carotid and aortic bodies. This is one cause of rapid pulse in heart failure, anaemia, haemorrhage, high altitude, CO poisoning, etc.

5. **CO_2 excess.** In moderate amounts it increases the heart rate, partly by its direct action and partly reflex. But in larger amounts it produces heart block and reduces heart rate. [It should be noted that *circulation* is more sensitive to O_2 lack but *respiration* is more sensitive to CO_2 excess.]

6. **Body temperature.** Rise of body temperature (muscular exercise, fever, hyperthyroidism, etc.) increases the heart rate (*a*) by acting on the S. A. node, and (*b*) by stimulating the cardio-accelerator centre.

7. **Increased intracranial pressure.** It slows the heart rate by directly stimulating the vagus.

8. **Adrenaline.** Since it acts like the sympathetic, it should accelerate the heart rate, but in intact animals the rate is often reflexly reduced. Because, by causing vasoconstriction, adrenaline raises blood pressure and thus mobilises the sino-aortic reflex, so that heart rate may be reduced. But the force of contraction invariably rises.

9. **Thyroxine.** It quickens the heart rate (*a*) by directly stimulating the metabolic rate of the sino-atrial node, (*b*) by increasing the basal metabolic rate of the body, and (*c*) by a probable stimulating effect on the sympathetic.

10. **Muscular exercise.** It increases the heart rate. Because most of the factors, concerned with acceleration, are operating during exercise. For instance, increased venous return initially mobilises Bainbridge reflex. Increased respiration, excitement, anoxia, CO_2 excess, adrenaline secretion, etc., all come in and increase the heart rate.

CARDIAC OUTPUT

At each beat, certain amount of blood is pumped out by each ventricle into the circulation. This is called *cardiac output*. Two terms are used: (1) **stroke volume** or **systolic discharge** (*output*), and (2) **minute volume** (*minute output*). *Stroke volume means the output per ventricle per beat. Minute volume means the output per ventricle per minute.* Hence, minute volume = stroke volume × heart rate.

As the volume of blood put out by both sides of the heart is same, cardiac output is to be multiplied by 2 so as to calculate the quantity of blood pumped by the heart as a whole.

Normal values. In adults the average stroke volume is **70** ml and the minute volume about **5–6** litres. In other words, the amount of blood expelled per ventricle per minute, is approximately the same as the total blood volume of the body.

The output is directly proportional to the *metabolic rate*, and as such, to the **surface area** and **body weight.** The cardiac output per minute per square metre of body surface is known as **cardiac index.** The average value is 3·3 litres. The surface area of an average-sized adult is about 1·7 m^2. Accordingly, the average cardiac index is about 3·3 litres/min/m^2 (5·6/1·7). The stroke volume per square metre of body surface is known as **stroke volume index.** The average value is 47 ml. Anything that increases or diminishes basal metabolic rate, body weight or surface area, also alters the minute volume proportionally.

Distribution of cardiac output. Since venous return per minute should be the same as the minute output, it follows that blood flow through the tissue per minute must also be the same. In other words, 5 litres of blood passes out per ventricle per minute, 5 litres of blood flows through the tissues per minute and the same 5 litres come back to heart per minute. It is really astonishing, how accurately these constant relations—of time and quantity—are maintained.

Although full data are not known, yet the **minute volume of heart** is mainly distributed as follows:

(1) Kidneys—1,300 ml per minute. (2) Brain—700–800 ml per minute. (3) Coronary—200 ml per minute. (4) Muscle—600–900 ml per minute. (5) Liver—about 1,500 ml per minute. Total quantity of blood distributed in these organs does not exceed 4,500 ml per minute. So the remaining amount is distributed to the skin, bones and gastro-intestinal tract.

Translocation of blood. For a short period of time, left and right ventricles may pump different amount of blood. When the cardiac output of left ventricle is greater than that of the right, there is *translocation of blood* from pulmonary circulation to systemic circulation. If the cardiac output of right ventricle is greater than that of the left, there may be *translocation of blood* to the pulmonary vessels.

Cardiac reserve. It is the capacity of heart to generate sufficient energy for expelling a large quantity of blood and for raising blood pressure above the basal level during emergency. Generally, the normal heart expels about 5 to 6 litres of blood per minute per ventricle and during exercise this amount may be about 30 to 40 litres per minute. According to *Starling's law*, it is nothing but *physiological capacity of the heart.*

Control of cardiac output. Cardiac output depends upon the following four factors: (1) **venous return,** (2) **force of heart beat,** (3) **frequency of heart beat,** (4) **peripheral resistance.** Their roles are summarised below:

1. **Venous return.** Anything that increases or diminishes the venous return, will alter the cardiac output accordingly. Venous return depends upon the following: (*a*) *Muscular exercise*—When muscles contract, they squeeze the capillaries and venules and help in venous return. This is aided by the valves of veins, which prevent the passage of blood back towards the capillary bed. (*b*) *Respiration*—During inspiration intrathoracic pressure falls and intra-abdominal pressure rises. Hence, with each inspiration venous blood is sucked up by the thorax and is pumped out by the abdomen. (*c*) *Pressure difference between capillaries and venules*—Normally, there is a slight positive pressure (32–12 mm of Hg) in the capillary area (*capillary tone*), while in the great veins it may be even negative. Vascular dilatation without fall of general blood pressure will increase the capillary pressure and thereby raise the venous return (viz., muscular exercise). But if it causes a general fall of blood pressure—as in shock,—venous return will fall. (*d*) The *vasomotor system* adjusts the lumen of the arterioles and venules and thereby alters the venous return.

2. **Force of heart beat.** The strength of contraction depends mainly on three factors: (*a*) *The initial length* of the cardiac muscle—Within physiological limits, *greater the initial length, stronger will be the force of contraction* **(Starling's law** (Fig. 209)—which is an inherent, self-regulating mechanism that permits heart to adjust to changing end-diastolic volumes). It is

obvious that the initial length is proportional to the degree of filling, which again, depends on the venous return. (*b*) *The length of diastolic pause*—Filling, rest and recovery take place during diastole. Hence, with a shorter diastolic period which is inadequate for these, the force of contraction will diminish unless the rate of venous return is raised. (*c*) *Nutrition and oxygen supply*—An adequate supply of nutrition and oxygen is essential for efficient cardiac activity.

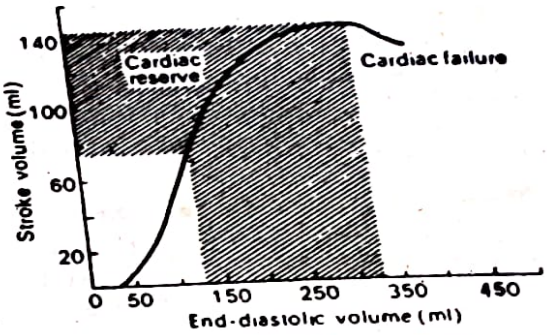

FIG. 209. Graphical representation of Starling's law of heart. Showing when the end-diastolic volume increases, ventricles contract more vigorously and thereby stroke volume increases. But when normal functioning (*shaded portion*) is beyond the range, the stroke volume decreases and the relationship is reversed (diagrammatic).

In addition to this, an optimum H-ion concentration, a proper balance of inorganic ions and an appropriate temperature and pressure, are also required for strong heart beat.

