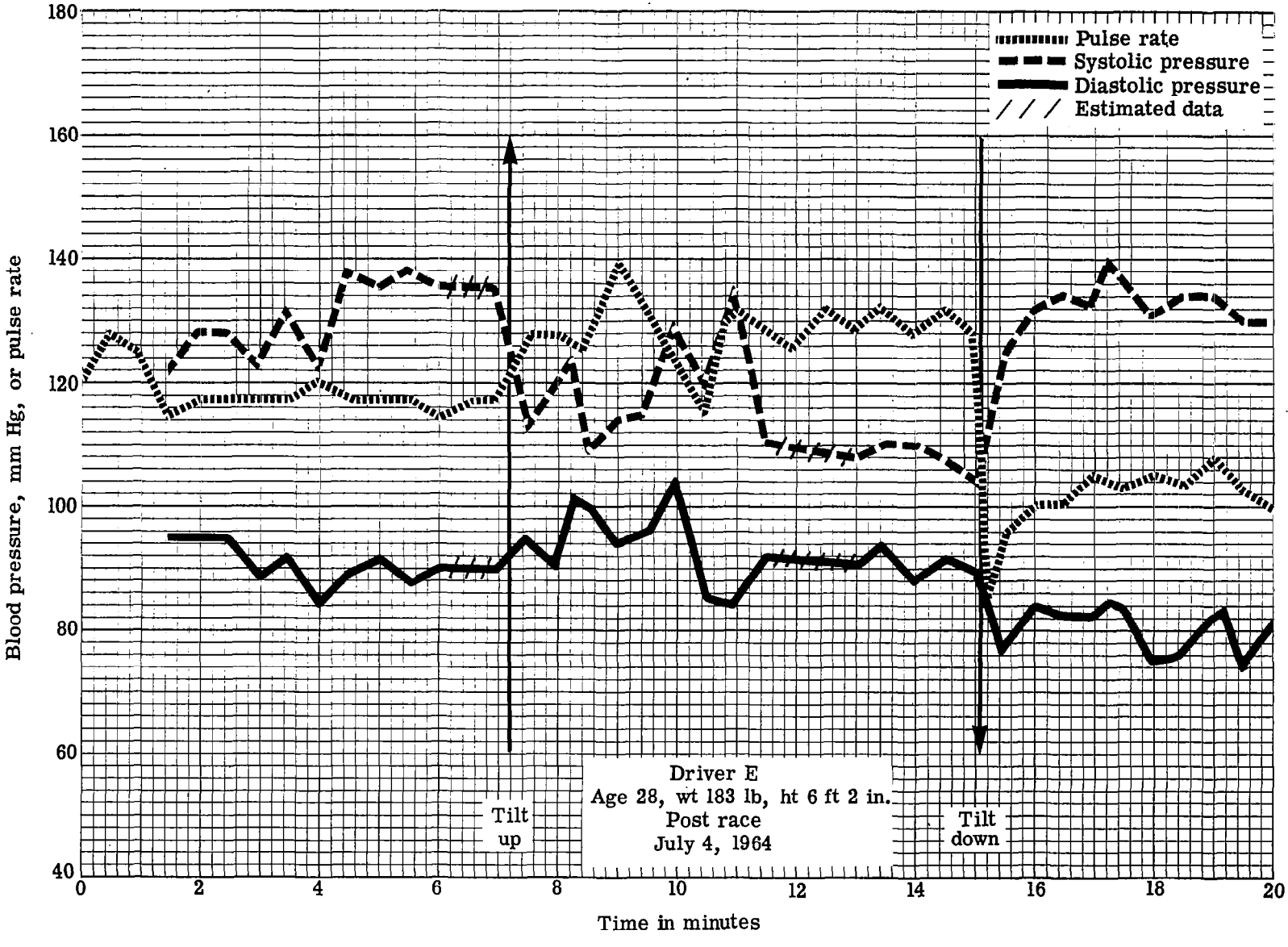



Figure 34. - Pulse rate and blood pressure during tilt-table testing.

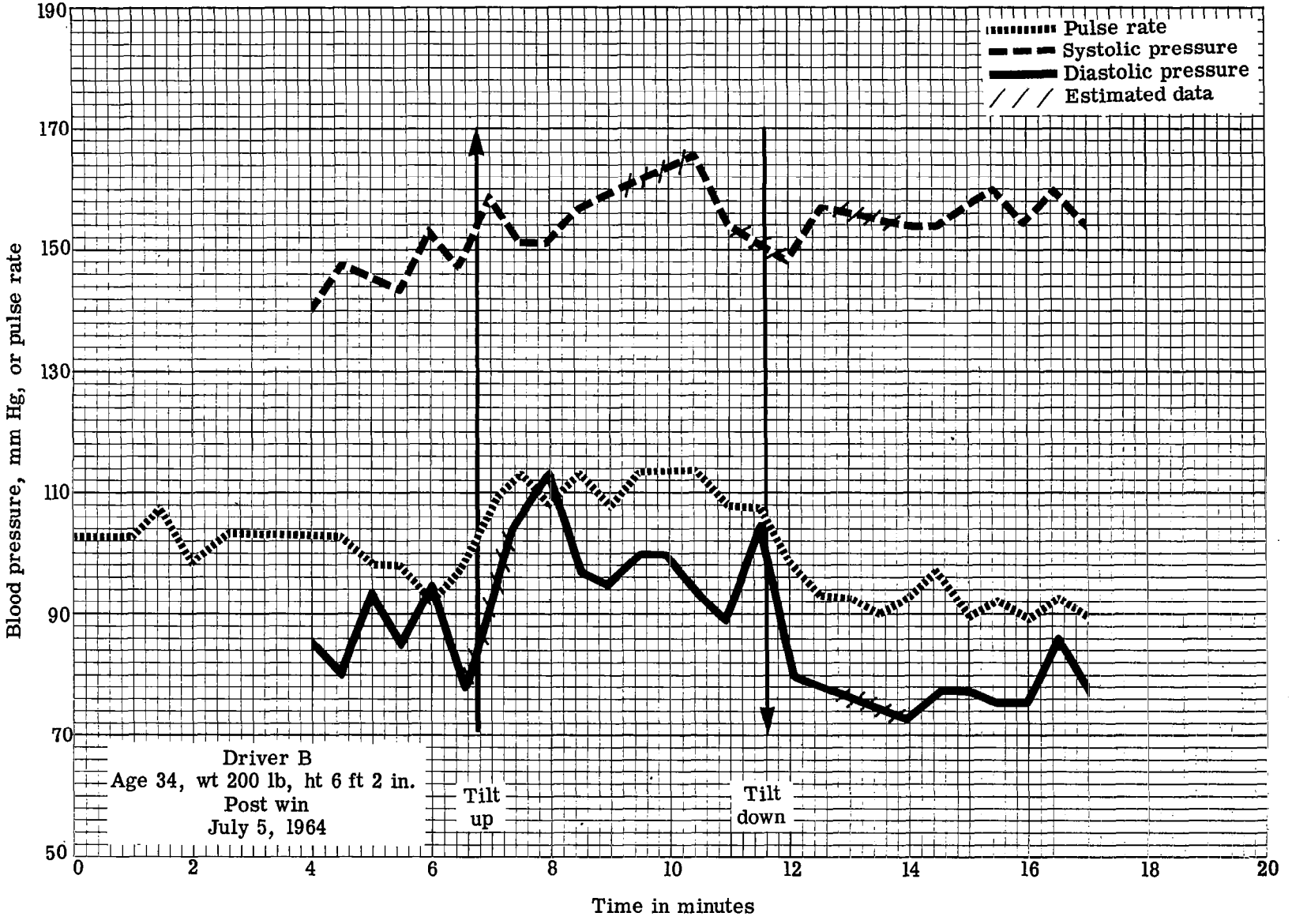



Figure 35. - Pulse rate and blood pressure during tilt-table testing.

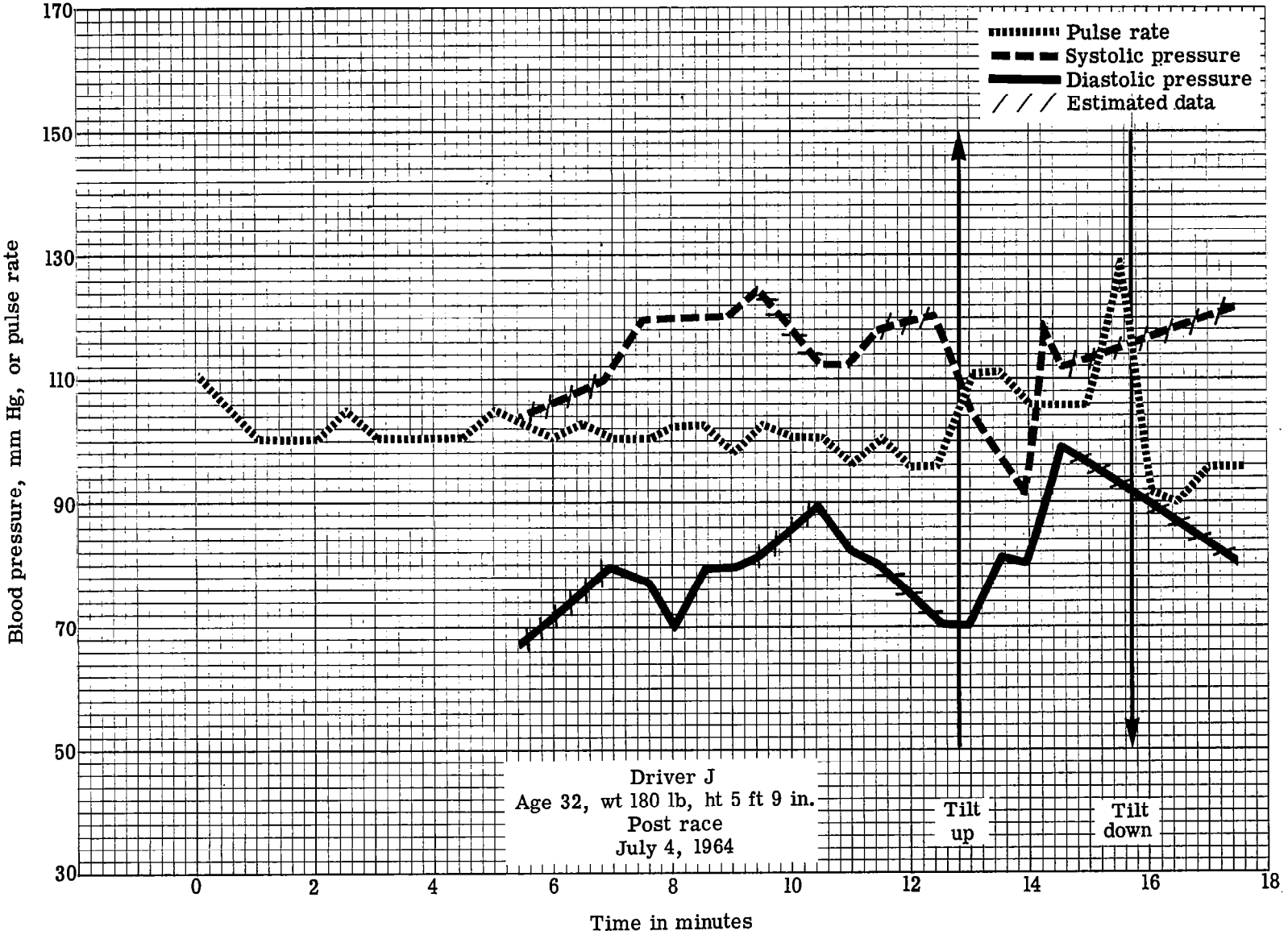



Figure 36. - Pulse rate and blood pressure during tilt-table testing.

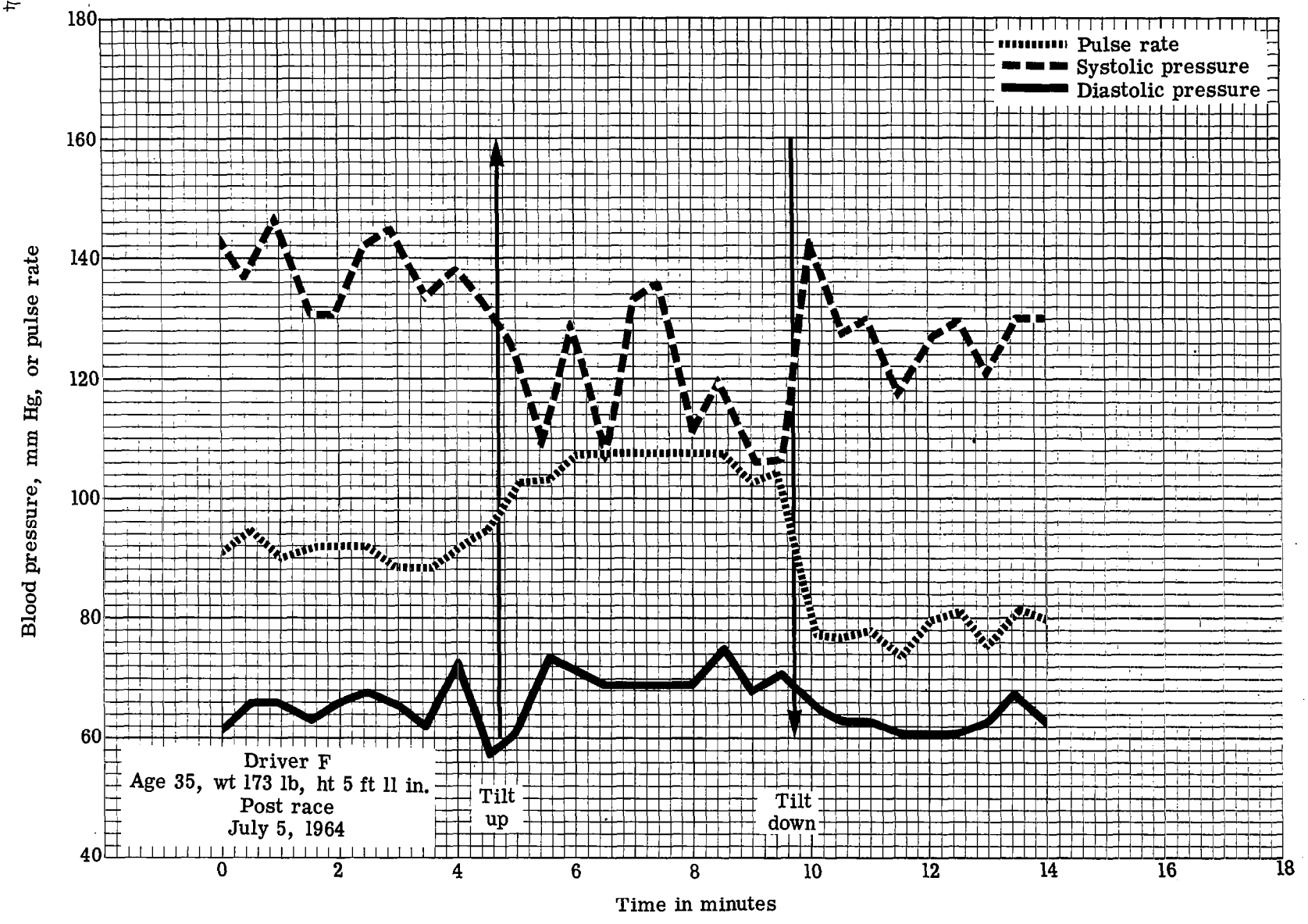



Figure 37. - Pulse rate and blood pressure during tilt-table testing.

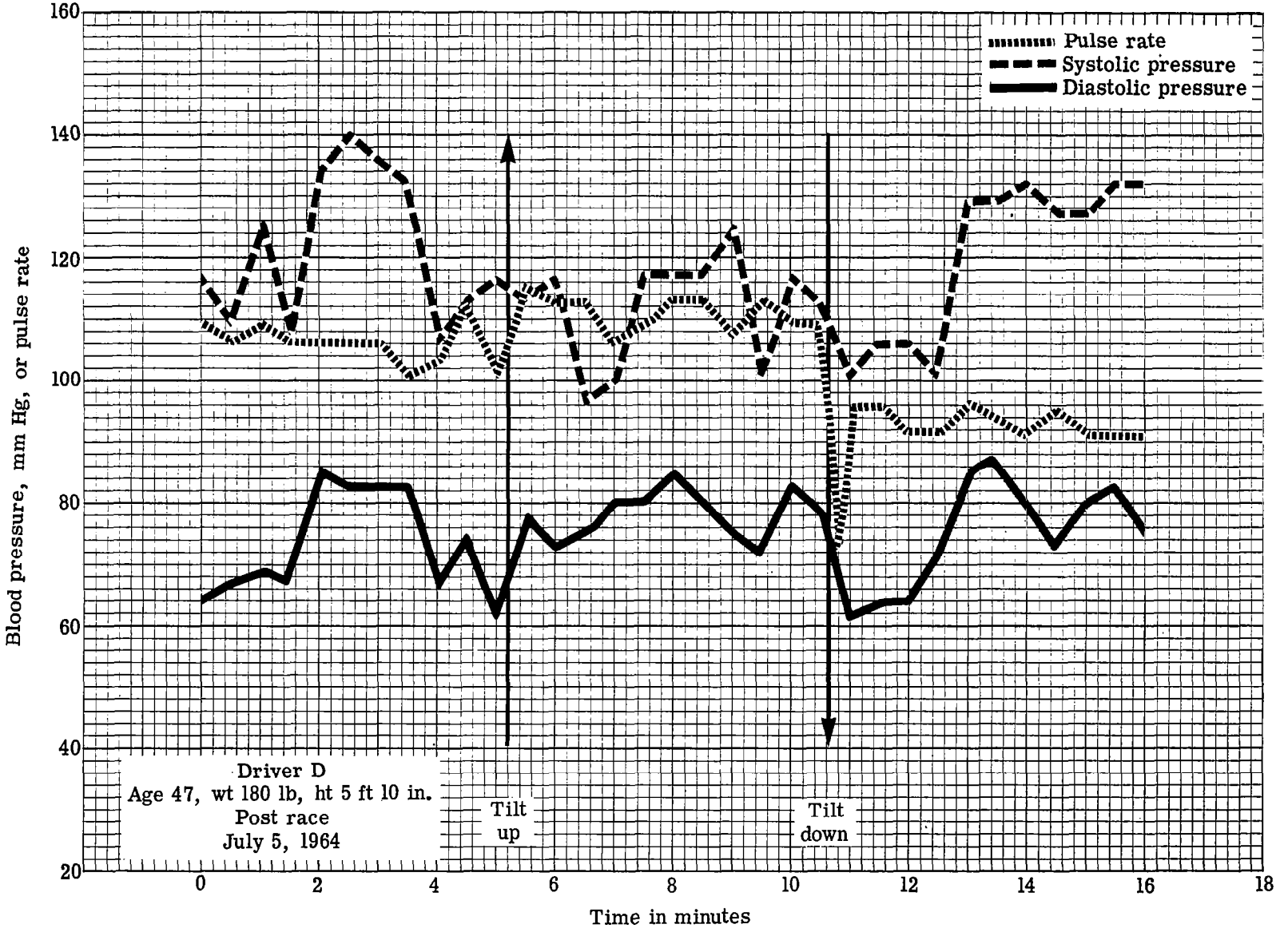



Figure 38. - Pulse rate and blood pressure during tilt-table testing.