3. **Frequency of heart beat.** Heart rate affects both stroke volume and minute volume by altering the length of diastole and thereby the degree of filling and force of contraction. It should be noted that blood pressure depends upon the minute volume and not on the stroke volume. The following consideration will clarify. Venous return remaining constant, the rise of heart rate will reduce the diastolic pause and therefore the stroke volume. But the product—stroke volume × heart rate—may not fall, even it may rise above the resting value. Thus minute volume and therefore blood pressure (B. P.) may rise even if the stroke volume falls. This happens with a moderate rise of the heart rate. But if the heart rate be too high, the stroke volume becomes so low that the minute output falls far below the normal (FIG. 210). Blood pressure drops and the subject may be unconscious. This happens in *paroxysmal tachycardia* when the frequency suddenly becomes 150–200 per minute. [Muscular exercise is an exception. Here, both the frequency of heart beat and the rate of venous return increase. Cardiac filling becomes more than normal even during the short diastolic period. Hence, both stroke volume and minute output increase.] On the other hand, when the heart rate becomes very slow (as in *Heart Block*)—although the stroke volume is much bigger than normal, yet, the minute volume may fall, because the product may be less than normal. But with a moderate slowing the minute volume may not fall at all. In some instances it may rise (recovery from heart failure). Thus alteration of heart rate on either side will generally raise the minute volume up to a certain extent. Beyond that, the minute output will fall.

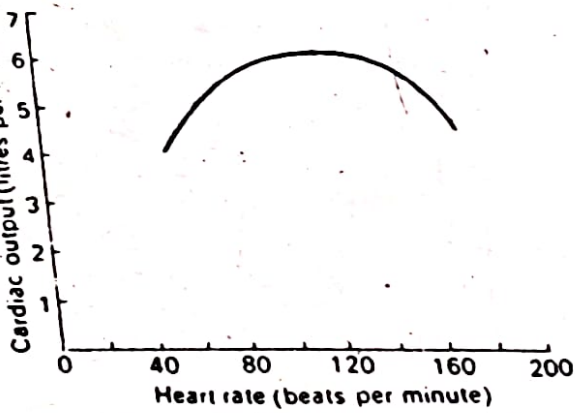

FIG. 210. Graphical representation of the relationship of cardiac output and heart rate assuming constant venous pressure and ventricular residue (diagrammatic).

4. **Relation with peripheral resistance.** Heart maintains a constant cardiac output and blood flow even against increased peripheral resistance. An optimum blood pressure is essential for adequate cardiac activity. General vasoconstriction of the arterioles will cause an increase in blood pressure. Heart at first fails to expel all its blood but in the next heart beat the filling becomes more, because the normal venous return is added upon the residual blood. Consequently, the initial length becomes bigger, the heart contracts with greater force and the normal output is restored.

Methods of measuring cardiac output

In Animals. The output can be measured with the help of (1) Cardiometers (page 255), (2) Heart-Lung preparation (FIG. 211), (3) Dye method, (4) Fick principle using O_2 or CO_2, (5) Physical method (Ballistocardiography).

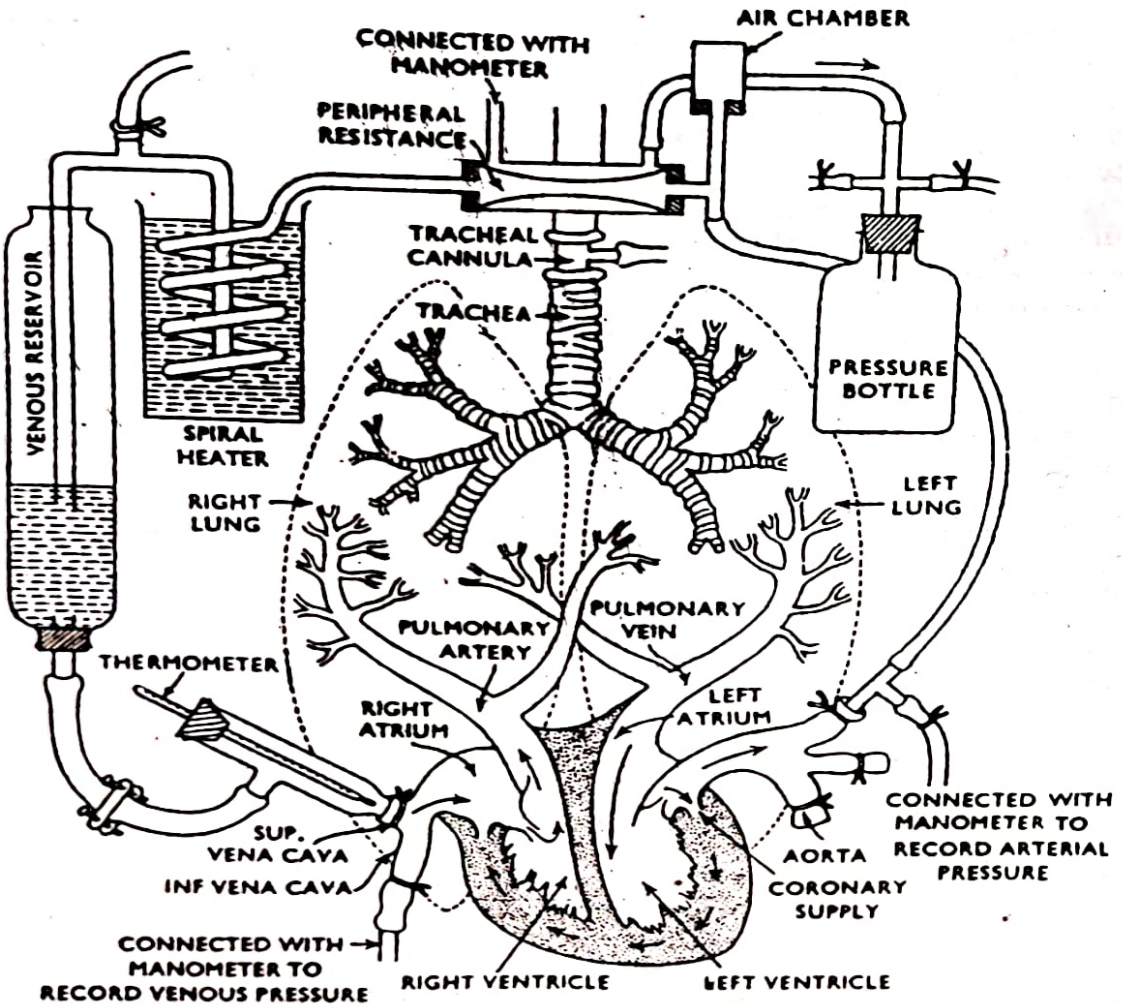

FIG. 211. Heart-Lung preparation.

Knowlton-Starling heart-lung preparation. This is a very useful method for studying cardiac output under different experimental

conditions, viz., variation of pressure, temperature, H-ion concentration, inorganic ions, etc. The procedure is as follows:

Thorax of an animal is opened, heart is exposed under anaesthesia. Artificial respiration is maintained. Both vagi are severed to prevent the variation of heart rate. The branches arising from the arch of the aorta, viz., the brachiocephalic and left subclavian arteries (in the dog) are ligated. A three-way cannula is inserted in the brachiocephalic artery. The cannula is connected with a mercury manometer to record the mean arterial pressure and through another three-way tube with a bottle containing air. The air of the bottle due to its compressing capacity serves the purpose of elasticity of the arterial wall. The other limb of the three-way tube is connected with a glass tube lined with a thin-walled rubber tube. The pressure inside this tube can be varied at will. This is an artificial resistance. The glass tube has got two side tubes—one of which is connected with a manometer for recording the pressure inside it and the other is connected with the pressure bottle from where air may be pumped inside it. This rubber-lined glass tube is connected with a spiral heater which opens in the venous reservoir filled with heparinised blood. The reservoir is connected through a rubber tube with a three-way cannula one limb of which inserted into the superior vena cava. A thermometer is kept in the cannula to record the temperature. The inferior vena cava is connected with a water manometer to record the pressure of the right atrium. There is a screw clamp on the rubber tube connecting the venous reservoir with the three-way cannula which adjusts the rate of flow from the venous reservoir into the right atrium. From the venous reservoir blood enters the right atrium, right ventricle and then through the lungs, left atrium, into the left ventricle. From the left ventricle through the brachiocephalic artery, resistance tube, spiral heater, blood comes again into the venous reservoir and the cycle is repeated. The output of the left ventricle can be determined by opening the clamp on the tube in between the spiral heater and venous reservoir and collecting blood for a specified period in a measuring cylinder. The blood flow through the coronary arteries must be added to give a correct figure of the output of the left ventricle. This experiment does not give any indication regarding the output in the human beings or in the intact animal.

Stewart and Hamilton's dye dilution method. There are certain difficulties invariably in introducing cardiac catheterisation. So the dye method is often the method of choice. A known quantity of Evans blue (non-diffusible dye, known as T–1824) is injected into the basilic vein. The dye will circulate through the heart, lungs and appear in the carotid artery. The concentration of the dye when it first appears in the artery is determined with the help of colorimeter from the samples of arterial blood taken every few seconds interval. The concentration of each sample of blood is determined by Photocolorimeter and plotted on a semilogarithmic paper. The dye concentration rapidly rises to a peak, then falls and rises again due to recirculation of dye. The mean concentration of the dye is calculated from the curves. The volume flow in litres per second is determined with the help of the following formula: $F = D/ct$, where F = volume flow in litres per second, D is the quantity of dye (Evans blue) injected, c is the mean concentration of the dye and t is the duration in seconds of the first passage of dye through the artery.

Fick principle. It was shown by Fick that cardiac output can be calculated by noting certain data about O_2 or CO_2 exchange. When applied to oxygen it will be as follows:

O_2% of mixed venous blood is determined,	say	15 ml.
O_2% of arterial blood is determined,	,,	19 ml.
Arteriovenous O_2 difference per 100 ml of blood is therefore—		4 ml.
Total O_2 consumption per minute (Douglas bag),	,,	200 ml.

Hence, each 100 ml of venous blood, while passing through lungs, takes away 4 ml of O_2. Therefore, 200 ml of O_2 will be carried away by $\frac{100}{4} \times 200$ ml = 5 litres of blood. Obviously, this is the minute output of right ventricle, which is same as that of left ventricle. Similar figures will be obtained by applying the principle to CO_2. So the cardiac output can be determined with the help of the following formula:

$$\text{Cardiac output (ml/min)} = \frac{\text{consumption of } O_2 \text{ (ml per minute)}}{\text{difference of } O_2 \text{ content in arterial and venous blood (ml/min)}}$$

In man there is difficulty in applying direct *Fick method* during heavy exercise. So the other method, viz., Dye method is preferred:

1. **Fick principle using O_2.** (*a*) Total O_2 consumption per minute is determined with Douglas bag. (*b*) O_2 content of arterial blood is calculated from Hb%, assuming 95% saturation. (*c*) A sample of mixed venous blood is collected from the right atrium by introducing a fine rubber catheter into the cubital vein and pushing it gradually into the right atrium (Cournand's method) and its O_2% is determined.

From these data minute volume can be calculated as shown above.

2. **Fick principle using CO_2.** *This method can be readily adopted at the bedside.* The steps are as follows: (*a*) CO_2 output per minute is determined with Douglas bag. (*b*) Alveolar air is collected and its CO_2 tension —which is identical with that of arterial blood—is determined. (*c*) Alveolar air is again collected after holding the breath for 5 seconds. The CO_2 tension of this air is same as that of venous blood.

CO_2% of arterial and venous blood is then determined from the CO_2 dissociation curves. From these data minute volume can be calculated.

Physical method. Another method of determining cardiac output in human beings is the Ballistocardiography which was originally devised by Henderson and later on modified by Starr and his associates. The method is based on the principle of Newton's third law of motion—"every reaction has an equal and opposite reaction." **Ballistocardiogram** is a record of the recoil of the body caused by the movement of heart and blood within it in opposite direction. It can be recorded by allowing subject to lie on a suitably suspended table. The more convenient method is the recording of the movement of a steel rod kept on the stretched legs while lying supine on a fixed table. The movement of the rod can be recorded by suitable sensitive electronic instrument. The normal Ballistocardiogram shows following distinct waves : H I J K L M N, of which H I J are the systolic waves. H wave is the small positive wave and occurs due to headward movement of the body in response to footward movement of the heart and blood within it during isometric phase of systole. I wave is the negative wave and is inscribed due to footward

movement of the body in response to ejection of blood into the aorta during ejection phase. J wave is a large positive wave occurring during headward movement of the body due to rapid flow of blood through descending aorta (Fig. 212). The stroke volume can be calculated by applying the formula:

$$\text{Stroke volume} = = 7 \sqrt{\tfrac{2}{3} (I+J) AC}$$

where I and J are areas of the waves, A is the diameter of the aorta and C is the duration of cardiac cycle. Stroke volume multiplied by the heart rate gives the minute output. This method is not accurate.

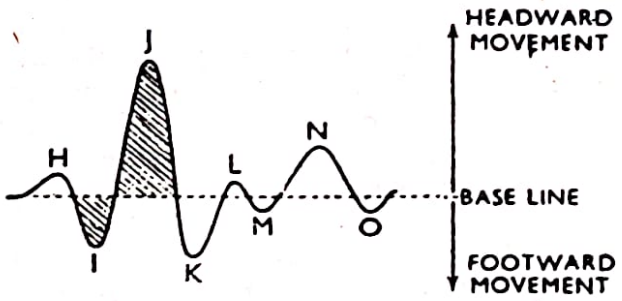

Fig. 212. Ballistocardiogram.

Factors influencing cardiac output

1. **Muscular exercise.** In heavy exercise the output may be 30–40 litres, i.e., 6–10 times the normal minute volume (stroke volume 170–200 ml; heart rate 150–180 per minute).
2. **Posture.** The minute volume is greater in the recumbent posture than in standing, because gravity retards venous return in the latter.
3. **Fever, hyperthyroidism, excitement** (10–25%), adrenaline, ingestion and digestion of food (10–20%), anoxia, CO_2 excess, intravenous (I.V.) saline, pregnancy (45–85% at full term), etc.,—increase cardiac output.
4. **Hypothyroidism, haemorrhage, shock, heart-failure,** etc., reduce cardiac output.
5. **Sleep.** May reduce slightly but usually not much.