Comment on telemetry record - Driver E (fig. 39).- The dominant events during this in-race telemetry record of heart rate are numbered on the graph.

(1) The pulse is approximately level at 110 while the drivers are waiting in their cars on the line for a plane to take off from an adjacent runway.

(2) At the signal "start your engines" the pulse rate rises sharply to 146.

(3) When the flag falls and the race starts there is another sudden rise to 176. Throughout the race this driver held second place with very little fluctuation in the pulse rate between 170 and 186.

(4) At index 4 there was malfunction and his car was suddenly out of competition. Thereafter the pulse rate dropped precipitously (5).

(5) As the car and driver retire to the pits the heart rate promptly falls. A longer period of observation would have been desirable but the driver has usually disconnected before he can be reached to hold him for a record of return to baseline.

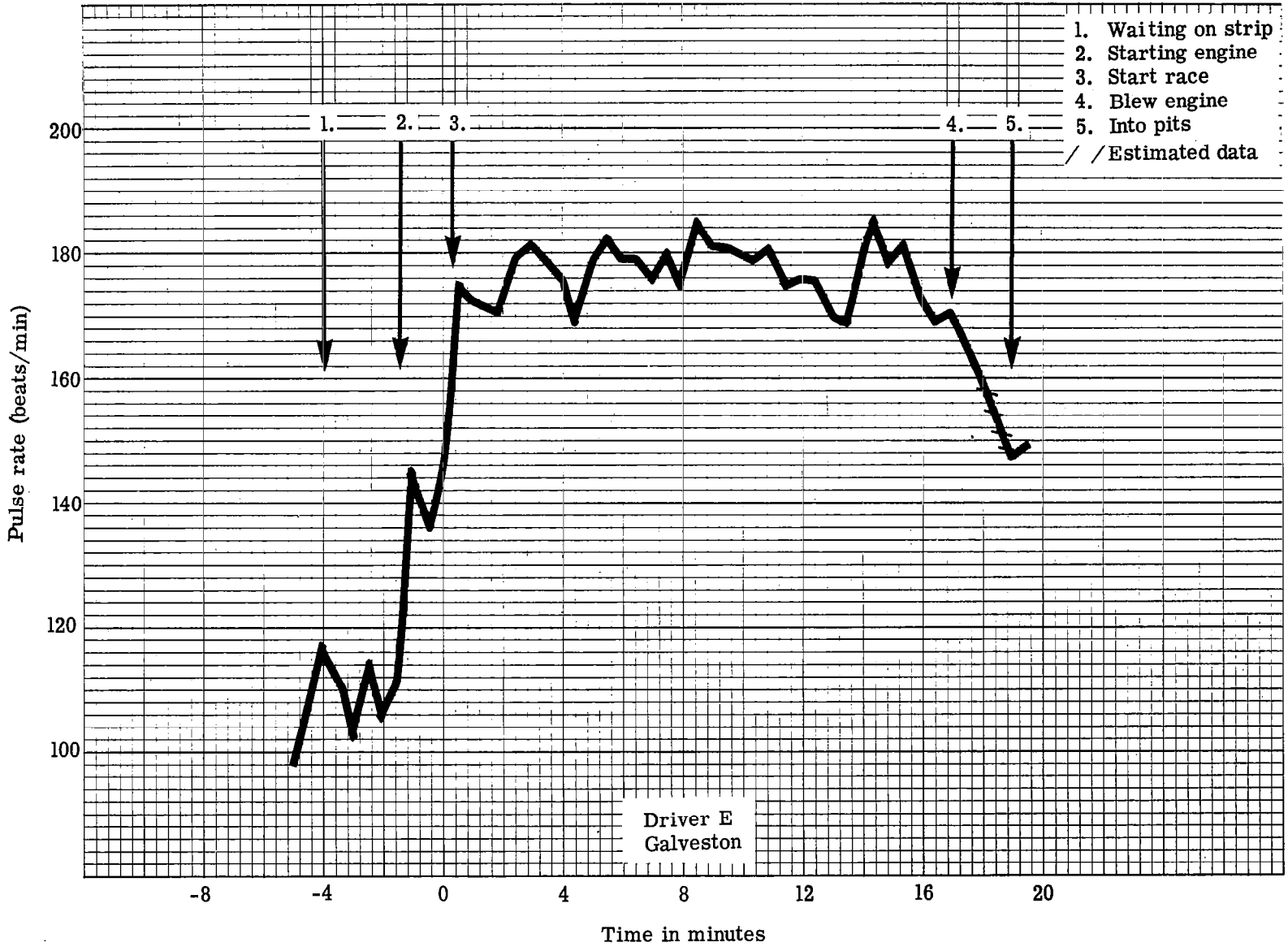



Figure 39. - Pulse rate history during race.

## AUSTIN SPORTS CAR RACE, AUGUST 1964

The course for this event was laid out on the city streets of Austin, around the City Auditorium in Austin. This is unusual for this part of the country; narrow roads, curbs, tight corners contribute some additional hazards and a large enthusiastic crowd tends to heighten excitement. The temperature varied during the day because of cloudiness with a maximum of 101° F but a high humidity because of heavy rain the previous night.

On this occasion the Hamilton Standard Company had provided a "cool suit" for testing. Two individuals (Drivers B and C) each ran two races, one with and one without the suit. One driver (F) accepted the "cool suit" and rejected the medical testing apart from pre- and post-race oral temperatures. This garment is well enough known to require only brief descriptions; it is a two piece set of thermal underwear lined with fine plastic tubing through which ice water is circulated by an electric fuel pump from a reservoir filled with ice water. No regulation of flow or temperature was possible and the initial effect on the wearer was one of shockingly effective cooling.

In-car records of electrocardiograms and temperature were obtained by a 3-channel recorder (PEMCO model 110) and by telemetry equipment provided by NASA. Pre- and post-race tilt table tests were carried out for comparison of the effects of driving with and without the "cool suit."

Driving without the cool suit, drivers showed minimal temperature rises of 1.0°, 0.6°, and 1.2°. With the cool suit the same drivers showed respectively temperatures rises of 2.0°, 1.4°, and 0°.

The number of drivers and the minor heat stress provided in the day preclude a useful impression of the effect on body temperature. However two drivers showed a greater rise in body temperature with the cool suit than without. It might be anticipated that for short periods, abrupt cooling of body surface would result in a superficial constriction and conservation of body heat with a temporary rise in internal temperature.

The blood pressure readings before and after racing, with and without the cool suit, do not provide an adequate basis for comment, but interesting correlations with temperature and tilt table testing should be found on future observations.

TABLE 14.- VITAL STATISTICS OF DRIVERS IN SPORTS CAR RACES AT AUSTIN, TEXAS, IN AUGUST 1964

| Driver | Physical description | | | Occupation | Marital status and no. of children | Education | Racing experience | | Rest prior to race |
|---|---|---|---|---|---|---|---|---|---|
| | Weight | Height | Age | | | | Type | No. of years | |
| Driver A | | 6 ft 2 in. | 34 | Owner of Culligan Water Company | Yes 1 child | 4 yr college | Sports | 8 | |
| Driver B | 170 | 5 ft 9 in. | 51 | M.D. | Yes 3 children | M.D., L.L.B. | Sports | 3 | 6 hr sleep |
| Driver C | 180 | 5 ft 11 in. | 29 | Electrical draftsman | Yes 4 children | Trade school some college | Sports | 1 | |
| Driver D | 180 | 5 ft 10 in. | 47 | Retired Colonel | Yes 2 children | West Point graduate | Sports | 1 | 9 hr sleep |
| Driver E | 200 | 6 ft 2 in. | 43 | Personnel Director | Yes 1 child | High school | Sports | 1 | 5 1/2 hr sleep |

TABLE 15.- PRE- AND POST-RACE TEMPERATURE READINGS OF DRIVERS

IN SPORTS CAR RACES AT AUSTIN, TEXAS, IN 1964

| Driver | Temperatures | | Blood pressure | |
|---|---|---|---|---|
| | Pre-race, °F | Post-race, °F | Pre-race | Post-race |
| Driver A | | | | |
| With ice-cooled suit | 99.8 | 101.2 | 124/96 | 130/78 |
| Without cooled suit | 99.6 | 100.2 | 110/78 | 130/78 |
| Driver B | | | | |
| With ice-cooled suit | 98.0 | 99.8 | --- | 120/70 |
| Without cooled suit | 99.0 | 100.2 | 22/70 | 142/75 |
| Driver F | | | | |
| With ice-cooled suit | 98.8 | 100.0 | --- | --- |
| Without cooled suit | 98.0 | 99.0 | --- | --- |

TABLE 16.- WEIGHT-CHANGE STUDIES CONDUCTED ON DRIVERS IN SPORTS CAR RACES AT AUSTIN, TEXAS, IN AUGUST 1964

| Driver | Pre-race weight | Post-race weight | Loss in lb | Percent of weight loss |
|---|---|---|---|---|
| Driver A | | | | |
| With cool suit | 201 | 198.5 | 2.5 | 1.2 |
| Without cool suit | | 198 | --- | |
| Driver B | | | | |
| With cool suit | --- | 169 | --- | |
| Without cool suit | 173 | 169 | 4 | 2.3 |
| Driver C | | | | |
| Without cool suit | 180.5 | 179.5 | 1 | .6 |
| Driver D | | | | |
| Without cool suit | 177 | 175 | 2 | 1.1 |

TABLE 17.- BLOOD-CHEMISTRY STUDIES CONDUCTED ON DRIVERS

IN SPORTS CAR RACES AT AUSTIN, TEXAS, IN AUGUST 1964

| Driver | Time | Glucose | Cholesterol | Uric acid | Creatinine | Chloride | Phosphorus |
|---|---|---|---|---|---|---|---|
| Driver A | Pre-race | 156 | 185 | 5.3 | 4.42 | 124 | 3.5 |
| With cool suit | Post-race 6 | 107 | 118 | 5.7 | 2.00 | 125 | 2.2 |
| Without cool suit | Post-race 12 | 105 | 147 | 6.4 | 1.96 | --- | 3.8 |
| Driver B | Pre-race | 121 | 196 | 4.5 | 1.28 | .24 | 3.5 |
| With cool suit | Post-race 10 | 91 | 200 | 6.4 | 1.92 | 129 | 2.0 |
| Without cool suit | Post-race 4 | 123 | 239 | 4.4 | 2.95 | 126 | 1.6 |
| Driver D | Pre-race | 123 | 235 | 6.8 | 1.79 | 125 | 2.4 |
| | Post-race | 144 | 147 | 7.3 | 1.79 | 128 | 1.7 |

Comments on figures 41 through 46.-

Glucose (fig. 41): The three drivers showed pre-race blood sugar levels at or above the usual upper limits of normal fasting values. However these were taken at the site of the race, after breakfast, and after varying exertions. Post-race, one driver (B) shows the slight rise that is commonly seen with stress of short duration, then a fall after his second race. A second driver (A) showed a fall to fasting levels after his first race (with cool suit) which is closely maintained after his second race (without cool suit). A third driver (D) (who did not wear a cool suit) showed a rise from 123 to 144 mgm percent over a period of 6 hours spanning two races.

These values demonstrate only the labile nature of serum glucose levels under the effect of stress and exertion and indicate that useful information might be gained from several samples closely coordinated with glucose intake and hematocrit estimations.

Cholesterol (fig. 42): The cholesterol values stayed within normal limits throughout the period of observation with only small changes of inconstant direction.

Uric acid (fig. 43): The uric acid concentration increased throughout the day in all three drivers. Driver B did not have an increase during his first race but did show a 2.0 mg percent increase after the last race. This was the maximum increase observed.

Creatinine (fig. 44): Three subjects showed the possible variations, one rose, one fell, and one remained level. The only comment is that the blood chemistry might be of value if the baseline observations were greatly extended before and after the period of stress.

Chlorides (fig. 45): The chlorides showed very slight increases in all three drivers. Pre-race values were all elevated between 16 and 18 percent. A slight increase was continuous throughout the day reaching 22 percent above normal.

Phosphorus (fig. 46): The pattern of changes in the phosphorus concentration varied from individual to individual. All values were below normal except the pre-race in one driver and after the final race in another. No explanation for the increase in concentration observed in Driver A can be offered.

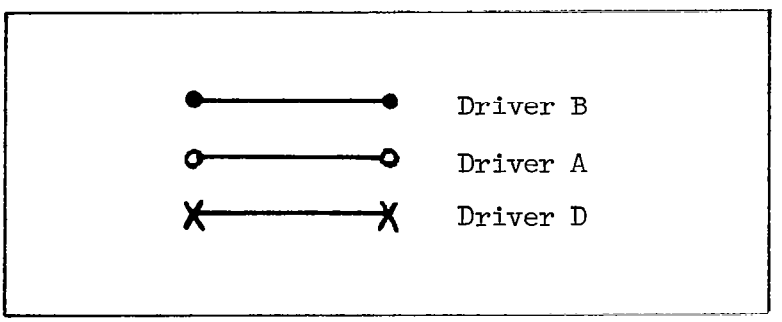



Figure 40.- Legends for blood chemistries,
Alamo Regional Race,
Austin, Texas,
August 16, 1964.

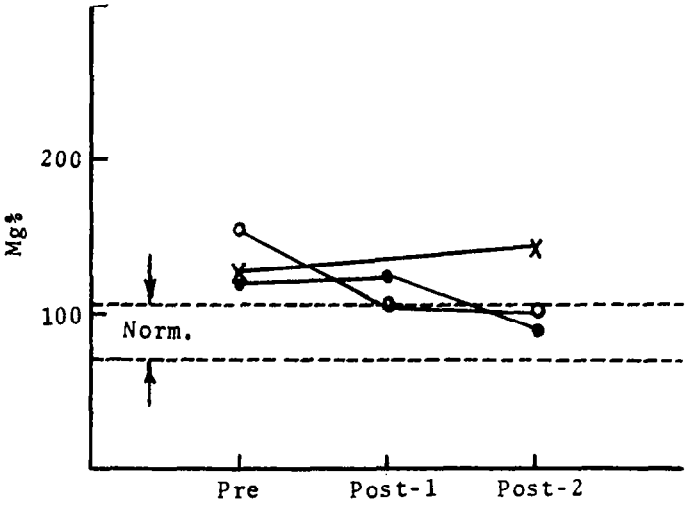



Figure 41. - Blood chemistry, glucose, Alamo Regional Race, August 16, 1964.

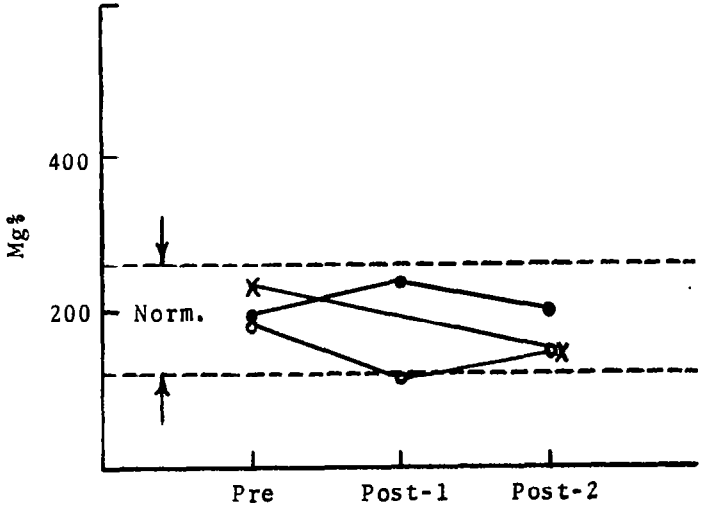



Figure 42. - Blood chemistry, cholesterol, Alamo Regional Race, August 16, 1964.

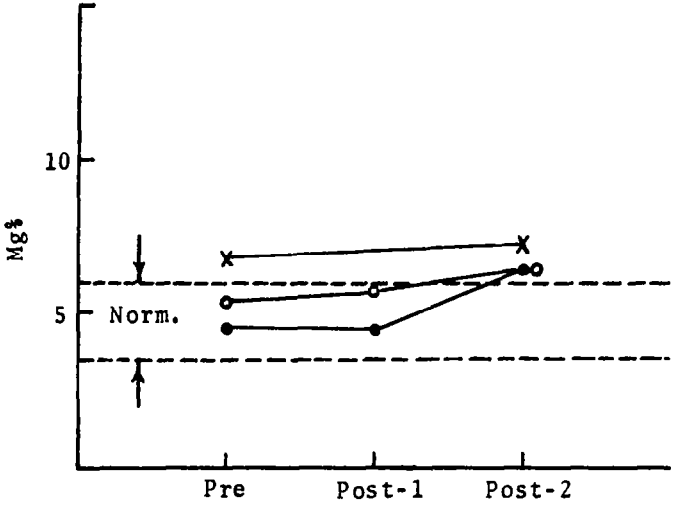



Figure 43. - Blood chemistry, uric acid, Alamo Regional Race, August 16, 1964.