BLOOD PRESSURE

Blood pressure is the lateral pressure exerted by blood on the vessel walls while flowing through it.* Four terms are in common use: (*a*) **Systolic pressure** (S. P.)—the maximum pressure during systole. (*b*) **Diastolic pressure** (D. P.)—the minimum pressure during diastole. (*c*) **Pulse pressure** (P. P.)—the difference between systolic and accepted diastolic pressure. (*d*) **Mean pressure** (M. P.)—It is roughly the arithmetic mean of the diastolic and the systolic pressures. But a close approximation to the mean pressure may be obtained by adding the diastolic pressure with *one-third* of the pulse pressure**. In true sense it is the level of the line halving area between the pulse wave contour and the diastolic pressure level.

In adults the relation between the three pressures is as follows: S.P./D.P./P.P.=3/2/1, viz., if systolic pressure be 120, diastolic pressure should be 80 and pulse pressure 40 mm of Hg.

Basal blood pressure. As blood pressure differs from an individual to another one and under different circumstances it varies in the same individual, it is permissible to use the term **normal range of blood pressure.** When an individual is with the least possible amount of strain or stress, **basal blood pressure** is generally considered. It may also be

* *Lateral pressure* is that pressure when force is exerted at right angles to the direction of flow at any point within a tube filled with a circulating fluid. *Resistance* is opposition to force.

** Sometimes, the M.P. is stated as the D.P. *plus* one-half of the P.P.

regarded as the lowest pressure necessary in maintaining blood flow sufficient for needs of the body. When a subject is in reclining state, 5–6 hours after last meal, in a comfortably warm room, after resting for at least 30–40 minutes and with a mind at possible ease, the basal pressure is obtained. In adult males, the average systolic pressure is 125–130 mm of Hg ± 15 (viz., from 110–145 mm of Hg) and average diastolic pressure, 70–90 mm of Hg. Although it is constant in a given individual, yet basal pressure varies in different ones with the following factors:

Physiological variations. (1) *Age*—Blood pressure rises with age. During infancy, the systolic pressure is from 70–90 mm of Hg; childhood, 90–110 mm of Hg; puberty, 110–120 mm of Hg; old age, 140–150 mm of Hg.* At any age, a systolic pressure persistently above 150 mm of Hg and a diastolic pressure above 100 mm of Hg should be accepted as high. On the other hand, systolic pressure below 100 mm of Hg and diastolic below 50 should be taken as low in the adults. Recent observations indicate that the pressure which is reached in adolescence does not normally rise with age any more.

The average systolic pressure is 110–120 mm of Hg and diastolic pressure 70–80 mm of Hg. The normal upper limits of systolic and diastolic pressures are placed at 140 and 90 mm of Hg respectively.

AVERAGE BLOOD PRESSURE AND STANDARD DEVIATIONS IN APPARENTLY HEALTHY PERSONS (assuming *diastolic end point is disappearance of sound*)

Age group	MALES		FEMALES	
	Systolic	*Diastolic*	*Systolic*	*Diastolic*
20–29	124·0 ± 13·2	77·0 ± 9·5	116·5 ± 11·6	73·0 ± 9·4
30–39	126·5 ± 13·9	79·5 ± 10·0	122·0 ± 14·0	76·5 ± 10·4
40–49	129·5 ± 16·0	81·5 ± 10·2	129·0 ± 18·3	81·0 ± 11·1
50–59	136·5 ± 19·0	83·5 ± 11·4	138·0 ± 21·4	84·0 ± 12·0
60–69	142·5 ± 23·5	84·0 ± 11·2	149·0 ± 25·7	85·0 ± 13·4
70–79	145·5 ± 24·0	81·5 ± 14·1	158·5 ± 26·0	84·5 ± 14·2
80–89	145·0 ± 25·0	80·5 ± 12·4	155·5 ± 28·0	82·5 ± 15·2

(2) *Sex*—In females both systolic and diastolic pressures are slightly lower than in males up to the age of 45–50 years.

(3) *Build*—The systolic pressure is usually high in obese person. In most of the overweight persons the blood pressure is found to be high.

(4) *Exercise*—In strenuous exercise the systolic pressure rises and may reach even up to 180 mm of Hg. In moderate exercise there is slight rise of systolic blood pressure.

(5) *Posture*—The diastolic pressure is slightly higher in the standing position. In the recumbent position the diastolic pressure is lower than in the standing or in the sitting position.

(6) *Sleep*—The systolic pressure falls by about 15 to 20 mm of Hg during sleep.

(7) *After ingestion of meals*—There is a slight rise of systolic pressure.

(8) *Emotion* or *Excitement*—It causes increase of systolic pressure.

* To obtain a rough estimate of the systolic pressure of a particular subject, several formulae are often used by clinicians. They are as follows: (1) 100 + age; (2) 100 + 2/3rds the age; (3) 90 + age, etc.

Significance of blood pressure. Systolic pressure undergoes considerable fluctuations. Excitements, exercise, meals, etc., increase it, while sleep, rest, etc., diminish it. The height of systolic pressure indicates: (1) The extent of work done by heart. (2) The force with which the heart is working. (3) The degree of pressure which the arterial walls have to withstand.

Diastolic pressure undergoes much less fluctuations in health and remains within a limited range. Increase of diastolic pressure indicates that the heart is approaching towards its failure. Consequently, variations of diastolic pressure are of greater prognostic importance than those of systolic. *Diastolic pressure is the measure of peripheral resistance.* It indicates the constant load against which heart has to work.

Pulse pressure generally varies directly as the stroke volume. But this quantitative relation may not be true in all cases.

The **normal function** of blood pressure is (1) to maintain a sufficient pressure head to keep the blood flowing; (2) to provide for the motive force of filtration at the capillary bed—thus assuring nutrition to the tissue cells, formation of urine, lymph and so on.

From the above considerations, it is seen that, the height of blood pressure gives correct informations about the state of the circulatory system as a whole and also about the functional condition of the tissue cells and organs.

Measurement and recording of blood pressure

Arterial blood pressure can be measured by two methods: (1) direct and (2) indirect. In **direct method,** the artery is exposed and an arterial cannula of which one tapering end is inserted directly into the lumen of the exposed vessel and the other end is connected to the U-shaped mercury manometer that shows the actual blood pressure in mm of Hg. As the mercury column in one limb (that has direct contact with the blood vessel) descends and the other limb of the U-tube ascends, the value in the scale will be doubled so as to get the actual blood pressure. For convenience it is generally considered to be 1 mm in the scale equivalent to 2 mm of Hg of pressure. Before recording the blood pressure, the mercury levels in both limbs of the U-tube must be adjusted to the 0 mark of the scale. For recording pressure a floating stylus with a writing pointer that marks on the smoked paper may be used (Fig. 213). This method is only suitable in animals and gives the idea about mean pressure. Due to high inertia of the mercury, the blood pressure changes associated with cardiac cycle are damped. Respiratory undulation of mean pressure waves are clearly seen in this direct method.

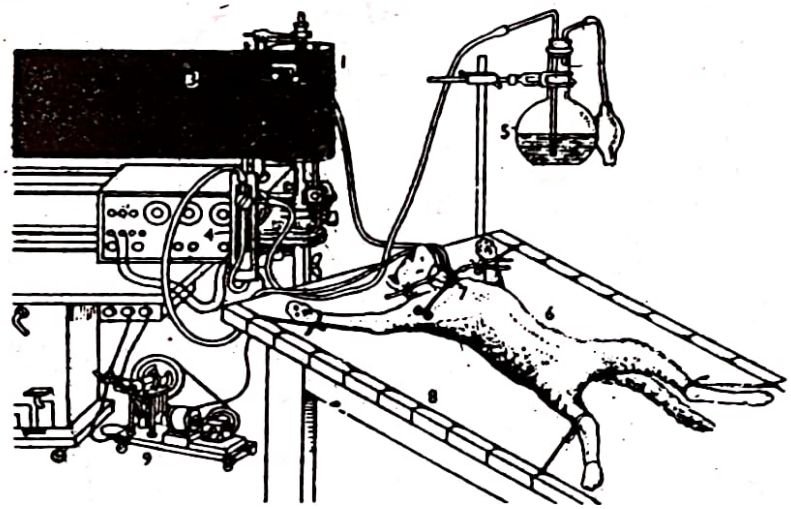

Fig. 213. Direct method for measurement of arterial blood pressure. 1=Kymograph. 2=floating stylus for blood pressure recording. 3=stylus for respiration recording. 4=mercury manometer. 5=reservoir. 6=animal (cat). 7=artery cannula. 8=operation table. 9=respiratory pump.

In **indirect method,** the pressure may be measured without any surgical procedure

regarded as the lowest pressure necessary in maintaining blood flow sufficient for needs of the body. When a subject is in reclining state, 5–6 hours after last meal, in a comfortably warm room, after resting for at least 30–40 minutes and with a mind at possible ease, the basal pressure is obtained. In adult males, the average systolic pressure is 125–130 mm of Hg ± 15 (viz., from 110–145 mm of Hg) and average diastolic pressure, 70–90 mm of Hg. Although it is constant in a given individual, yet basal pressure varies in different ones with the following factors:

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From the above considerations, it is seen that, the height of blood pressure gives correct informations about the state of the circulatory system as a whole and also about the functional condition of the tissue cells and organs.

Measurement and recording of blood pressure

Arterial blood pressure can be measured by two methods: (1) direct and (2) indirect. In **direct method,** the artery is exposed and an arterial cannula of which one tapering end is inserted directly into the lumen of the exposed vessel and the other end is connected to the U-shaped mercury manometer that shows the actual blood pressure in mm of Hg. As the mercury column in one limb (that has direct contact with the blood vessel) descends and the other limb of the U-tube ascends, the value in the scale will be doubled so as to get the actual blood pressure. For convenience it is generally considered to be 1 mm in the scale equivalent to 2 mm of Hg of pressure. Before recording the blood pressure, the mercury levels in both limbs of the U-tube must be adjusted to the 0 mark of the scale. For recording pressure a floating stylus with a writing pointer that marks on the smoked paper may be used (Fig. 213). This method is only suitable in animals and gives the idea about mean pressure. Due to high inertia of the mercury, the blood pressure changes associated with cardiac cycle are damped. Respiratory undulation of mean pressure waves are clearly seen in this direct method.

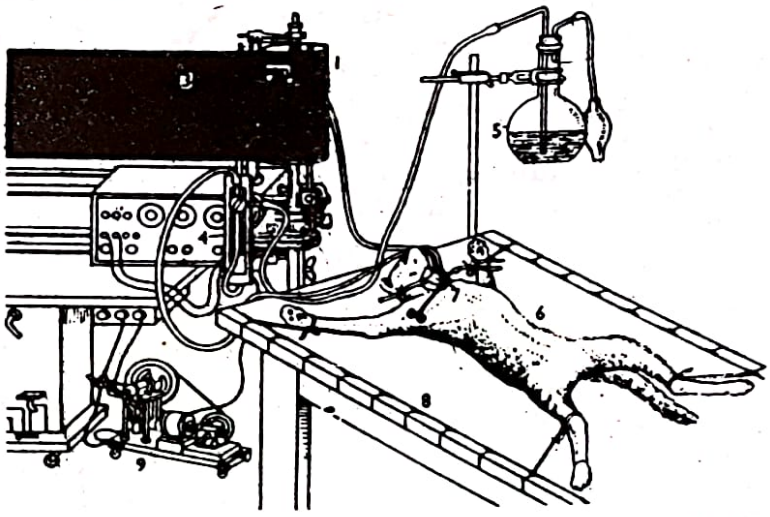

Fig. 213. Direct method for measurement of arterial blood pressure. 1=Kymograph. 2=floating stylus for blood pressure recording. 3=stylus for respiration recording. 4=mercury manometer. 5=reservoir. 6=animal (cat). 7=artery cannula. 8=operation table. 9=respiratory pump.

In **indirect method,** the pressure may be measured without any surgical procedure

and thus it is very convenient clinically in human being. Riva-Roci (1896) first introduced this indirect method and afterwards **Korotkoff** (1905) introduced a convenient method by which the systolic and diastolic pressures could be ascertained only through listening to a sound. This is the standard method of recording blood pressure all throughout the world.

In this method commonly the pressure of the brachial artery is measured. The instrument used is known as *Sphygmomanometer*. Three methods: (*a*) **oscillatory,** (*b*) **palpatory,** (*c*) **auscultatory.**

Oscillatory method. Inspection of oscillation in spring gauge or mercury manometer is the basis of this method. In this method a pressure cuff is wrapped over the brachial artery and the oscillations that are produced by the pulsations, are observed. The instrumeut is always kept at the heart level. When the cuff pressure is increased and raised above the systolic pressure, the oscillations disappear, but on releasing the pressure gradually, the oscillations become larger and prominent. The pressure head at which the larger oscillations are seen, is considered as systolic pressure. But on further release of pressure, the oscillations become smaller and disappeared. The pressure at which the oscillation just becomes smaller or disappears, is known as diastolic pressure.

Palpatory method. The instrument is kept at the level of the heart and the cuff is tied round the upper arm. Pressure is raised to 200 mm of Hg and then gradually released. When the pulse just appears at the wrist, the pressure is noted. This is the systolic pressure. This method is not accurate. By this method the diastolic pressure cannot be determined.