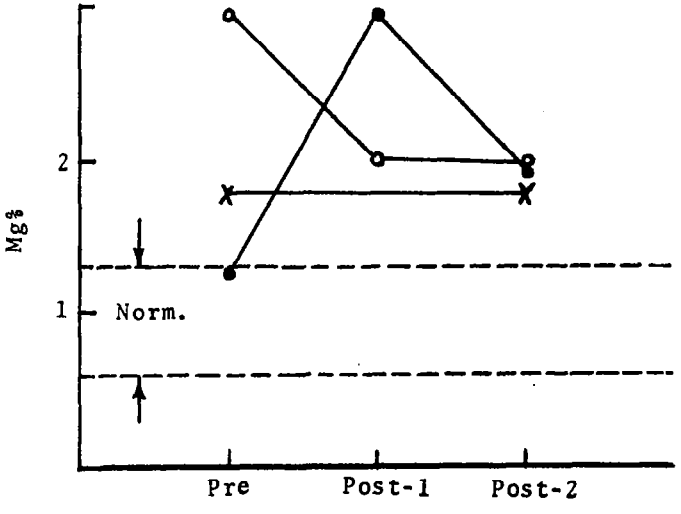



Figure 44. - Blood chemistry, creatinine, Alamo Regional Race, August 16, 1964.

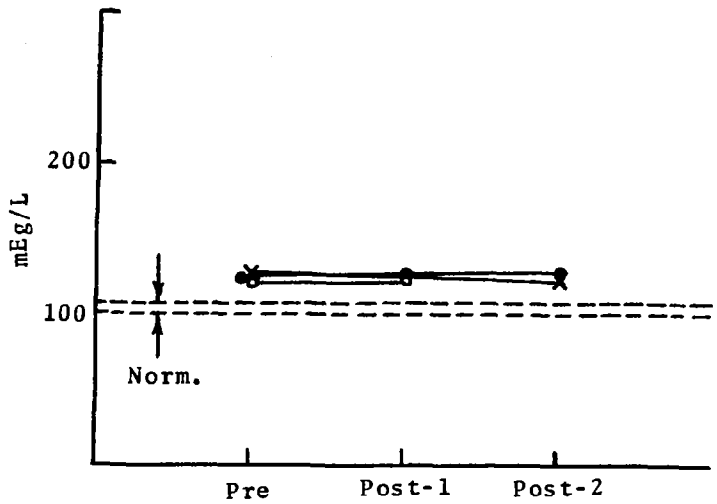



Figure 45. - Blood chemistry, chlorides, Alamo Regional Race, August 16, 1964.

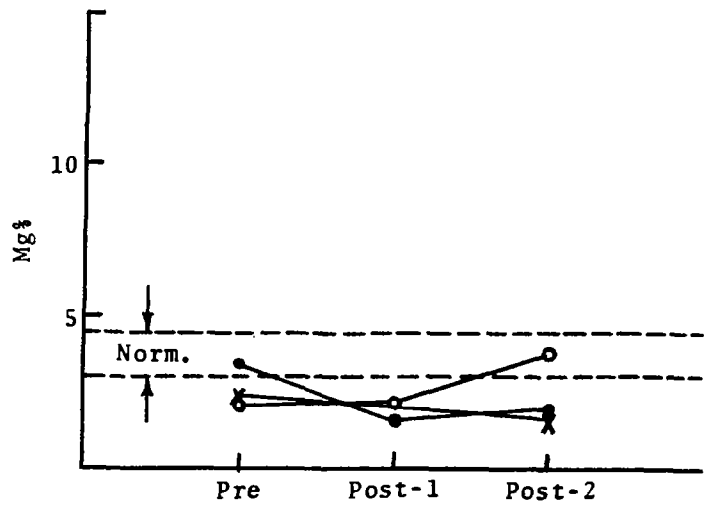



Figure 46. - Blood chemistry, phosphorus, Alamo Regional Race, August 16, 1964.

Urine excretion and chemistries - Austin (table 18).- Urine samples were collected on four drivers. Two were followed throughout the day for total excretion, excretion rate, glucose, and acetone concentrations. The other two had single specimens collected. A urine sample was collected on each driver and studied immediately on his return to the testing area after active racing.

One driver, A, was unable to relate the time of voiding prior to the first sample collected; therefore only the late morning and afternoon specimens were considered in determining excretion rates. Over a period of 6.6 hours, he excreted 160 ml of urine, an average excretion rate of 24 ml per hour. During the same time of day, but over a 7.8-hour period, Driver B excreted 156 ml, an average excretion rate of 20 ml per hour. This was a decrease from the 47-ml-per-hour excretion rate observed in his first collection period. Similar low excretion rates are indicated by the other two drivers who excreted 43 and 47 ml, respectively, over a period which was probably greater than 2 hours.

The decrease in urine excretion rates correlates well with an expected decrease in ERPF and GFR along with an increased reabsorption. This is probably in an attempt to conserve body water during periods of excessive loss of water by perspiration.

Sugar and acetone concentrations were determined on each urine sample collected. Driver A had an increase in urine sugar concentration with no increase in acetone. Driver B exhibited an increase in urine acetone with no corresponding increase in urine sugar. A moderate amount of acetone was present in the post-race urine of one of the other drivers. The other was essentially negative although in a previous race he had a significant increase in urine glucose and is a known diabetic.

Although glucose and acetone are not normally present in the urine, sufficient elevations in blood glucose concentrations were present, even prior to racing, to have some "spilled" into the urine. This "spilling" of glucose and acetone into the urine prior to racing is evident by small amounts of each being detected in the first samples of urine collected. The result of body water conservation by the kidneys is the production of a more concentrated urine. The concentrating effect may have been sufficient to produce increased urine sugar and acetone concentrations.

TABLE 18.- URINE VOLUME AND CHEMISTRY STUDIES CONDUCTED ON DRIVERS IN SPORTS CAR RACES AT AUSTIN, TEXAS, IN AUGUST 1964

| Driver | Time, hr | Volume, ml | Volume/hr, ml | Glucose | Acetone |
|---|---|---|---|---|---|
| Driver A | -11:20 | 54 | -- | Trace | Small |
| | 11:20-3:35 | 110 | 26 | Trace | Small |
| | 3:35-5:55 | 50 | 21 | 1+ | Small |
| Totals[a]: | 6.6 | 160 | Average 24 | | |
| Driver B | 6:30-9:40 | 150 | 47 | Trace | Small |
| | 9:40-2:50 | 98 | 19 | Trace | Small |
| | 2:50-5:30 | 58 | 22 | Trace | Moderate |
| Totals: | 11 | 306 | Average 28 | | |
| Driver C | -2:30 | 47 | -- | Trace | Moderate |
| Driver D | -10:55 | 43 | -- | Negative | Small |

[a]The total time, volume, and average excretion rate is taken from the last two periods.

Comment on tilt table testing - Austin, Texas (figs. 47 through 54).-

Figures 47 and 48: These two records compare the cardiovascular and vasomotor response after racing, with and without the cool suit.

Without the cool suit, the pre-tilt resting pulse rate fell from 175 to 130. Then, on tilting up, it climbed to 174. This maintained the systolic pressure at the level of 100 except for a momentary fall to 80. Assuming some increase in cardiac output to maintain systolic pressure, this is due, and possibly largely, to the increase in heart rate rather than an increase in stroke volume. On tilting back to horizontal, a rise in systolic pressure from 96 to 135 coincides with a fall in pulse rate from 174 to 150, suggestive of an improved stroke volume.

Diastolic pressure, pre-tilt, was less than 40, and during the tilt rose as high as 62. The increase is small but firm, and on tilting back to horizontal there is a decrease in diastolic pressure back to a low of 30.

The principal evident response to the severe heat stress is the heart rate ranging up to 180. Nevertheless systolic, diastolic, and pulse pressure are well maintained by this response together with an effective peripheral vasomotor response.

By comparison, the tilt table test after racing, while wearing the cool suit, is much closer to the pattern of a resting control. On tilting up, a pulse rate increase from 105 to 140 maintains a systolic pressure level in the range of 100 to 120. Diastolic pressure rises during the tilt from a low of 48 to a high of 84 and promptly falls on resuming horizontal.

Figures 49 and 50 compare Driver A's tilt table response with and without the use of the cool suit. The difference is striking but in a different pattern than that of B in graphs 1 and 2. Pulse rate without the cool suit rises from a resting 95 to 114 during the tilt and falls as promptly to previous levels on tilting down. This maintains a systolic pressure between 150 and 170 except for a momentary dip to 122. The small rise in pulse rate suggests an important increase in stroke volume.

The resting diastolic pressure rises from 70 to the level of 100 with a peak at 132.

The principal feature is high systolic and diastolic pressure with a rather narrow pulse pressure and relatively slow pulse rate. With the ice-cooled suit all elements are remarkably level. Pulse rate is level at 120 to 140, appreciably higher than without the cool suit. Both systolic and diastolic pressure are much more stable and pulse pressure is particularly wide.

Both Drivers B and A demonstrated a more stable tilt table response after wearing the cool suit, than after racing without it.

Figures 51 and 52 compare Driver D in tilt table tests, after practice (lacking a resting control), and after racing.

The post-race tilt table test shows a comparable pulse rate in the vicinity of 140 maintaining a systolic pressure of 150 to 160, as compared to a systolic pressure of about 120 at lesser stress, post-practice. The post-race diastolic pressure shows a swinging response with a rise from 92 to 140 down to 104 and back to 132 before tilting down and resuming horizontal diastolic pressure at approximately 80.

The post-practice systolic, diastolic, and pulse pressure records are much more stable.

Figure 53: In a single tilt table test, post-practice, Driver C (age 27) shows a rise in pulse rate from 114 to 180. This maintains systolic pressure at 120 to 140 and should mean very little change in stroke volume. A strong rise in diastolic pressure from 64 to a peak of 124 indicates a strong peripheral vasomotor response and is responsible for a very narrow pulse pressure during tilt.

Figure 54: The single tilt table test of Driver E, done as a pre-race control, is unusual in the rise of systolic pressure from 120 to 148, and of diastolic pressure from 90 to a maximum of 118 during the tilt, with a pulse rate which levels off at 102. This should indicate that an increase in cardiac output during tilt is dominantly by stroke volume. Driver E did not finish this race and his response to stress must be obtained on another occasion.

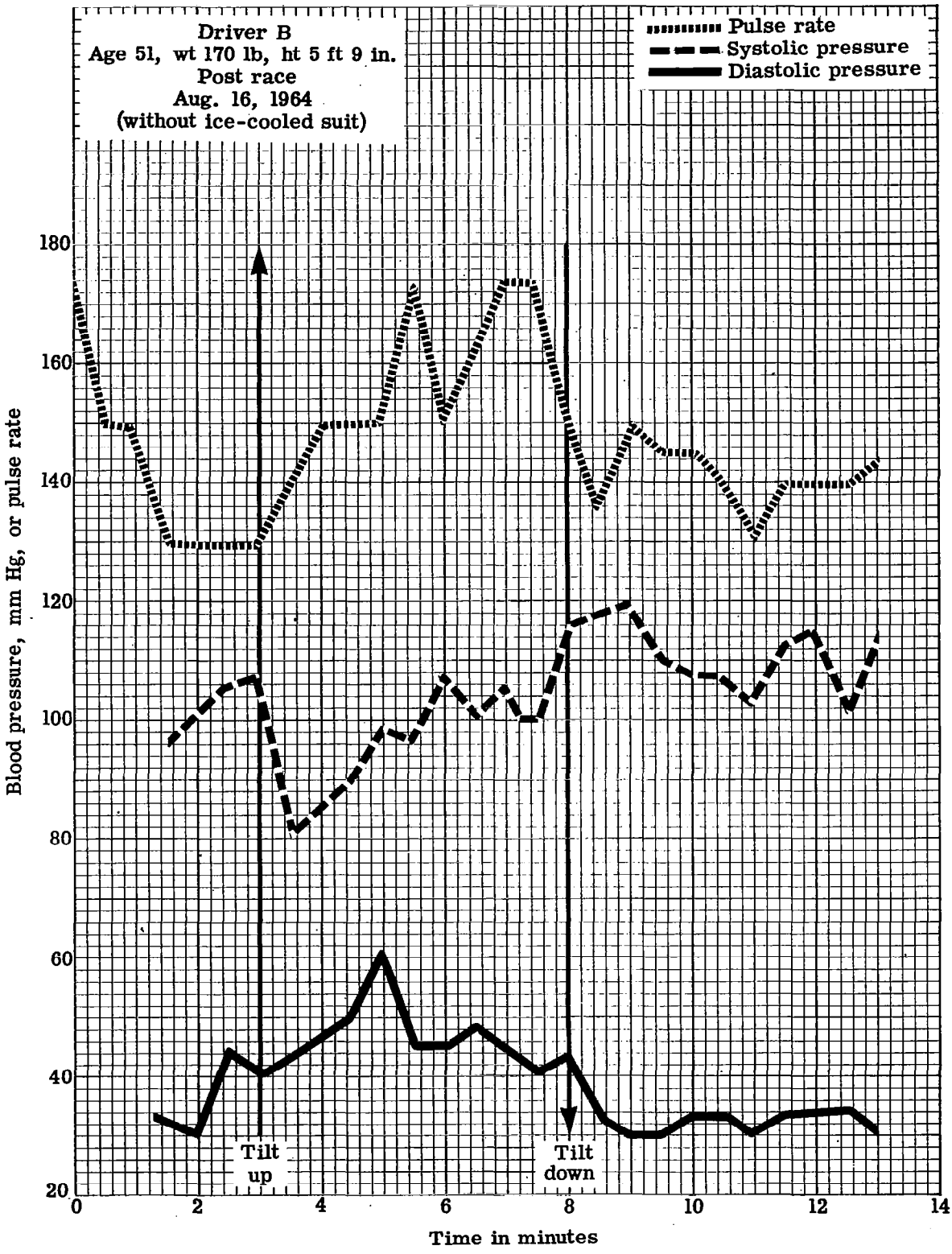



Figure 47. - Pulse rate and blood pressure during tilt-table testing.

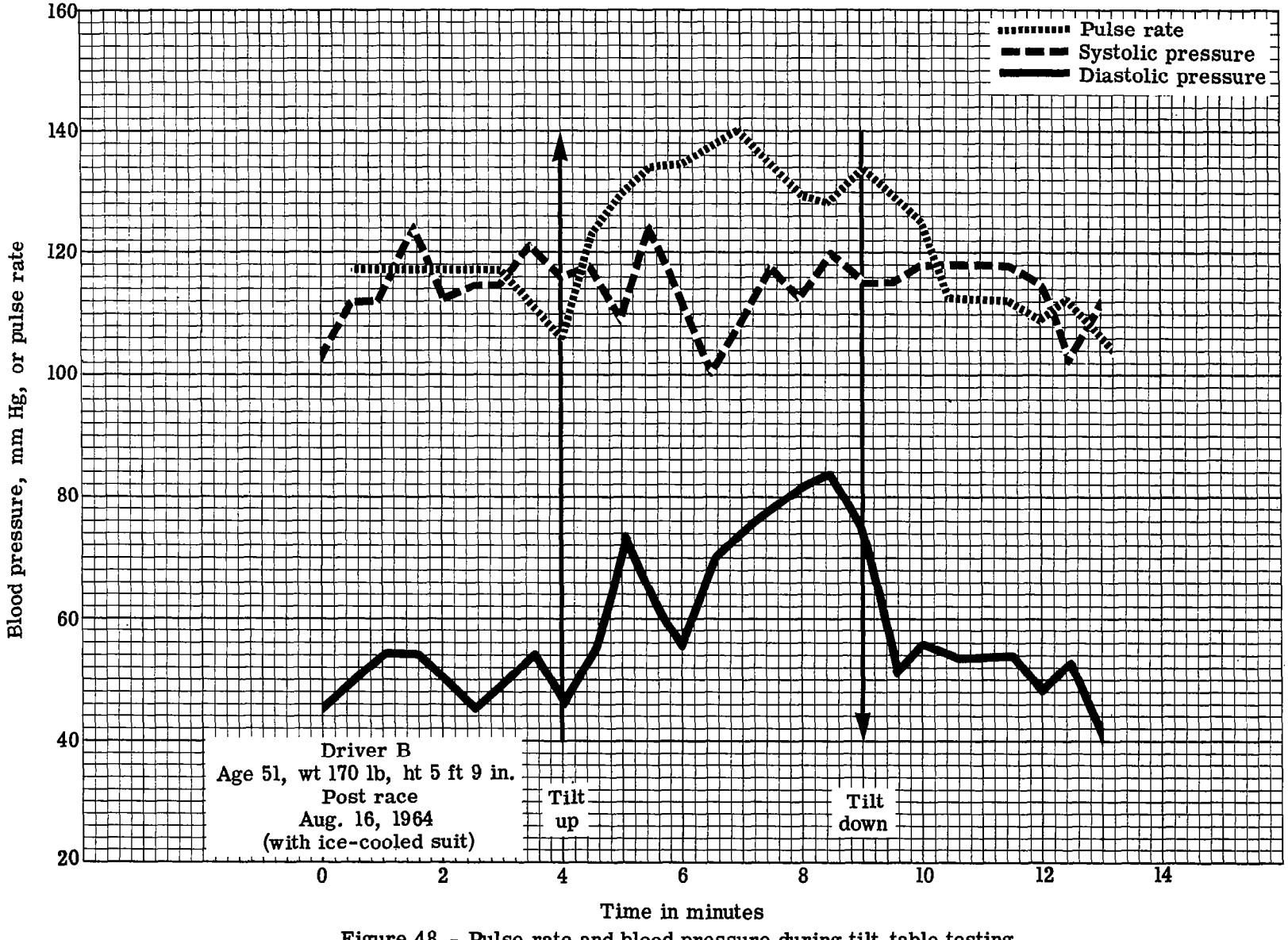



Figure 48. - Pulse rate and blood pressure during tilt-table testing.