Auscultatory method. The instrument is kept at the level of the heart and the cuff is tied round the upper arm. Pressure is raised to 200 mm of Hg and then gradually released. Variations of sounds are heard with a Stethoscope placing its chest piece on the brachial artery, a little below the cuff. *The sounds are heard due to occurrence of turbulence in the flow of blood through the narrowed blood vessels when the manometric pressure just coincides with the systolic blood pressure.* Due to giving air pressure in the cuff, the vessel is pressed and blood flow is obliterated. But while releasing the air pressure gradually, blood just begins to flow through the narrowed blood vessels and the pattern of flow is changed from streamline flow (silent) to turbuient flow (noisy). When the pressure is further released, normal streamline flow sets in and the sound is no longer heard. At this point manometric pressure coincides with the diastolic blood pressure. So, as the pressure is released the following variations of sounds are heard: *First phase*—sudden appearance of a clear *tapping sound. This indicates systolic pressure.* It persists while the pressure falls through 15 mm of Hg. *Second phase*—the tap sound is replaced by a *murmur* persisting for another 15 mm of Hg. *Third phase*—

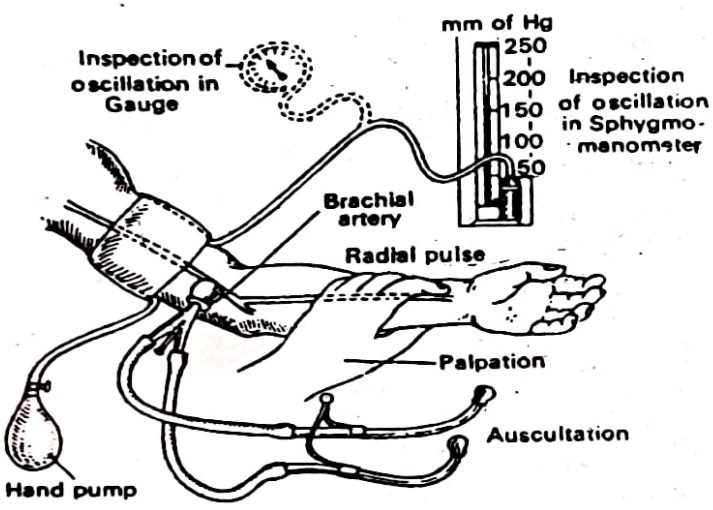

FIG. 214. Measurement of arterial blood pressure in human beings (after Harris).

the murmur is replaced by a clear loud *gong sound* lasting for the next 20 mm of Hg. *Fourth phase*—the loud sound suddenly becomes *muffled* and rapidly begins to fade. *This point indicates diastolic pressure. Fifth phase*—absence of all sounds.

Venous pressure. It is the pressure which is exerted by the blood within the veins. Average venous pressure of human being in recumbent position is about 60–120 cm of H_2O. The venous pressure can be measured by inserting a needle directly into the anticubital vein and by connecting the needle to a water manometer (FIG. 215). Venous pressure is a valuable index in determining the efficiency of the heart.

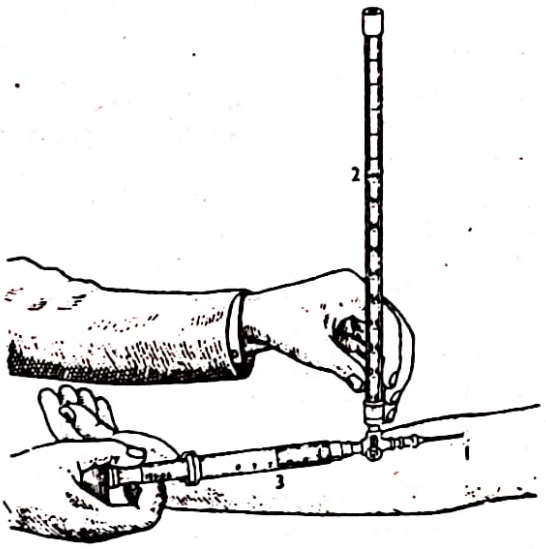

FIG. 215. Use of water manometer for measurement of venous pressure. 1=anticubital vein. 2=manometer. 3=syringe.

Factors controlling arterial blood pressure

1. **Pumping action of the heart.** Effectual contraction of the heart is the main factor for controlling the cardiac output, blood pressure and flow within the blood vessel. Because in each effectual contraction of the ventricle certain amount of blood is ejected out into the aorta. The driving force of blood is mainly created by the pumping action of the heart. The efficiency of the heart is considered upon how much amount of blood is driven out by the heart into the aorta in each beat.

2. **Cardiac output.** Alterations of cardiac output will alter blood pressure. Cardiac output depends upon venous return, force and frequency of heart beat. Blood volume affects blood pressure directly, by mainly modifying the cardiac output.

3. **Peripheral resistance.** *It is the resistance which blood has to overcome while passing through the periphery. The chief seat of peripheral resistance is the arterioles* and to a smaller extent the capillaries (*vide* below). Peripheral resistance depends on the following: (1) Velocity of blood, (2) Viscosity of blood, (3) Elasticity of arterial walls, (4) Lumen of the blood vessels. Resistance is directly proportional to the first two and inversely to the last two factors.

(1) *Velocity*—A rapidly flowing stream will have more frictional effect than a slower one. Hence, pressure is high in the aorta but low in the capillaries. (2) *Viscosity*—Other factors remaining constant, a more viscid blood will have a higher friction than a lesser one. For this reason, plasma transfusion is sometimes more effective to maintain blood pressure than ordinary saline. (3) *Elasticity*—Due to elastic properties, the arteries can dilate and accommodate considerable amount of blood with relatively less rise of blood pressure. In old age, the arterial walls become stiff. Hence, blood pressure rises. (4) *Lumen of the vessel*—Peripheral resistance is inversely proportional to the lumen of the vessels. In other words, smaller the vessel, higher will be the resistance. One should expect therefore that the capillaries, having the smallest lumen, should have the highest pressure.

But this is not the case. Because the velocity of blood being lowest in the capillaries, the frictional effect is very low. Hence, the pressure is also low. The seat of peripheral resistance is found to be chiefly in the arterioles, where the velocity is fairly high and the lumen is narrow.

$$\text{Peripheral resistance} \propto \frac{\text{Mean arterial pressure}}{\text{Cardiac output}}.$$

Mean arterial pressure can be expressed in *dynes* per square centimetre by multiplying the pressure in mm of Hg by 1,332.

4. **Elasticity of the arterial walls.** In normal diastolic pressure arterial walls are stretched but due to the presence of elastic tissues in their walls, they tend to recoil. Due to elasticity of the arterial walls the blood flow is pulsatile in the arteries. In the capillaries and venules the flow is continuous. In old age the expansion of the arterial walls becomes limited due to sclerotic changes and the blood pressure rises.

5. **Blood volume.** Increase in blood volume will raise both the systolic and diastolic blood pressures due to the increased quantity of blood in the arterial system and greater stretching of the arterial walls.

6. **Viscosity of the blood.** Alteration in blood viscosity will affect the diastolic pressure by its effect on the peripheral resistance. The intramolecular friction is greater when the viscosity is high.

Poiseuille's law. In 1841 the French Physician J. L. M. Poiseuille studied the factors regulating the flow of viscous fluids through the capillary tubes. He showed that **resistance to blood flow** in any blood vessel proportionally varies directly with the viscosity of the blood and also with the length of the blood vessel, and inversely with the *fourth power* of the radius of the blood vessel. It can be represented by the following formula:

$R = \frac{\eta l}{r^4} \times \frac{8}{\pi}$, where R stands for resistance to blood flow, η for viscosity of blood, l for length of blood vessels, r for radius of the blood vessel, 8 for Hagen's integration and π factor for a cylindrical tube.

Taking this value for resistance in the formula it is found that blood flow proportionally varies directly with the blood pressure and the fourth power of the radius of the blood vessel, and inversely with the viscosity of the blood and the length of the blood vessel. The following formula, known as *Poiseuille's law*, expresses the above relations.

$BF = K\frac{BP}{R}$ or $BF = BP \times \left(\frac{\pi}{8}\right) \times \left(\frac{1}{\eta}\right) \times \left(\frac{r^4}{l}\right)$, where BF stands for blood flow, BP for blood pressure, r for radius of the blood vessel, η for viscosity of blood, l for length of the blood vessel and π for 3·14. This law is not applicable when the arterial pressure falls below 20 mm of Hg.

Adjustment of blood pressure

In the normal individual the constancy of the internal environment is being adjusted by the well-organised controlling system—which is called *Milieu intérieur* after Claude Bernard and *Homoeostasis* after Cannon. Adjustment of blood pressure, according to the needs of the body, may be carried out by the several complex reflexes whose centres are lying in the *cerebral cortex, formatio reticularis, hypothalamus, medullary and spinal vasomotor centres*. The (A) efferent and (B) afferent pathways constituting the above reflexes are lying within the *sympathetic and parasympathetic nervous systems* whose activities are modified by the hypothalamus and other centres.

A. **Efferent pathways of this self-adjustment or homoeostasis of blood pressure** are the vagi and the sympathetic nerves which control the blood pressure by (*a*) *modifying the cardiac activity*, (*b*) *altering the cardiac output*, and (*c*) *altering the lumen of the blood vessels*. The relative activities of the vagi and the sympathetic of the efferent pathways are under the control of *vasomotor systems*, which are described below:

Vasomotor system. This system consists of (1) **vasomotor centre**, (2) **vasoconstrictor nerves**, (3) **vasodilator nerves.** They supply vasomotor nerves—mainly to the arterioles but to some extent to the capillaries and venules. This vasomotor centre is highly developed in higher animals and human beings. In infants and children it is imperfect. By regulating the radius of the blood vessels this system takes part in adjusting blood pressure and blood supply to a particular part. It also plays an immense role in heat regulation.

Vasomotor centre (V.M.C.). Vasomotor centre is situated on the floor of the fourth ventricle in the reticular formation at the level of the **calamus scriptorius.** It extends from the lower part of the pons to the obex and forms a diffuse network of neurones. After section of the brain stem at the level of the calamus scriptorius there is fall of blood pressure. There are practically two areas in the reticular formation of the medulla:

(*a*) *Pressor centre*—which causes rise of blood pressure.

(*b*) *Depressor centre*—which causes fall of blood pressure.

The depressor centre is not the *vasodilator* centre. This centre causes inhibition of the vasoconstrictor tone. The depressor centre relays the inhibitory impulses to the pressor centre. Pressor and depressor centres form one functional unit and it is defined as the vasomotor centre. The vasomotor centre discharges impulses which pass down the lateral white column of the spinal cord in the cervical, thoracic and lumbar segments of the spinal cord and form synaptic connections with the lateral horn cells of the spinal cord.

Vasomotor reflexes. (1) Depressor reflex. (2) Pressor reflex.

Depressor reflex. Blood pressure falls due to diffuse dilatation of the arterioles. Rise of blood pressure stimulates the **baroreceptors** of the carotid sinuses and aortic arch, and causes slowing of the heart and arteriolar dilatation. The vasodilatation is due to inhibition of vasoconstrictor effect of the sympathetic.

Pressor reflex. Blood pressure rises due to diffuse constriction of the arterioles. Diminution of blood pressure fails to stimulate the baroreceptors of the carotid sinuses and aortic arch, and the parasympathetic inhibitory tone over the heart and blood vessels is withdrawn. Blood pressure is raised reflexly through overactivity of the sympathetic. Vasoconstriction of the arterioles is due to activity of the vasoconstrictor centre. Reflex vasoconstriction also occurs due to stimulation of **chemoreceptors** during the fall of blood pressure.

Control of V.M.C. Vasomotor centre is under the superior control of cerebral cortex and hypothalamus (Fig. 216). Factors influencing V.M.C. have been described as follows:

1. **Higher centre** (including Hypothalamus). *Emotion* generally stimulates, causing vasoconstriction. But *shock* may depress the centre—leading to a sudden fall of blood pressure and fainting (vasovagal attacks).

2. **Respiration.** During inspiration systemic blood pressure is generally decreased but increased during expiration. This is due to the decrease of left ventricular cardiac output during inspiration. Reverse is the effect during expiration. There is no evidence of direct respiratory centre—effect on vasomotor centre.

3. **CO_2 excess.** Excess stimulates. The action is mainly on the centre but partly reflexly through the sino-aortic nerves.

4. **O_2 lack.** Generally stimulates V.M.C. The effect is mainly reflex through the sino-aortic nerves and slightly direct on the centre.

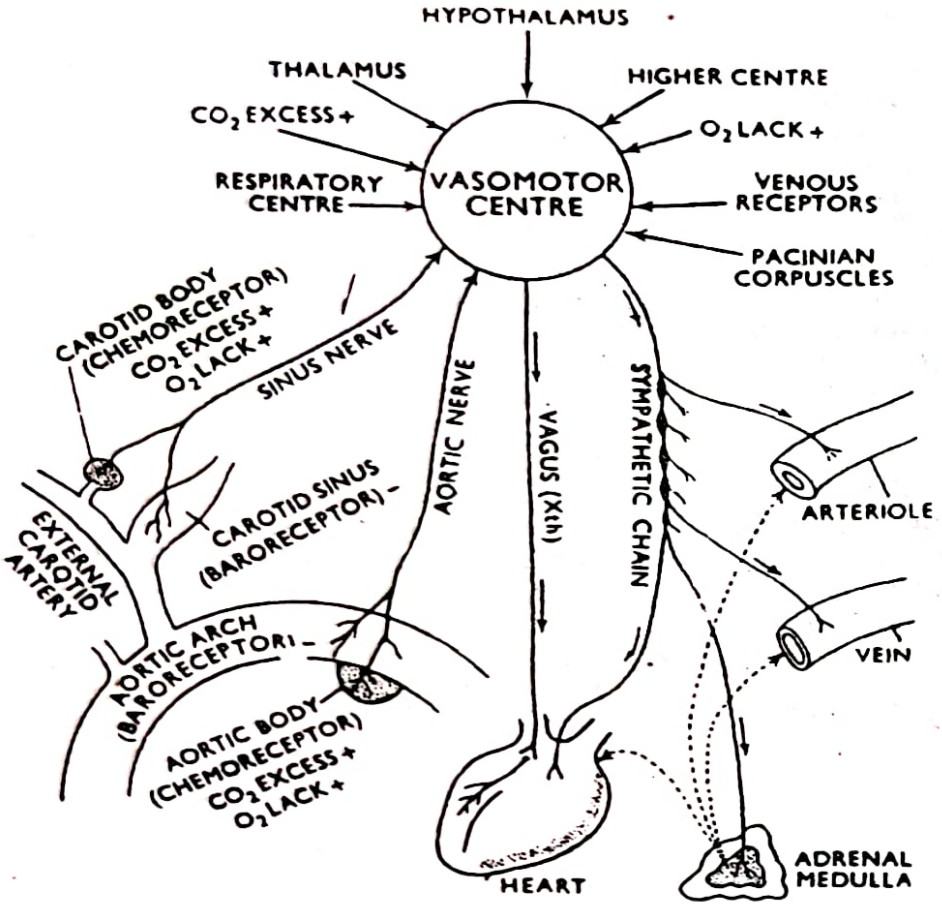

FIG. 216. Diagrammatic representation of different factors that influence the vasomotor centre.