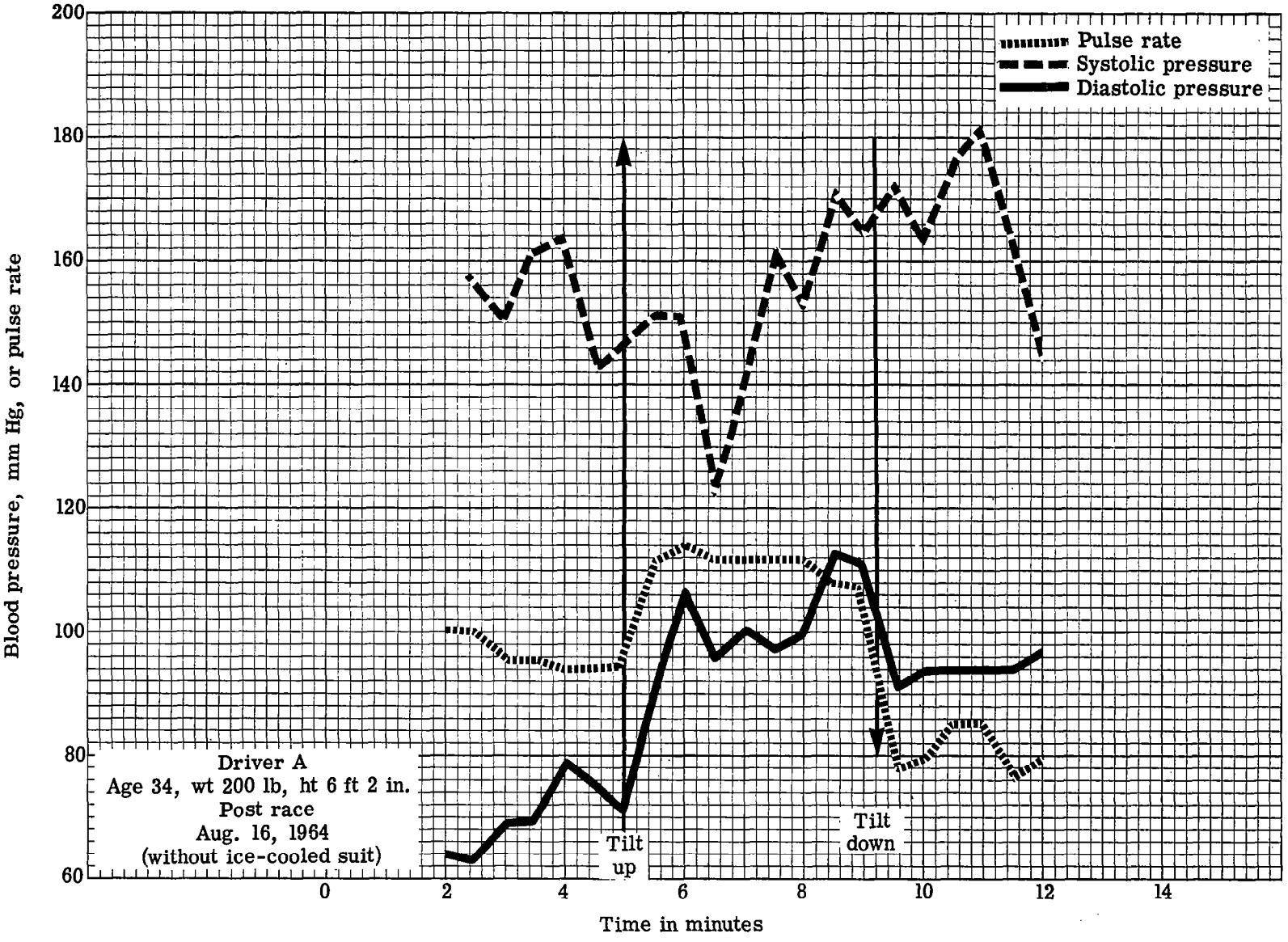



Figure 49. - Pulse rate and blood pressure during tilt-table testing.

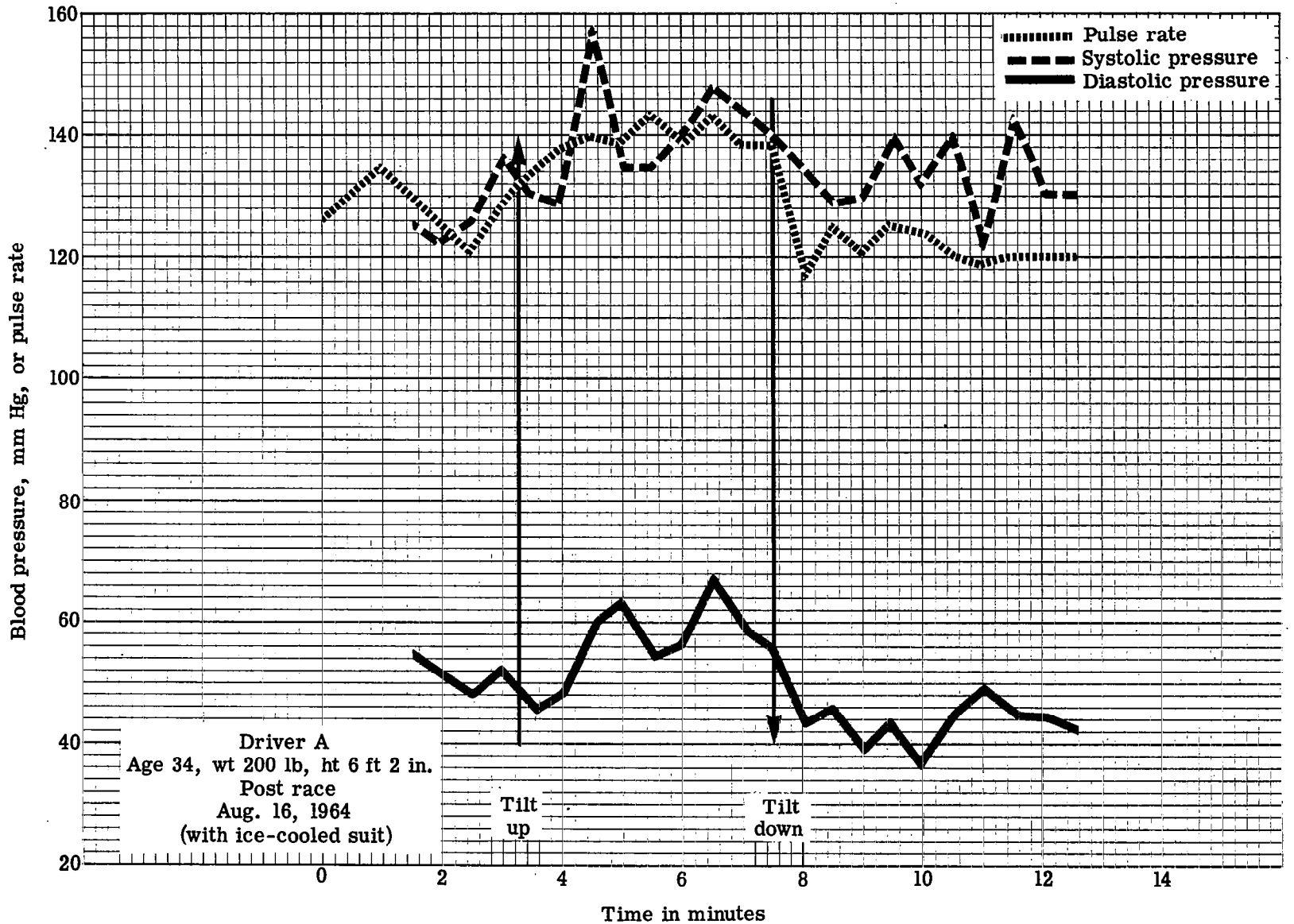



Figure 50. - Pulse rate and blood pressure during tilt-table testing.

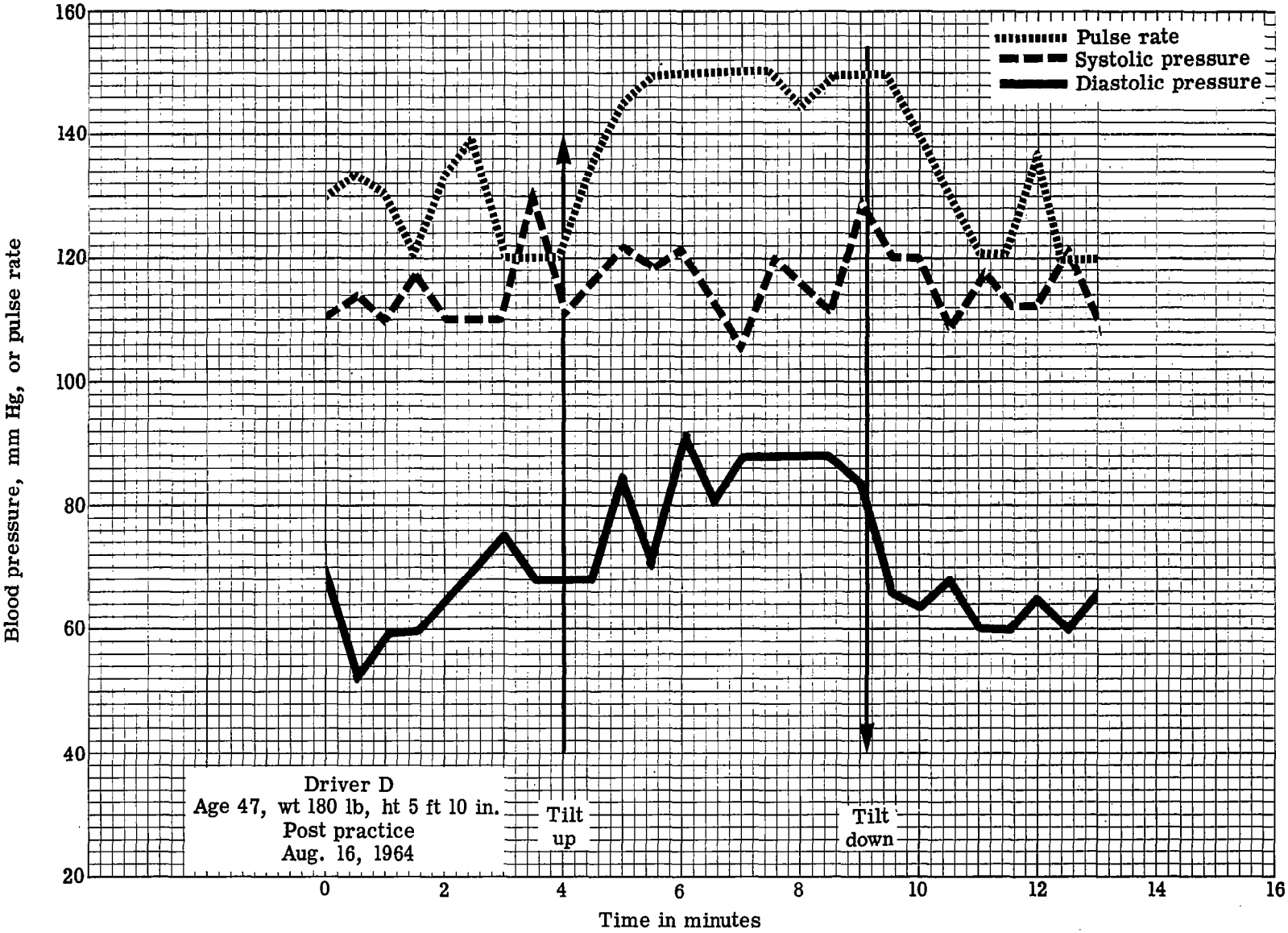



Figure 51. - Pulse rate and blood pressure during tilt-table testing.

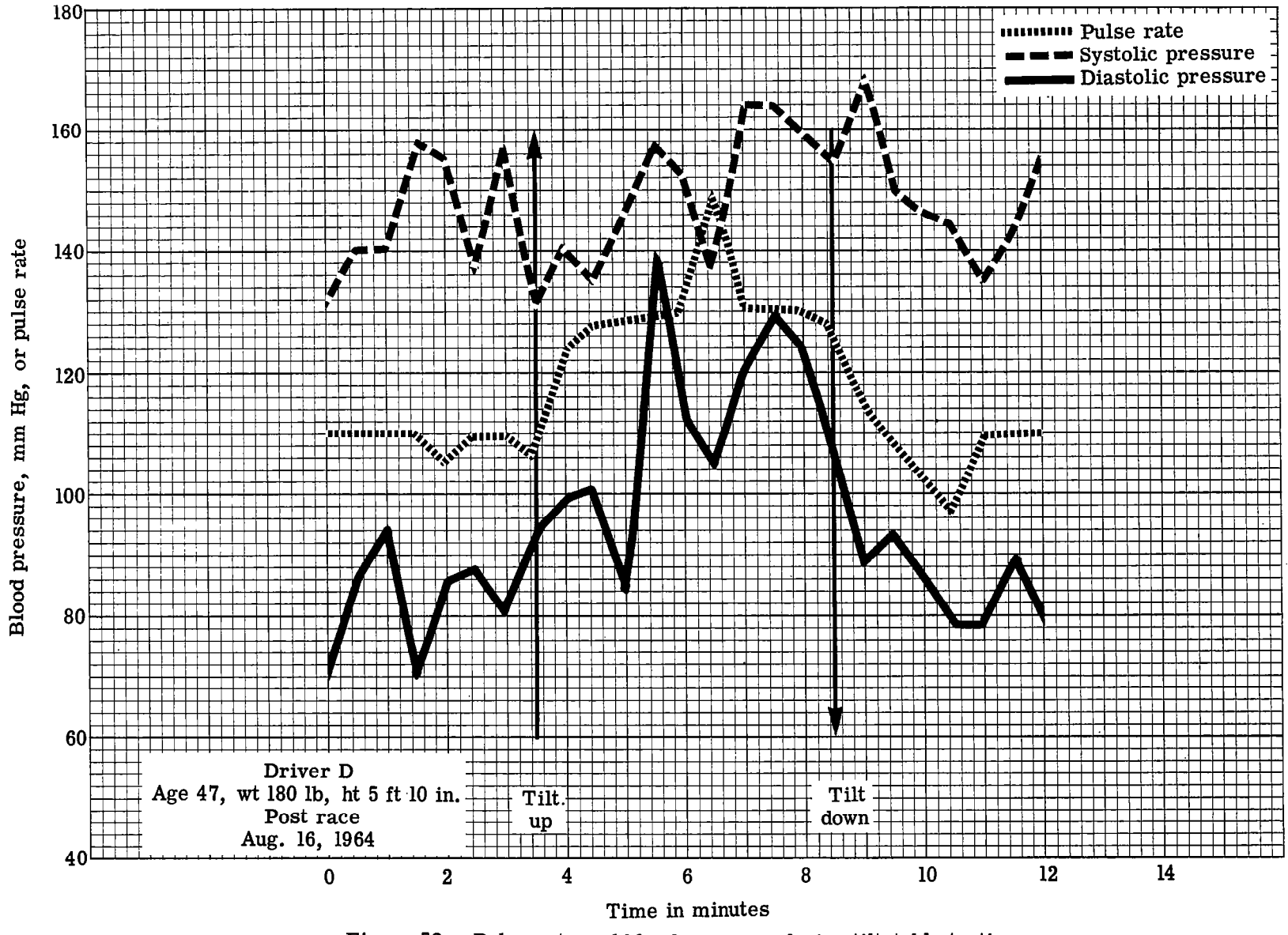



Figure 52. - Pulse rate and blood pressure during tilt-table testing.

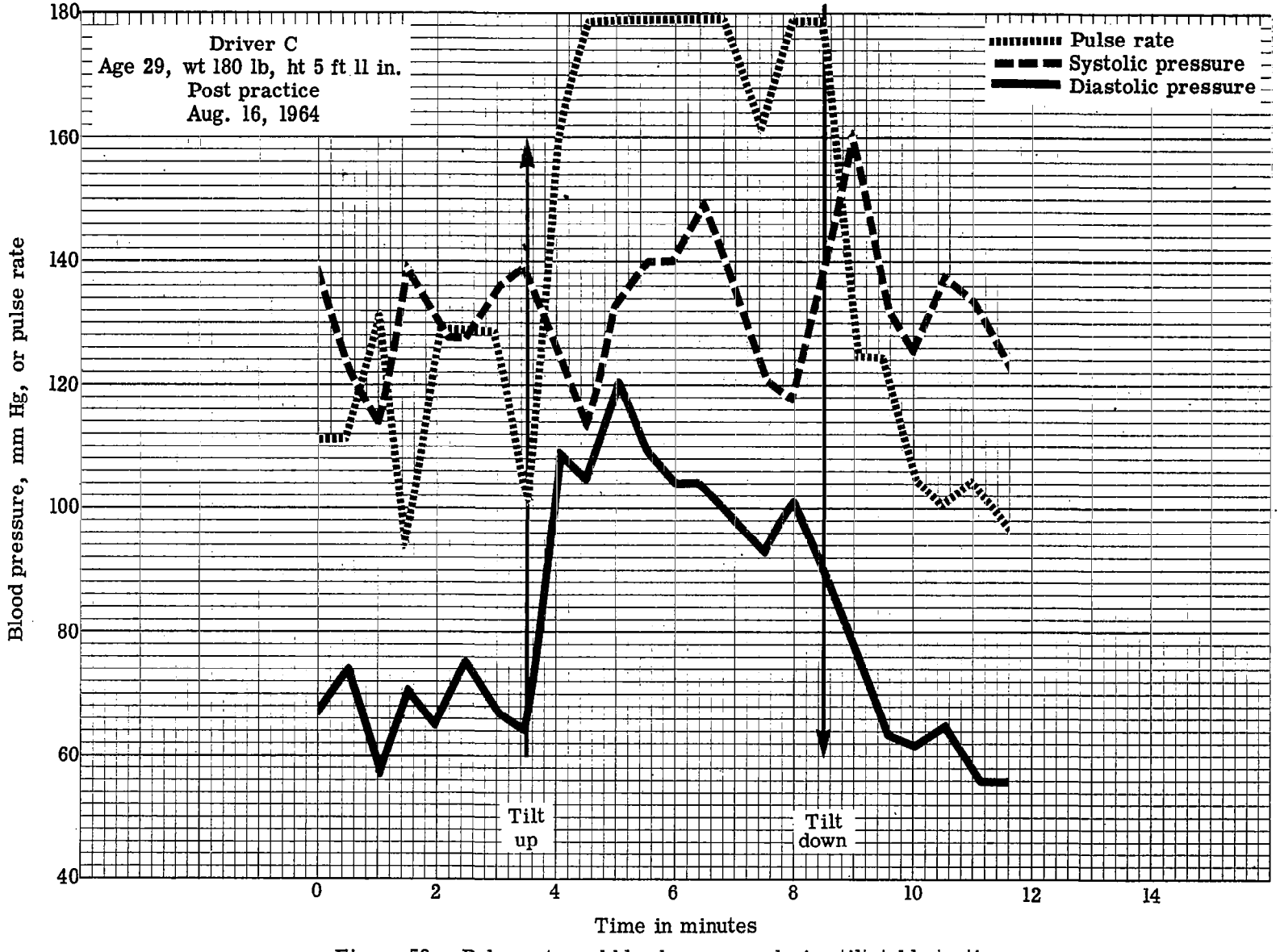



Figure 53. - Pulse rate and blood pressure during tilt-table testing.

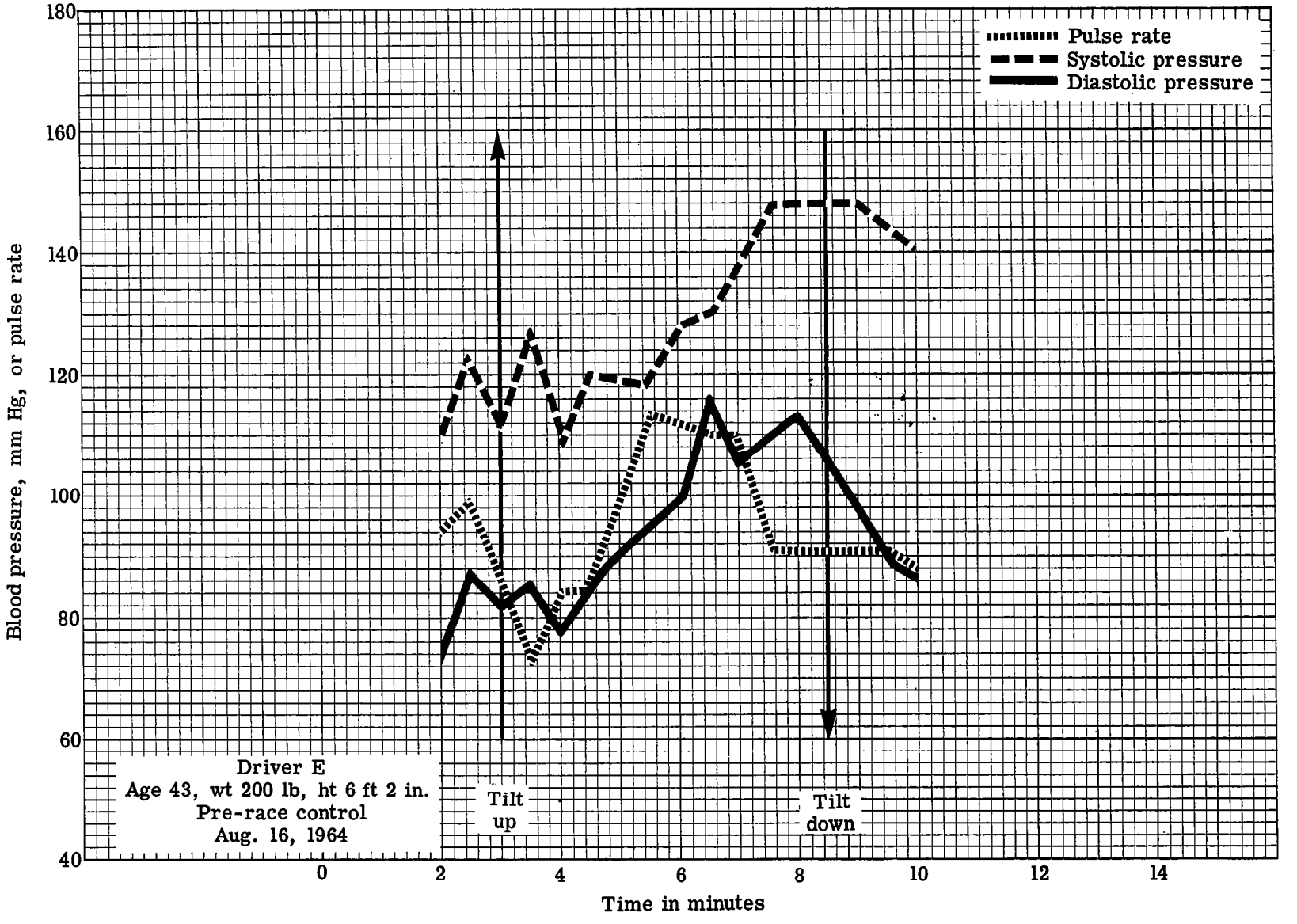



Figure 54. - Pulse rate and blood pressure during tilt-table testing.

In-race telemetry record of heart rate and temperature (figs. 55 and 56).- Two graphs compare the in-race records of heart rate and temperature of one individual (A), figure 55, without the benefit of the "cool suit" and, figure 56, while wearing the "cool suit."

Both tracings show the now familiar sudden acceleration in heart rate at the instant starting of the race. Without the ice-cooled suit the rise is in the range of 120 to 170; heart rate is maintained at an average of about 160 through the race; at the finish of the race there is a return to the pre-race level of 120 over a 4-minute period. When the driver is wearing the ice-cooled suit the initial rise in heart rate is from 130 to 180; the rate is maintained at an average of about 170; at the end of the race there is a precipitous fall in pulse rate from 165 to 120 in approximately 1 minute.

Without the benefit of the cool suit the pulse is maintained at about 10 beats per minute lower. One observation does not justify a conclusion, but the heart is bearing a greater burden of body cooling.

The sudden decrease in heart rate at the finish of the race may indicate that no oxygen debt was incurred by the degree of physical exertion required for racing. Recovery is slower when the driver is not protected from heat by the ice-cooled suit. This may reflect a greater fatigue under this condition.

Without the cool suit, temperature rose from 99.5° to 101° F during the first 10 minutes of the race and was maintained at this level for the duration.

Wearing the cool suit, the driver's temperature rose more gradually from 99.5° to 101° F over a period of 15 minutes.

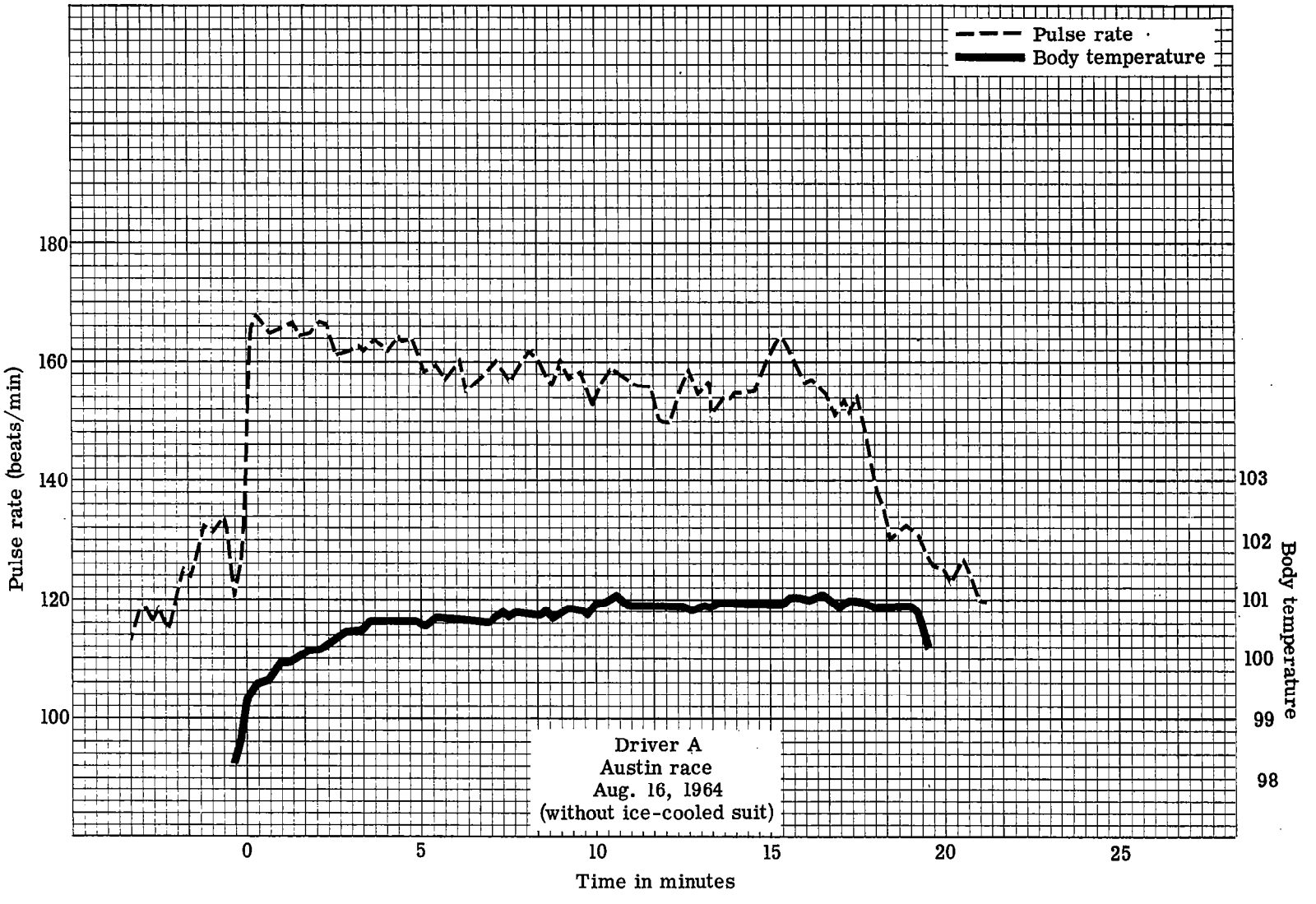



Figure 55. - Pulse rate and body temperature.

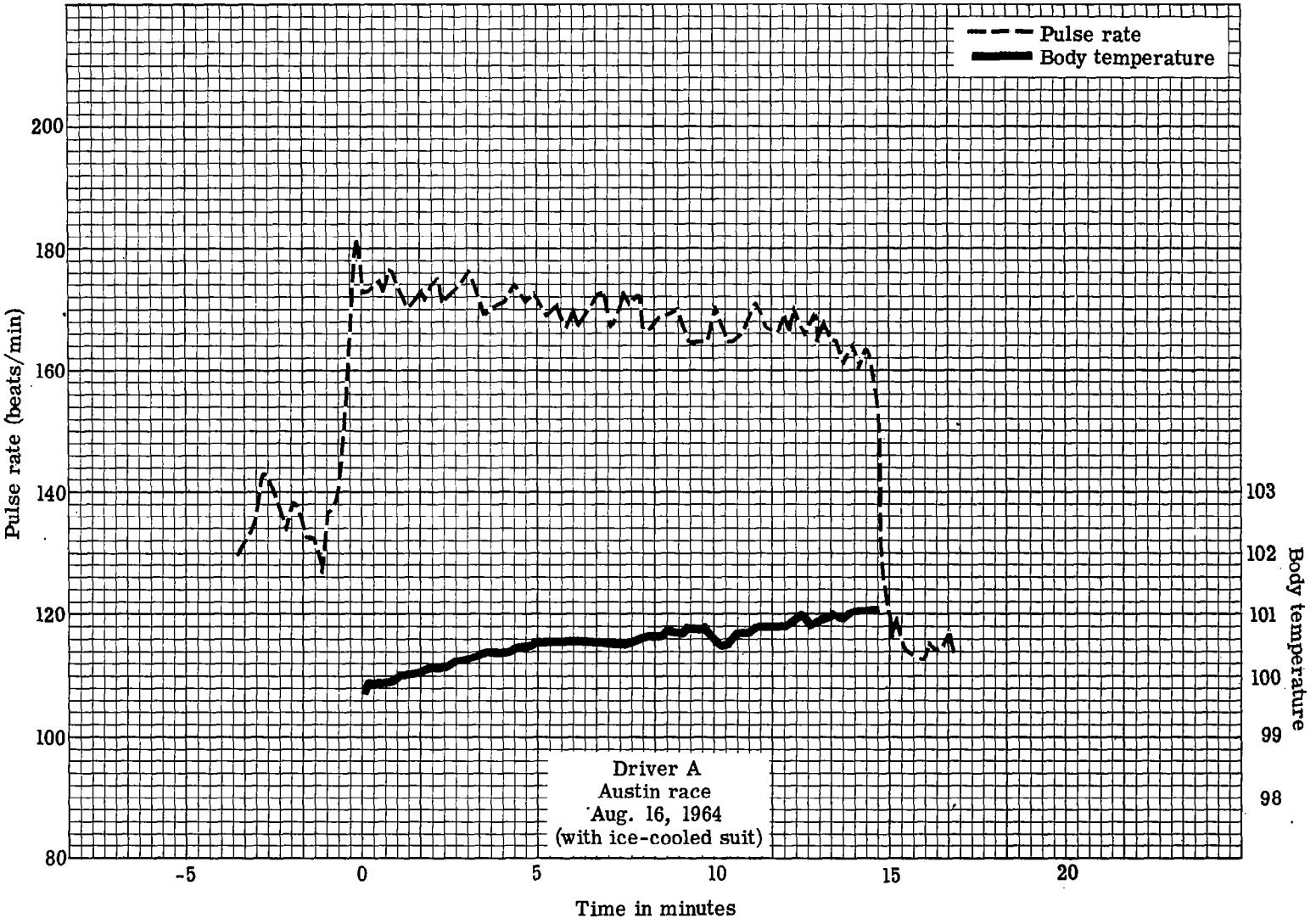



Figure 56. - Pulse rate and body temperature.

Tilt table tests - Houston Race, October 1964.- Figures 57 through 65 show tilt table tests of six individuals including a 21-year-old woman driver. All can be considered as within the range of variation of satisfactory responses.

The first, a 23-year-old man (fig. 57) shows a wide pulse pressure maintained through the tilt. Systolic pressure is maintained at about 140 with a rise in pulse from 100 to a maximum of 120. Diastolic falls gradually within the period of tilt from 80 to 50 with only slow recovery during the post-tilt record.

The second, a 29-year-old man (fig. 58) shows a mild narrowing of pulse pressure during tilt due to maintenance of a relatively level systolic pressure with a rise in diastolic pressure. The systolic pressure is maintained by a rise of pulse rate from 84 to 114. Post-tilt there is an increased pulse pressure due to a continuance of a slightly high systolic pressure and slightly lower pulse pressure.

The third subject (fig. 59), a 21-year-old woman maintains a virtually level systolic and diastolic pressure with a rise in pulse rate from 110 to 120 during tilt. Post-tilt there is a tendency toward fall in systolic pressure, diastolic pressure, and pulse rate, and a slight increase in pulse pressure.

The fourth subject (fig. 60) a 24-year-old male shows a narrowing of pulse pressure due to an initial sharp fall in systolic pressure with a gradual recovery and an initial rise in diastolic pressure which gradually falls off. Pulse rate during tilt rises from 115 to 130 but following tilt shows a precipitous fall to below pre-tilt levels.

At this time none of these subjects had control resting tilt table tests.

The fifth subject, a 51-year-old male, is presented with two controls. In the post-race tilt (fig. 61) there is a sharp drop in systolic pressure with a gradual recovery associated with a rise in pulse rate from 105 to 120. Diastolic pressure shows a slight but definite rise during tilt and then resumes the previous level.

Two resting controls (figs. 62 and 63) are similar to each other in pattern but differ from the post-race pattern. Instead of the precipitous drop in systolic pressure on tilting up, both show a prompt rise in systolic pressure of 25 to 30 mm Hg associated with an increase in pulse rate of about 30 pulses per minute. The diastolic pressure shows a commensurate rise during tilt to maintain an essentially stable pulse pressure during and after tilt.

The sixth subject, a 24-year-old male, has both control and post-race tilt table tests (figs. 64 and 65).

In the control there is a slight rise in systolic and diastolic pressure with little change in pulse pressure. Pulse rate shows an increase in the range of 75 to 95. In the post-race tilt table test, there is a slight fall in systolic pressure (150 to 130) and a moderate rise in diastolic pressure (80 to 110) with some narrowing of pulse pressure. Pulse rate increases in the range of 85 to 115.

Control testing tilt table tests are clearly necessary to recognize the influence of stress and these are being filled in during the present season.

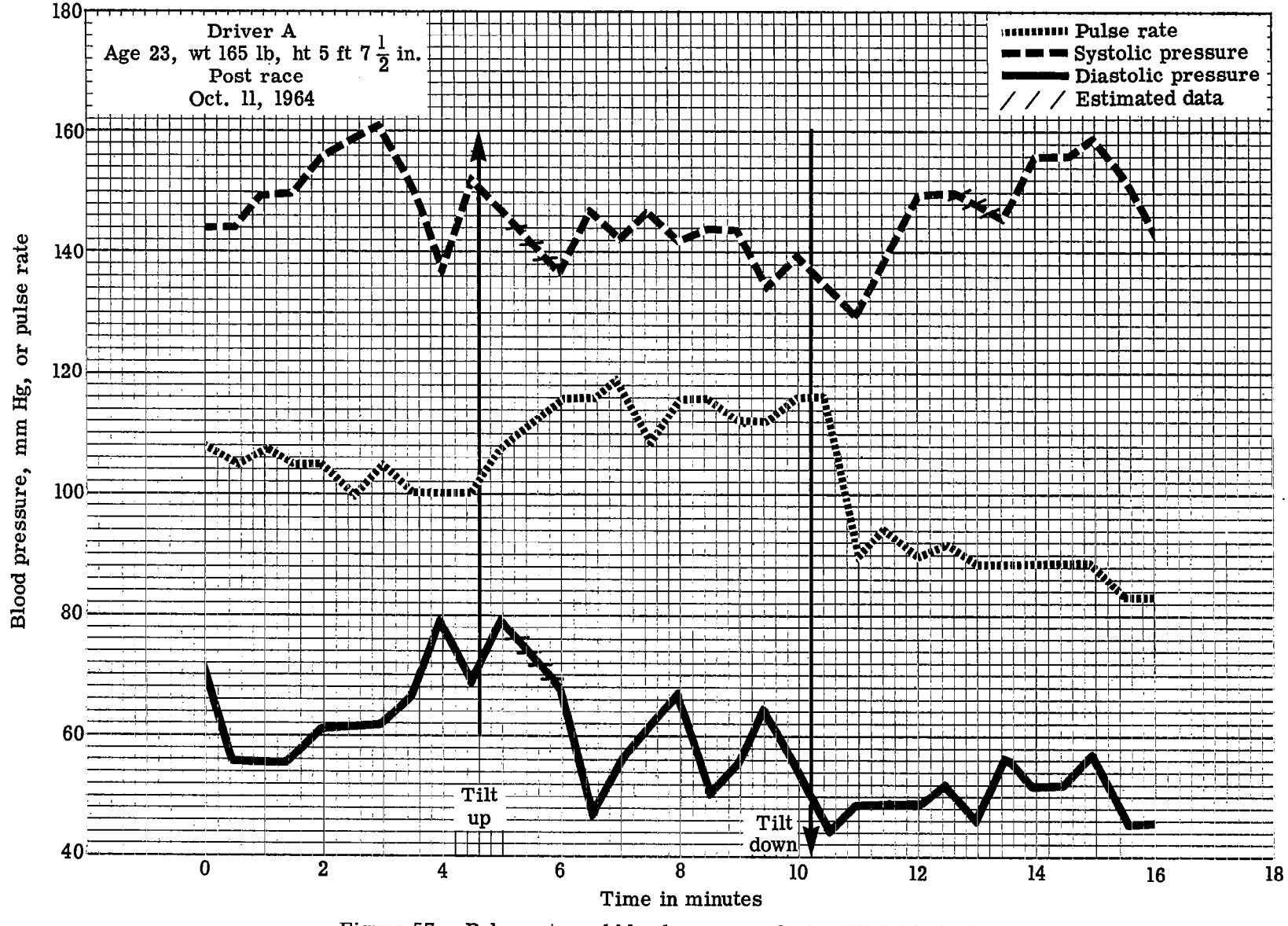



Figure 57. - Pulse rate and blood pressure during tilt-table testing.

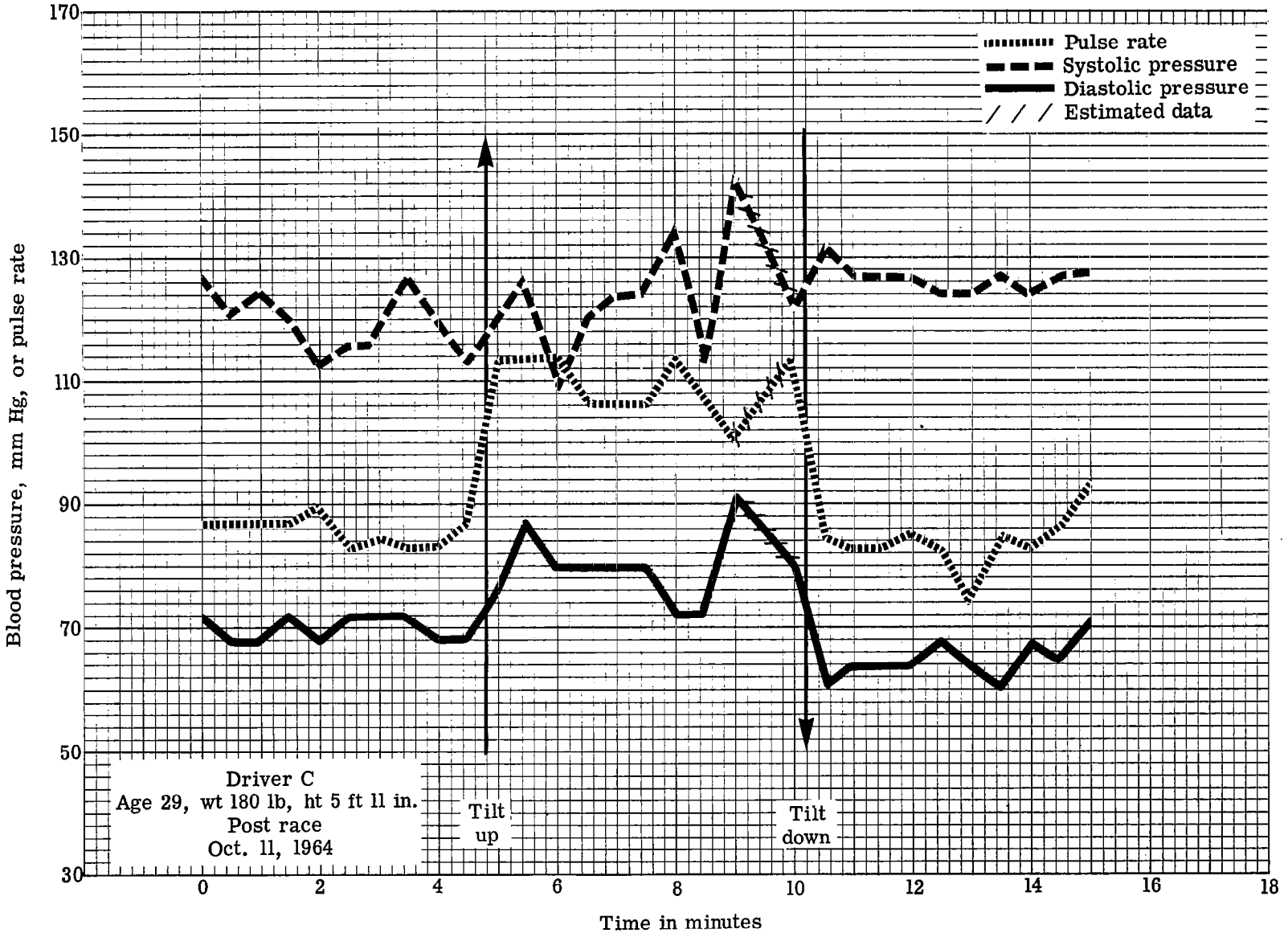



Figure 58. - Pulse rate and blood pressure during tilt-table testing.

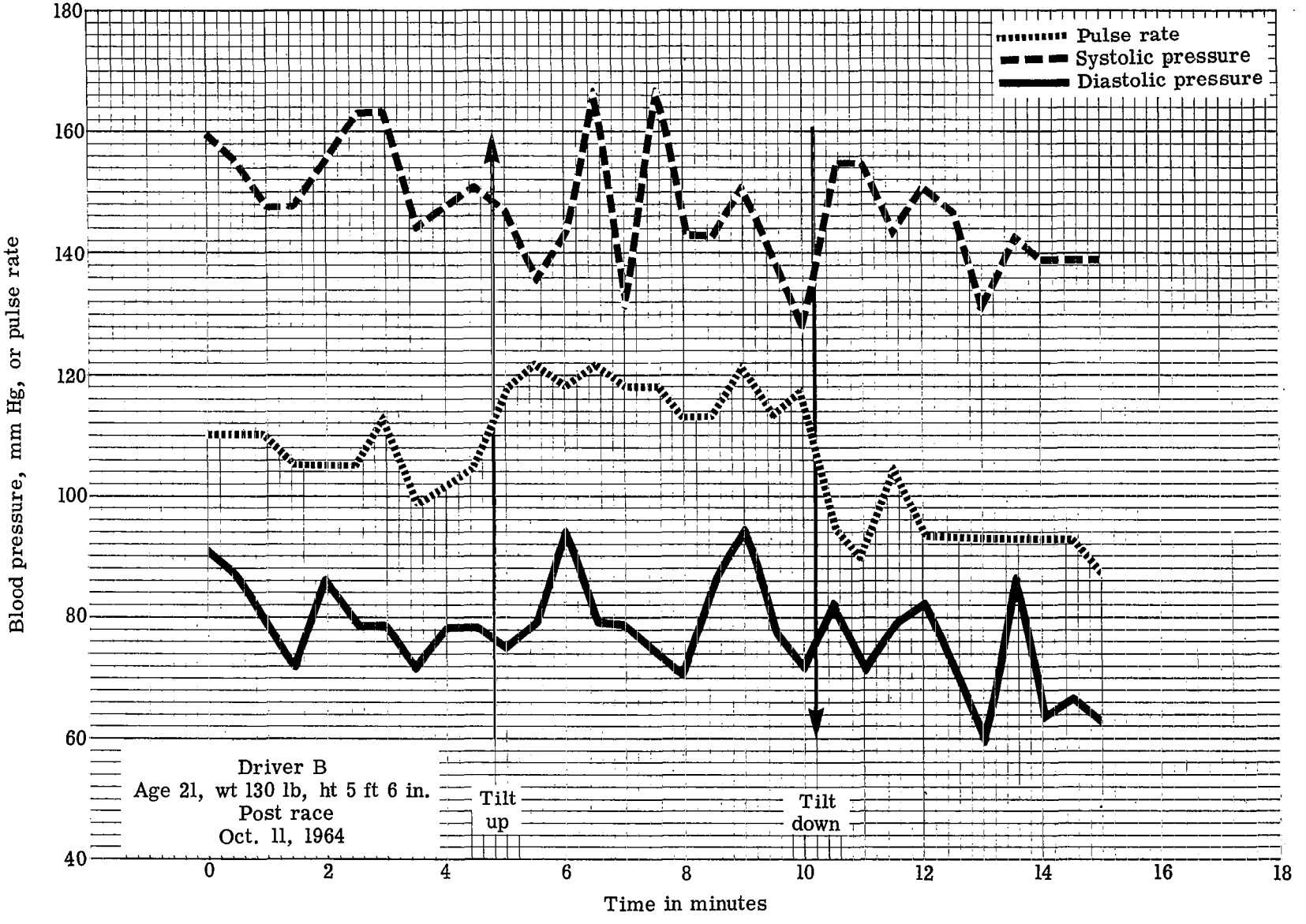



Figure 59. - Pulse rate and blood pressure during tilt-table testing.

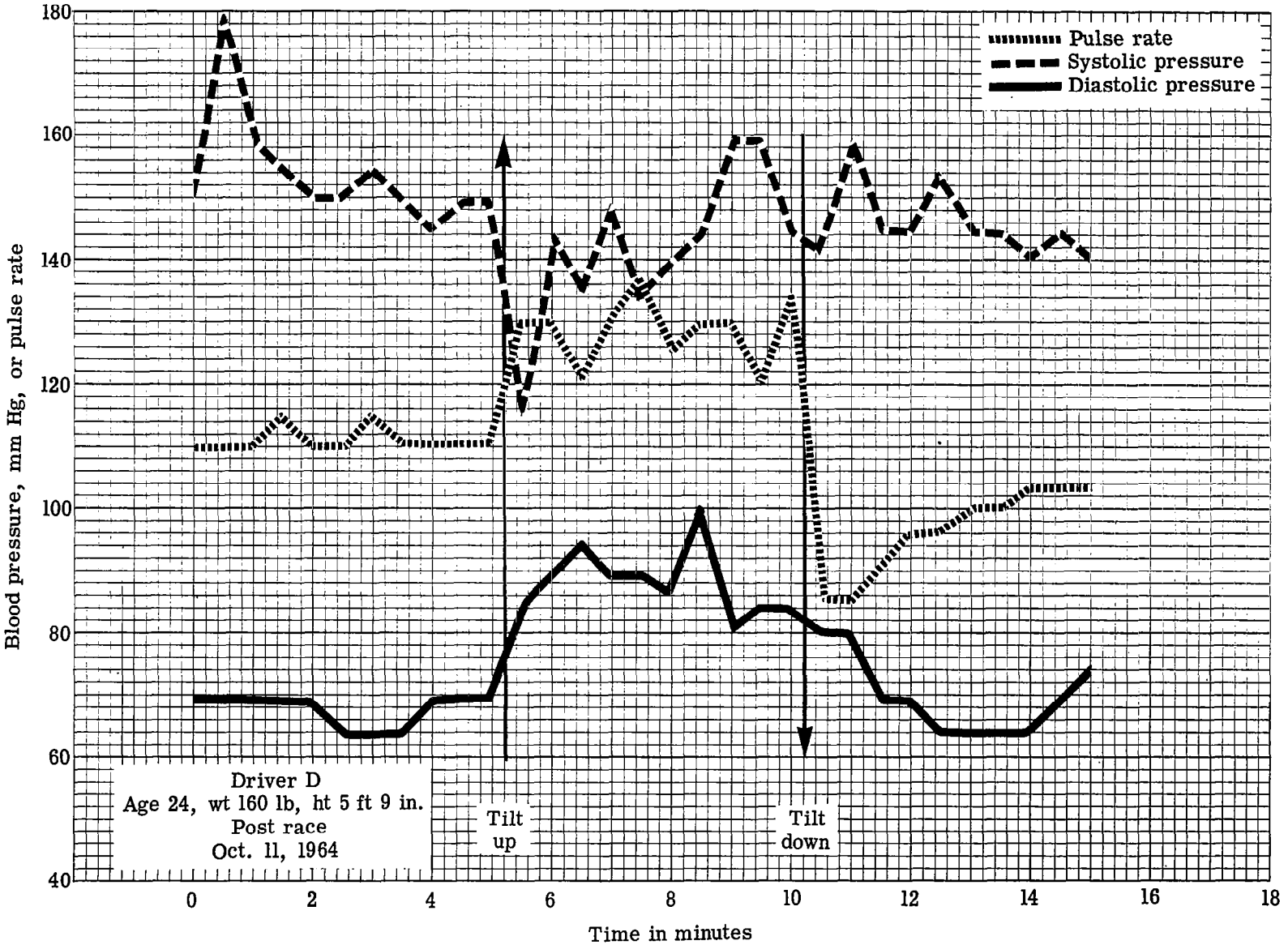



Figure 60. - Pulse rate and blood pressure during tilt-table testing.

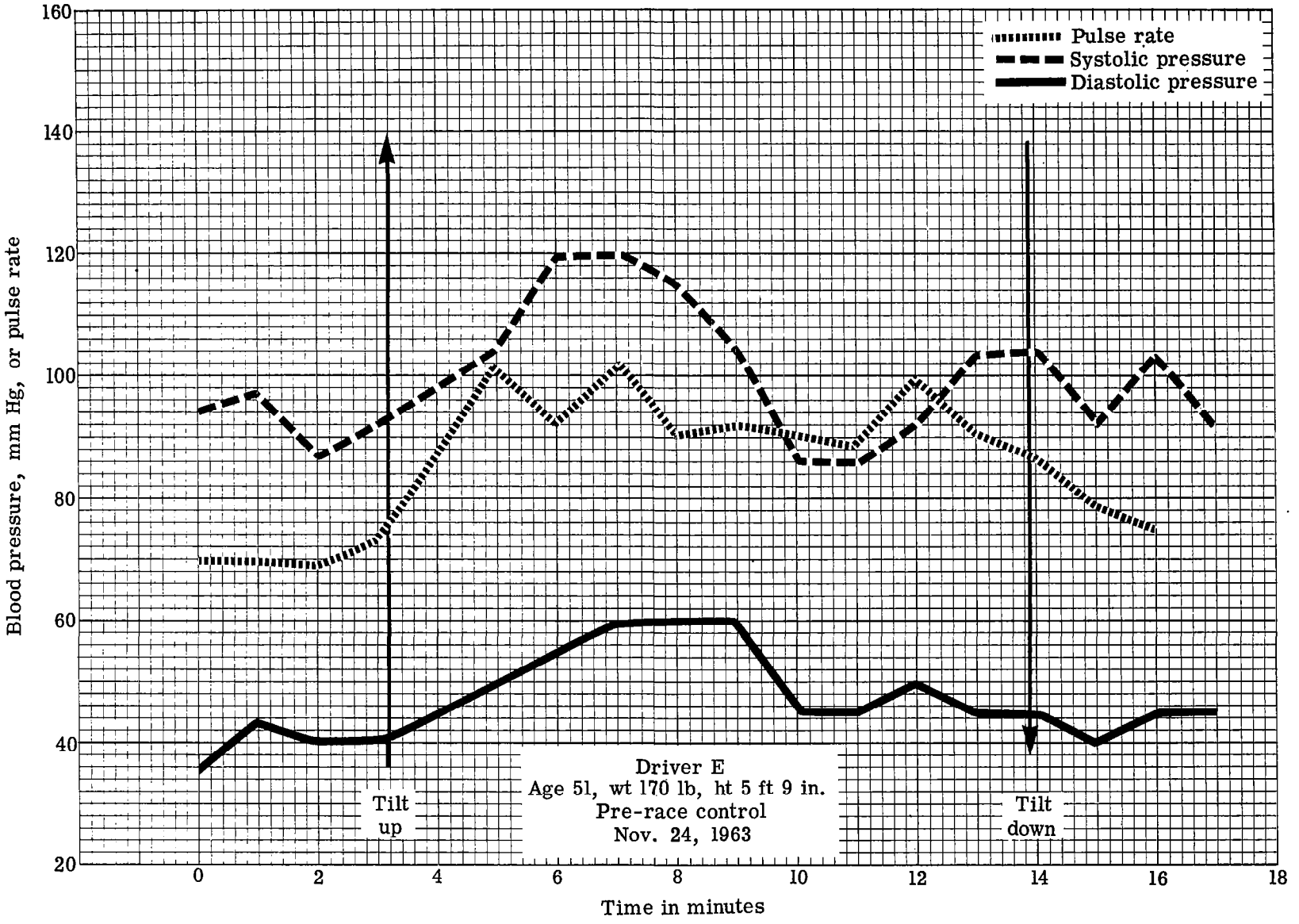



Figure 61. - Pulse rate and blood pressure during tilt-table testing.

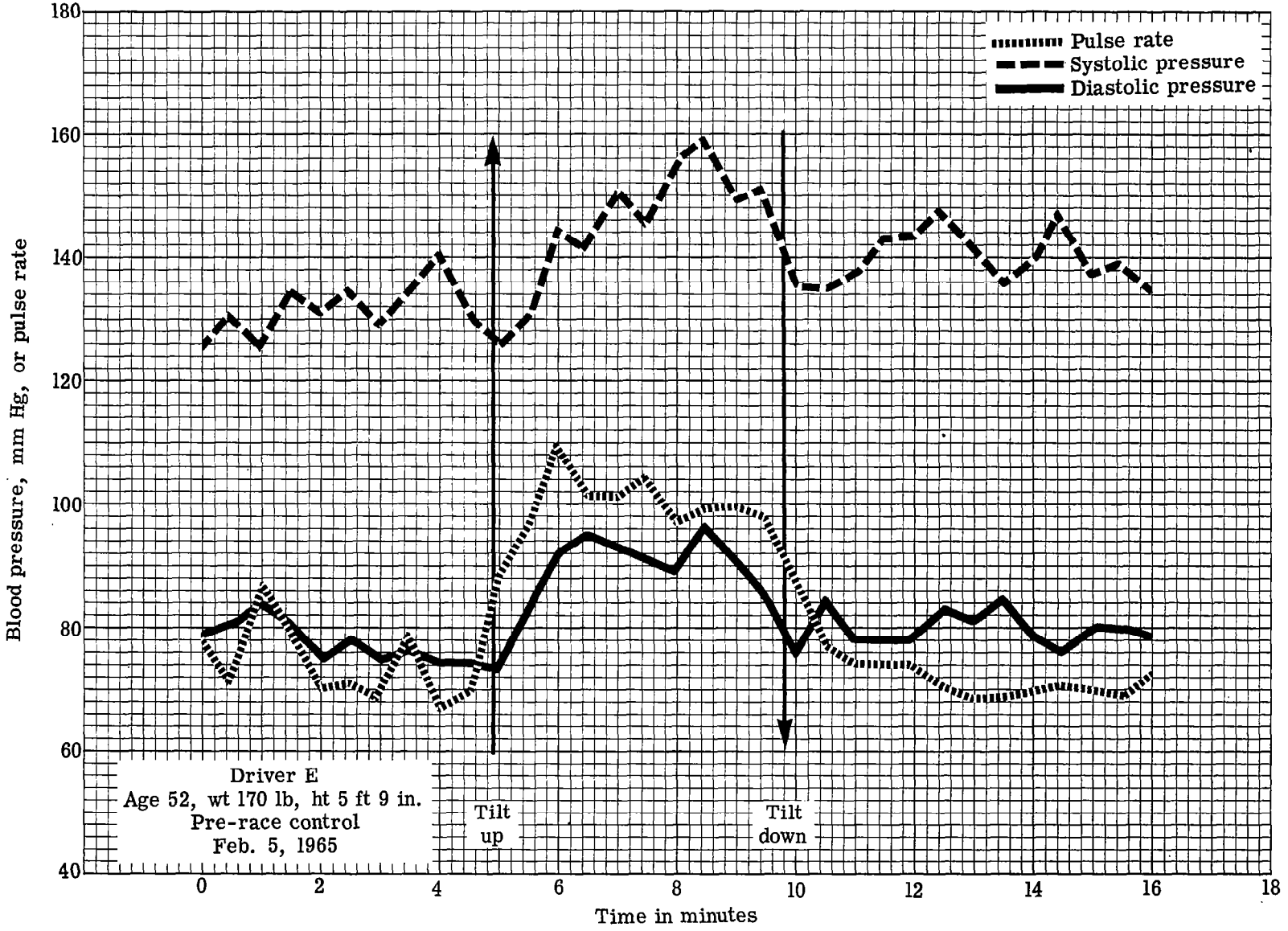



Figure 62. - Pulse rate and blood pressure during tilt-table testing.

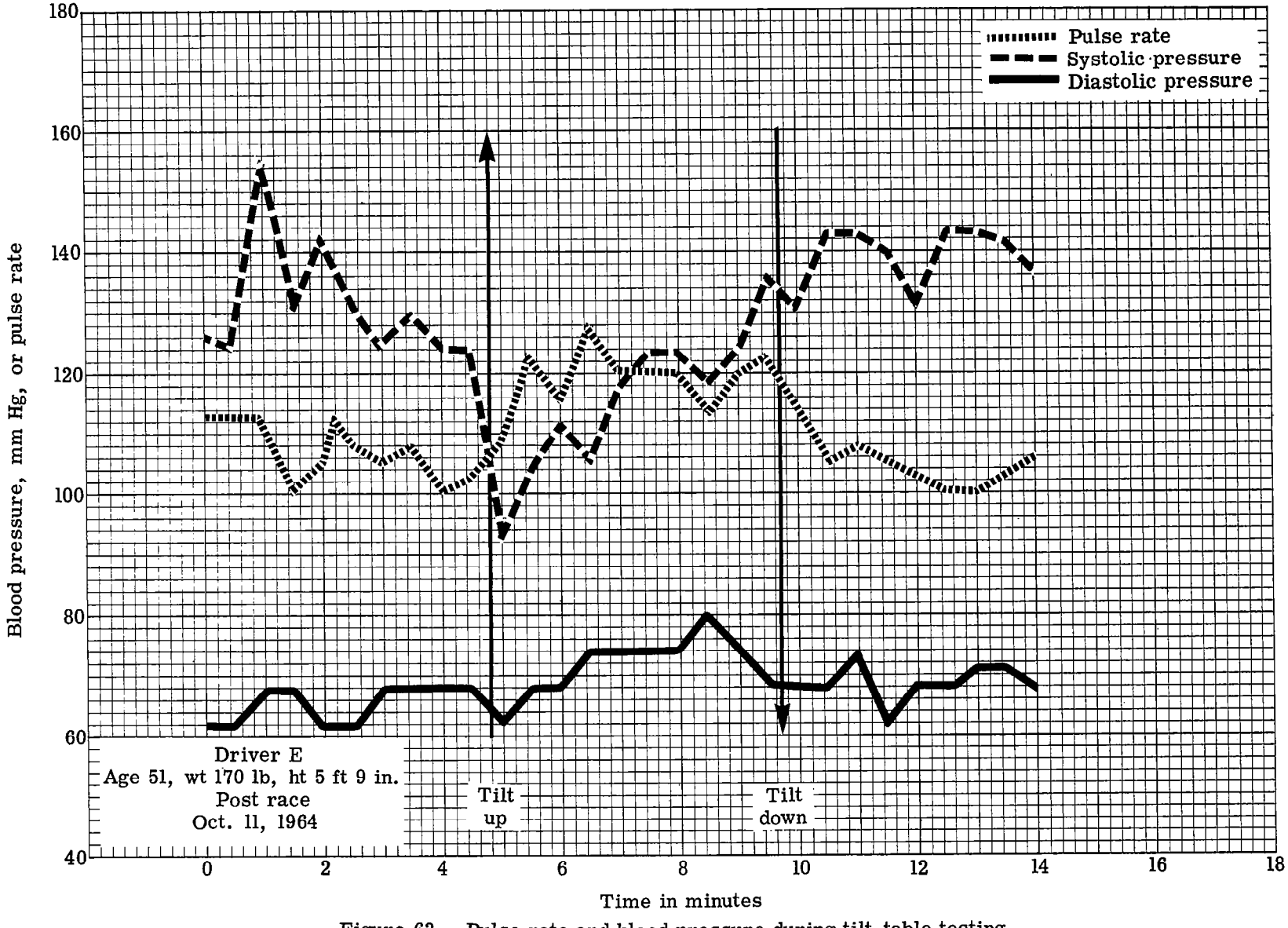



Figure 63. - Pulse rate and blood pressure during tilt-table testing.

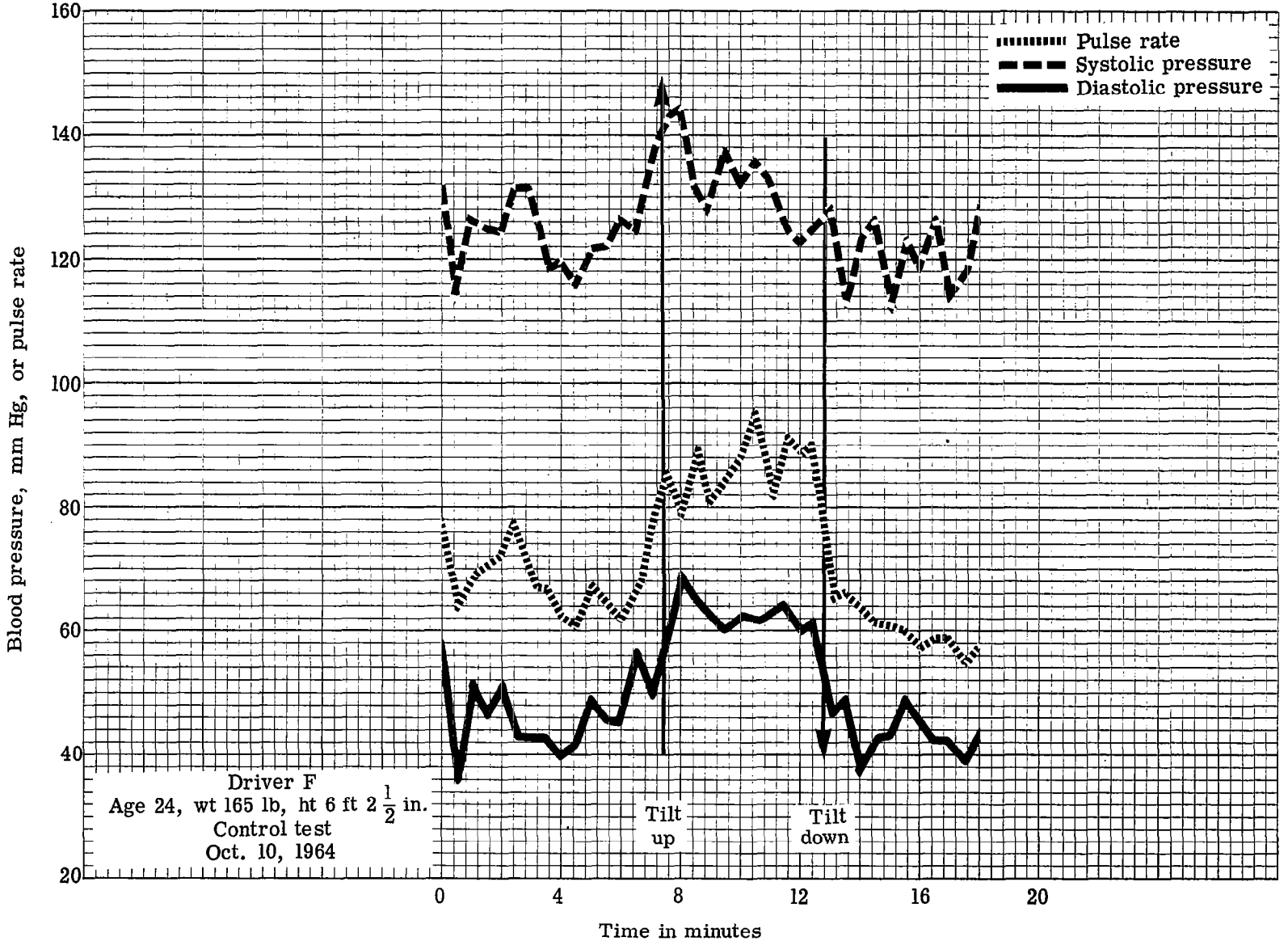



Figure 64. - Pulse rate and blood pressure during tilt-table testing.

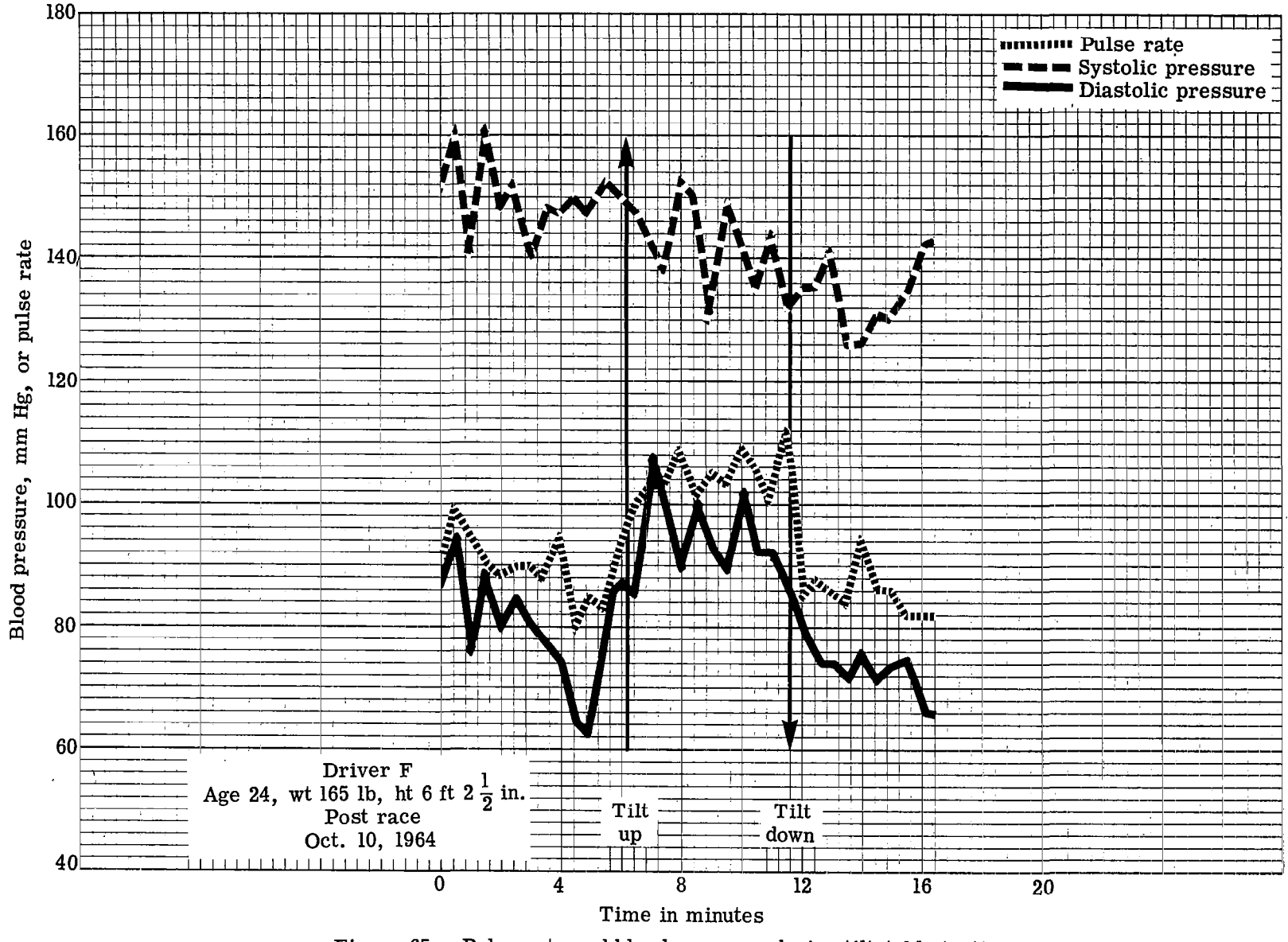



Figure 65. - Pulse rate and blood pressure during tilt-table testing.

In figure 66 heart rate is monitored during a race by an in-car tape recorder. The ECG signal is recorded and at a later time read out as a cardiotachometric tracing to permit recognition of rapid changes in heart rate.

The recording shows an abrupt increase in heart rate from 140 to 180 beats per minute at the start of the race (1). This is maintained until a minor accident (2) damages a wheel, necessitating a slower pace. As the driver continues at reduced speeds, and no longer in competition, the heart rate slows. A second accident (3) removes the two leading cars from competition and, with the prospect of winning or placing restored, speed is increased and heart rate obediently rises. At the end of the race (4) the heart rate abruptly falls.

An in-race heart rate of the order of 180 is commonly recorded and there are frequent examples of over 200. This is of particular interest since the physical exertion involved in driving is far below the level of energy expenditure which in a runner might predictably evoke such rate. It may be deduced that this rate is not due to oxygen consumption and that cardiac output is not greatly increased. That no oxygen debt accrues is suggested by the sudden decrease in rate at the end of the race. A question that arises as to what purpose is served by such increase in heart rate. The answer may be sought in comparing the heart rates of a large group of drivers and relating this to the variables of age, physical condition, experience, and performance. It may not be assumed that lower rates are optimal without consideration of performance, which must be evaluated at the next stage in this investigation.

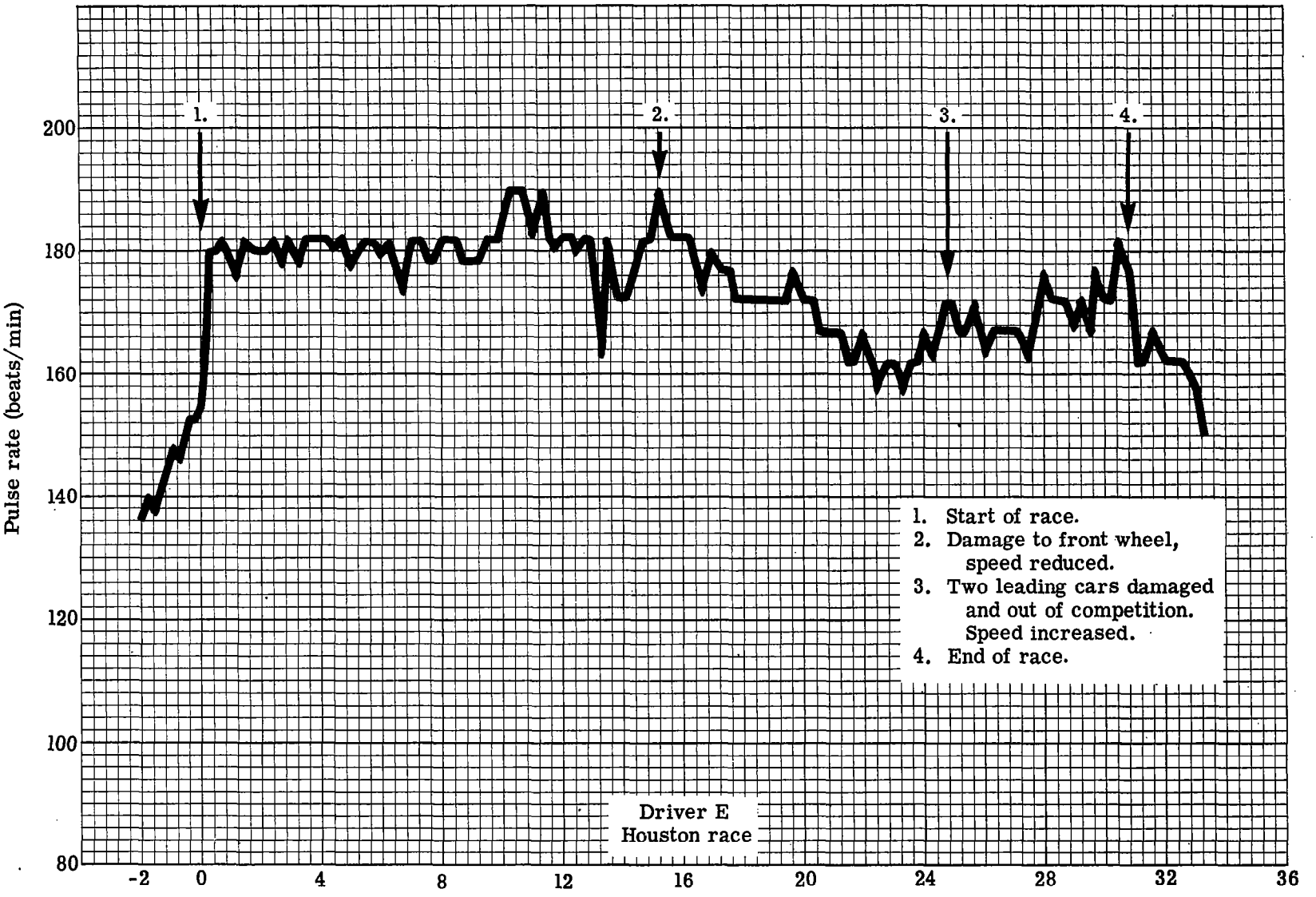



Figure 66. - Pulse rate history during race.

TABLE 19.- VITAL STATISTICS OF SUBJECTS IN RACES AT HOUSTON, TEXAS, IN 1964

| Driver | Physical description | | | Occupation | Marital status and no. of children | Education | Racing experience | | Rest prior to race |
|---|---|---|---|---|---|---|---|---|---|
| | Weight | Height | Age | | | | Type | No. of years | |
| Driver A | 165 | 5 ft 7 1/2 in | 23 | Student, car sales | No | Student, Jr. | Sports | 2 1/2 | 6 hr sleep |
| Driver B | 130 | 5 ft 6 in | 21 | College student | Yes no children | Math. major | | Less than 1 yr | 8 hr sleep |
| Driver C | 180 | 5 ft 11 in | 29 | Electrical draftsman | Yes 4 children | Trade school some college | Sports | 1 yr | 6 hr sleep |
| Driver D | 160 | 5 ft 9 in | 24 | Salesman | Yes no children | | Sports | 4 yr | 4 1/2 hr sleep |
| Driver E | 170 | 5 ft 9 in | 51 | M.D. | Yes 3 children | M.D., L.L.B. | Sports | 3 yr | |
| Driver F | 165 | 6 ft 2 1/2 in | 24 | Circuit eng., college student | Yes no children | Attending college | | | 5 hr sleep |

TABLE 20.- BLOOD-CHEMISTRY STUDIES CONDUCTED ON DRIVERS

IN THE RACES AT HOUSTON, TEXAS

| Driver | Glucose | Cholesterol | Uric acid | Creatinine | Chloride | Phosphorus |
|---|---|---|---|---|---|---|
| Driver C | | | | | | |
| Pre-race | 76 | 200 | 5.2 | 1.5 | 117 | 2.3 |
| Post-race | 114 | 192 | 5.6 | 1.5 | 140 | 2.4 |
| Driver D | | | | | | |
| Pre-race | 76 | 252 | 3.6 | 1.2 | 109 | 3.0 |
| Post-race | 74 | 252 | 4.5 | 1.0 | 109 | 2.5 |
| Driver F | | | | | | |
| Pre-race | 56 | 270 | 5.5 | 1.0 | 114 | 2.3 |
| Post-race | 71 | 288 | 6.0 | 1.2 | 110 | 2.5 |

Glucose - Pre-race values two drivers were low normal and one was below normal. Post-race, one was elevated to upper limits of normal and two were in normal range.

Cholesterol - No significant changes occurred during the race. One driver's cholesterol levels were above normal.

Uric acid - The pre- and post-race uric acid concentrations were within normal limits.

Chlorides - Pre-race values of all three drivers were just above normal. Only one driver showed a 20 percent increase during the race; two showed no change.

Phosphorus - The pre-race levels were at low normal or below normal and were essentially unchanged after racing.

SUMMARY

The field of sports presents a broad opportunity for physiologic observations under conditions of physical and mental stress that vary greatly with the particular type of activity. Competitive auto racing has certain distinctive characteristics that lead to initiating this investigation.

(1) There is an environment of stress involving only moderate physical exertion but a relatively high element of danger.

(2) In this environment the individual must exercise mechanical control of a vehicle and judgment as to factors of motion, speed, and distance that are acquired skills far removed from instinctive or reflex responses that are utilized in other stressful sports.

(3) There is an element of motivation to aggressive action, which cannot be simulated under laboratory conditions, that may influence responses and performance.

(4) It is considered that these factors simulate in some degree the stress environment of space flight, particularly on launching or reentry.

The present investigation explores a variety of physiologic responses to this particular type of environmental stress as the first of two parts of the total study. The importance of the study lies in how alterations in physiologic status may alter performance. The evaluation of performance is the second part of the study presently in the planning stage.

In field research of this type there is a tenuous control of strongly individualistic subjects, race cars operating beyond intended capabilities, and laboratory equipment exposed to the elements. The questions revealed in these circumstances may be studied more meticulously at a later date in the controlled environment of the laboratory.

The study includes observations on four auto races during 1964 (Daytona, Galveston, Austin, and Houston), and 29 subjects, some of whom have participated more than once.

## Tilt Table Tests

Tilt table tests were obtained on 29 individuals immediately after racing, sometimes more than once. Where possible, resting controls were obtained prior to or after race day; where controls are missing they may still be obtained when opportunity presents.

The stress of auto racing induces changes in the pattern of the tilt table record which vary in magnitude and type.

Patterns encountered among tilt table test subjects:

(1) Decrease in both systolic and diastolic pressure:

| | |
|---|---|
| Driver A | Figure 57 |
| Driver B | Figure 12 |
| Driver C | Figure 13 |
| Driver D | Figure 22 |
| Driver I | Figure 14 |

Three of five (C, I, and B) showed an increase in heart rate of over 30. All of these were after stress and Drivers D and C showed syncope.

It is observed that whenever diastolic pressure falls, systolic pressure also falls. This apparently indicates a failure or inadequate response of peripheral vasomotor reflex construction with pooling in dependent spaces. Diminished venous return to heart would preclude an increase in cardiac output. This must be overcome or syncope occurs as it did in three of these subjects, D, C, and I.

(2) Decrease in systolic pressure and increase in diastolic pressure:

| | |
|---|---|
| Driver A | Figure 10 |
| Driver B | Figure 63 |
| Driver B | Figure 47 |
| Driver D | Figure 38 |
| Driver D | Figure 60 |
| Driver F | Figure 37 |
| Driver F | Figure 65 |
| Driver H | Figure 32 |
| Driver I | Figure 30 |
| Driver I | Figure 31 |
| Driver J | Figure 36 |

This is the common post-race pattern apparently showing a stress effect adequately compensated by a strong peripheral vasomotor reflex constriction of the capillary bed. Only Driver I showed the drop in systolic pressure in his control tilt and this may be related to his bizarre post-race tilt record with a remarkably narrow pulse pressure.

(3) Increase in both systolic and diastolic pressure:

| | | | |
|---|---|---|---|
| Driver A | Figure 9 | Driver F* | Figure 15 |
| Driver B | Figure 35 | Driver G* | Figure 17 |
| Driver C | Figure 58 | Driver H | Figure 33 |
| Driver D | Figure 52 | Driver H* | Figure 16 |
| Driver E | Figure 62 | Driver J* | Figure 18 |
| Driver E | Figure 61 | Driver K* | Figure 20 |
| Driver E* | Figure 21 | Driver L* | Figure 19 |
| Driver E | Figure 54 | | |

This is presumed to represent the optimum tilt response and the most effective effort to maintain cerebral circulation by increased cardiac output and by peripheral vasomotor reflex constriction and adequate venous return. It is notable that all the professional race car drivers* fall in this category.

(4) Ten individuals showed a moderate to marked decrease in pulse pressure:

| | |
|---|---|
| Driver A | Figure 10 |
| Driver B | Figure 12 |
| Driver C | Figure 53 |
| Driver C | Figure 13 syncope |
| Driver D | Figure 52 |
| Driver D | Figure 22 syncope |
| Driver E | Figure 34 |
| Driver I | Figure 31 |
| Driver I | Figure 14 syncope |
| Driver J | Figure 36 |

A prominent decrease in pulse pressure may call for a more thorough investigation of the individual's cardiovascular status. Driver I's extremely narrow pulse pressure, post-race, is unique in this series. Driver D has been treated for hypertension and diabetes. Drivers C and E are very young individuals in apparent good health but both are particularly tense and responsive to their environment. Drivers C, B, and J tend toward overweight. Driver A is of American Indian stock, slender, wiry and calm in manner.

(5) Heart rate:

The range of increase in heart rate on passive tilt to the orthostatic position in 40 tests on 26 subjects, ranged from 0 to 68, with a mean of 27. A correlation was sought with age or apparent physical condition but no evident relationship was found. Initial heart rate in the recumbent position before tilting was not consistently related to increase in heart rate on passive tilt. Of individuals with an initial rate of less than 90, five of seven showed an increase above the mean. Of individuals with an initial rate between 90 and 150, 16 of 31 had an increase above the mean. Two individuals with an initial rate over 150 had increases above the mean. Of those subjects with control tilts (E, I, F, B, and A) all but one showed a higher heart rate in the recumbent position after stress than in the resting control, and a greater increase in heart rate on tilting after stress than in the control.

There were 10 tilt tests in which the systolic pressure showed a fall during passive tilting. All but one of these showed an increase in pulse rate during tilting which was less than the mean.

There were 14 subjects who showed an increase in systolic pressure during passive tilt. This was always associated with an increase in diastolic pressure. These subjects uniformly showed an increase in pulse rate during tilting which was greater than the mean.

## In-Car Recordings

The in-car recordings of electrocardiogram and temperature obtained at Daytona are in the hands of NASA personnel.

This report contains the in-car records of three subjects E (fig. 39), A, with and without the cool suit (figs. 55 and 56), and E (fig. 66).

The dominant features of interest are the instantaneous rise at the start of a race to levels of 169 to 183 beats per minute. This level tends to be maintained during races lasting up to 30 minutes and then falls almost as suddenly to pre-race levels. Although driving is moderately strenuous exertion, it is not such as to greatly increase oxygen consumption and the purpose served by the high pulse rate is not clear. It may be explained as anticipatory but is more prolonged than is expected on this basis. Oxygen consumption studies during this type of driving would be feasible for a trained individual and would be of interest in explaining this phenomenon.

Observations on one individual racing in summer heat with and without the cool suit show suggestive changes indicating diminished fatigue with cooling. This portion of the study should be extended to more individuals and for more protracted events so that the difference in fatiguability might be more clearly demonstrated.

## Blood and Urine Studies

Blood chemistries included glucose, $CO_2$ combining power, LDH, urea nitrogen, cholesterol, SGOT, SGPT, uric acid, creatinine, chloride, and phosphate. Of these serum glucose may be of greatest importance because of the possible influence of hypoglycemia on performance. In short races (fig. 24(a) and 24(b)), a duration of the order of 30 minutes, there was consistent elevation of serum glucose. This is explicable on a basis of mobilizing of glucose stores. In endurance races where driving stress is of 2 hours duration or more, the trend is toward a decrease in serum glucose post-race. This may be augmented by irregular eating habits

under the existing conditions. One individual (C, fig. 2(b) who showed only a moderate drop in serum glucose during two races, (fig. 3(a) and 3(b)), had an episode of weakness and fainting at his hotel about 2 hours after the race. The clinical features were compatible with the hypoglycemic episode and he was revived with available carbohydrative fluids without an opportunity to obtain a blood sample.

$CO_2$ combining power closely parallels the observations on serum glucose.

Creatinine is the only other blood chemistry of apparent significance in this study (fig. 27(a) and 27(b)). There is consistent trend to increase during stress. It is not suggested that the increase is directly related to stress, but rather that it may indicate alteration in renal blood flow, since creatinine is cleared by the kidneys but not subject to tubular reabsorption.

In continuing studies where heat is an important factor, sodium and potassium values are important but rigid control of fluid and salt intake and close observation of fluid loss as reflected in body weight change are necessary. This is difficult under field conditions except for a limited number of closely controlled subjects.

Urine chemistry determinations included sugar, acetone, sodium, potassium, and osmolality.

There were urinary sugar determinations on 16 subjects with pre- and post-race samples in 14 subjects. Of a total of 30 specimens 28 showed a trace or more of sugar. Of 14 subjects with pre- and post-race samples, 7 showed an increase, 4 were unchanged, and 3 showed a decrease. No significance can be attributed to these findings since the intake of food and fluid was uncontrolled.

In four individuals with pre- and post-race determinations all showed a diminished urinary sodium post-race, and three of four showed an increased urinary potassium (table 13). This correlates with the retention of chloride in blood (fig. 28(b)). Osmolality of the urine was determined pre- and post-race. The elevated values obtained indicate an attempt to conserve body water probably by a decreased effective renal plasma flow or a high reabsorption of water in the renal tubules. The high pre-race values are probably a reflection of the samples being taken after the morning practice.

## Blood Volume

Blood volumes by the RISA technique were done in association with passive tilt. Two values were obtained, one recumbent prior to tilt, and one in the orthostatic position. In two subjects during the control study, a third value was obtained on return to the recumbent position.

In two control tilt studies, one of the two subjects showed a decrease in the calculated blood volume which returned to above normal upon again assuming the recumbent position. In post-race tilt studies 8 of 10 subjects showed a decrease in calculated blood volume presumably due to exclusion of pooled blood from the measured circulating blood. The two subjects having an increase in calculated blood volume also showed an increase in red cell mass. The source of this increase in red cells is not known.

Two subjects were lost to the study due to development of syncope while in tilt.

The changes in blood volume recorded in tables 6 and 7 are summarized:

| | Number of studies | Due to plasma | Due to red cell mass |
|---|---|---|---|
| Control studies | | | |
| (1) Blood volume increase on tilting up | 0 | - | - |
| (2) Blood volume decrease on tilting up | 1 | 1 | - |
| (3) No change | 1 | - | - |
| Post-race studies | | | |
| (1) Blood volume increase on tilting up | 2 | - | 2 |
| (2) Blood volume decrease on tilting up | 8 | 7 | 1 |
| (3) No change | 0 | - | - |

The further investigation indicated is to seek a correlation between blood volume and hematocrit changes and the maintenance of diastolic pressure in passive tilting.

## CONCLUSIONS

(1) Competitive auto racing provides an opportunity for studying the physiologic effects of stress which differ from most other sports in that there is an element of risk of injury and avoidance is by a learned manipulative skill which is not served by the fight or flight mechanisms of instinctive reflex responses.

(2) The passive tilt test is a useful index of alteration in cardiovascular responses following stress. Three patterns are discerned:

(a) a temporary fall in systolic pressure with a rise in diastolic pressure,

(b) a rise in both systolic and diastolic pressures, and

(c) a fall in both systolic and diastolic pressures.

It appears that if diastolic pressure falls, systolic will also fall. If systolic pressure rises, diastolic will also rise. However a rise in diastolic pressure is not necessarily associated with a rise in systolic pressure; a fall in systolic pressure is reversed if diastolic pressure increases. This suggests that peripheral vasomotor constriction is the essential feature in maintaining an adequate venous return to heart, an adequate cardiac output, and an adequate blood supply to brain.

A greater number of subjects with repeated examination along with more sophisticated analysis might determine whether the individuals are characterized by varying adequacy of their peripheral vasomotor responses.

(3) In-race recording of ECG shows a prompt and marked increase in heart rate coinciding sharply with the beginning and end of a race. Variations during a race are apt to be related to incidents increasing or decreasing stress.

A greater number of such in-race recordings is necessary to show individual patterns of response as to magnitude of increased heart rate, range of variation, and recovery.

(4) Of blood chemistries, the serum glucose is most labile under the conditions of this investigation and may be a sensitive index to stress and an important influence on performance. $CO_2$ combining power closely parallels serum glucose. Creatinine was the only other serum chemistry showing a consistent change. It may indicate a decreased renal blood flow during stress.

Of urine chemistries, the only notable finding is the consistent spilling of sugar but the significance is unknown under the varying conditions of food ingestion and fluid balance.

(5) Blood volume determination on passive tilting tends to show a decrease due to an apparent decrease in plasma volume. Less commonly, blood volume may show an increase on tilting up and this is due to an apparent increase in red cell mass. On resuming recumbent position after tilt, blood volume of two subjects shows an increase, presumably due to return of pooled blood to the general circulation and some augmentation of either plasma volume or red cell mass. The interrelation of alteration in blood volume and hematocrit values with peripheral vasomotor constriction and maintenance of diastolic pressure in passive tilting, should be studied further.



NASA-Langley, 1966 CR-570

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—NATIONAL AERONAUTICS AND SPACE ACT OF 1958

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