5. **Sino-aortic nerves.** Variations of blood pressure, CO_2 tension, O_2 tension, etc., reflexly regulate the activity of the vasomotor centre through the sino-aortic nerves. Normally, a stream of inhibitory impulses is carried up by these nerves depressing the vasomotor centre. When blood pressure rises, V.M.C. is depressed, vasodilatation occurs and further rise of blood pressure is checked. When blood pressure falls, the centre is released causing vasoconstriction and raising blood pressure. [Sino-aortic nerves also control cardiac centre, respiratory centre and adrenaline secretion.] [*Vide* pages 306–07.]

6. **Other afferents.** Local vasomotor tone is altered by afferent nerves originating from different baroreceptor and chemoreceptor areas, distributed all throughout the body. The baroreceptors are located in the *right atrium*, in the *left atrium and left ventricle*, in the *pulmonary arch of aorta*, in the *junction of the superior thyroid artery and common carotid artery, junction of the subclavian artery and common carotid artery*, and *all throughout common carotid artery in between the superior thyroid artery and subclavian artery, mesenteric blood vessels* (Pacinian corpuscles), *thoracic arch of the aorta* and in the *central vein* (venous receptor). The chemoreceptors are located in the ventricular cavity and all throughout the blood vessel wall. *Reactive hyperaemia* is the consequence of local chemoreceptor activity on the blood vessel wall by the accumulated metabolites. Heat dilates and cold constricts the skin vessels reflexly.

Vasoconstrictor nerves. The fibres pass along the sympathetic outflow from the first thoracic to the second lumbar segments. Brief details are as follows:

1. *To the skin and muscles*—pass out through the grey rami communicantes—to the mixed spinal nerves—and finally distributed through ordinary motor and sensory nerves. The distribution is strictly unilateral, stopping sharply at the midline.
2. *To the head and neck*—come from the first to the fourth thoracic segments—enter the superior cervical ganglion from which postganglionic fibres arise and pass along the carotid artery and its branches.
3. *To the fore limbs*—arise from the fourth to tenth thoracic segments—enter the stellate ganglion from which the postganglionic fibres arise and pass along the spinal nerves and supply the blood vessels.
4. *To the hind limbs*—arise from the eleventh thoracic to the second lumbar segments—relay in the lower lumbar and upper sacral ganglia, the postganglionic fibres accompany the nerves of the sacral plexus.
5. *To the abdominal viscera*—from the lower thoracic and upper two lumbar segments—pass through the splanchnic nerves to coeliac ganglion—the postganglionic fibres pass along the blood vessels.
6. *To the thoracic viscera*—*Heart* receives constrictor fibres through the vagus; *lungs* from the sympathetic.

Vasodilator nerves. There are three types of vasodilator nerves: (1) **parasympathetic** (craniosacral), (2) **sympathetic,** (3) **antidromic fibres** of the posterior spinal root.

1. **Parasympathetic vasodilators**

(*a*) Cranial: (*i*) Chorda tympani—to the submaxillary or submandibular gland. (*ii*) Lesser superficial petrosal—to the parotid gland. (*iii*) Lingual—to the vessels of tongue.

(*b*) Sacral: Nervi erigentes—to the vessels of genitalia.

2. **Sympathetic vasodilators**

Sympathetic fibres are mostly vasoconstrictor in nature. But some vasodilators are also present. For instance, (*a*) the dilator fibres of the coronary vessels come through the sympathetic. (*b*) Sympathetic dilator fibres have been demonstrated in the peripheral nerves in human beings. (*c*) Stimulation of the last anterior thoracic root produces dilatation of the kidney vessels. (*d*) Stimulation of the right splanchnic nerve sometimes causes vasodilatation and fall of blood pressure.

3. **Antidromic vasodilators** in the posterior spinal root (FIG. 217). When posterior spinal root is cut, distal to the ganglion and the peripheral end is stimulated—although the nerve is afferent, yet the vessels in the periphery—both skin and muscles—dilate **(axon reflex).** In the

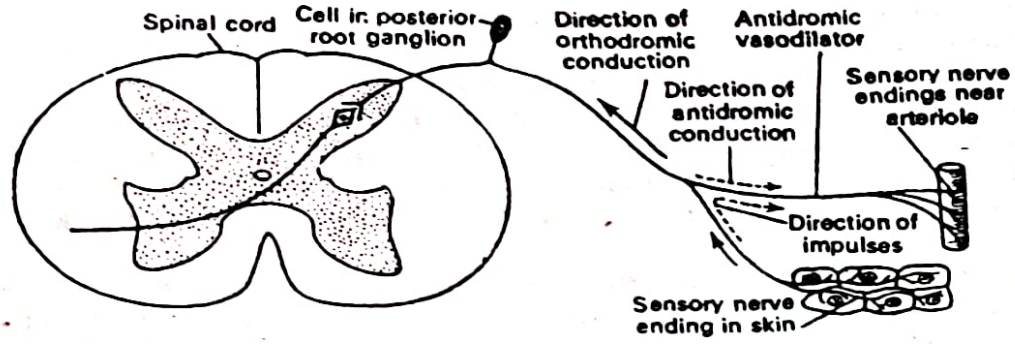

FIG. 217. Antidromic nerve fibres in the posterior nerve root.

skin, it is due to liberation of histamine and as such produces the typical triple response: dilatation, flare and wheal. In the muscle it liberates acetylcholine and thereby causes vasodilatation.

B. **Afferent pathways** are lying in two sets of receptors that carry the information of the instantaneous circulatory status to the centre. These sensory receptors are (*i*) **chemoreceptors** and (*ii*) **baroreceptors** distributed all throughout the cardiovascular system. The relative roles of the different afferent pathways have been described under separate headings, viz., (1) **sino-aortic mechanisms** controlling systemic blood pressure and flow, and (2) **vascular receptors** other than sino-aortic—controlling mostly the local blood pressure and flow.

I. ROLE OF SINO-AORTIC MECHANISM IN THE REGULATION OF NORMAL BLOOD PRESSURE. From the above, it is evident that blood pressure can be adjusted according to the needs of the body in various ways. Of all the factors, the sino-aortic mechanism plays the chief role. The sino-aortic mechanism is carried on by *baroreceptors* and *chemoreceptors*. This mechanism regulates blood pressure by adjusting the heart rate, vasomotor centre, and secretion of adrenaline and noradrenaline. It also adjusts respiratory centre in such a way that the functions of heart and respiration may run parallel.

SINO-AORTIC MECHANISM

(*a*) **BARORECEPTORS.** This includes *carotid sinus* and *aortic arch* (FIG. 218).

Carotid sinus. It is a dilatation at the root of internal carotid artery, often involving the common carotid. The exact location varies in different species. The wall of the sinus is thinner due to less muscle fibres in the media. In the deeper parts of adventitia, an extensive network of afferent nerve fibres is present. The fibres end in free nerve terminals and characteristic minisci. These pressor receptors are sensitive to stretch (*distortion effect*) being stimulated by rise of blood pressure.

The **sinus nerve** (afferent) arises from the carotid sinus and carotid body, passes along the glossopharyngeal nerve and ends in the medulla in close relation with respiratory, cardiac and vasomotor centres.

Aortic arch. Afferent nerves and stretch receptors—similar to those

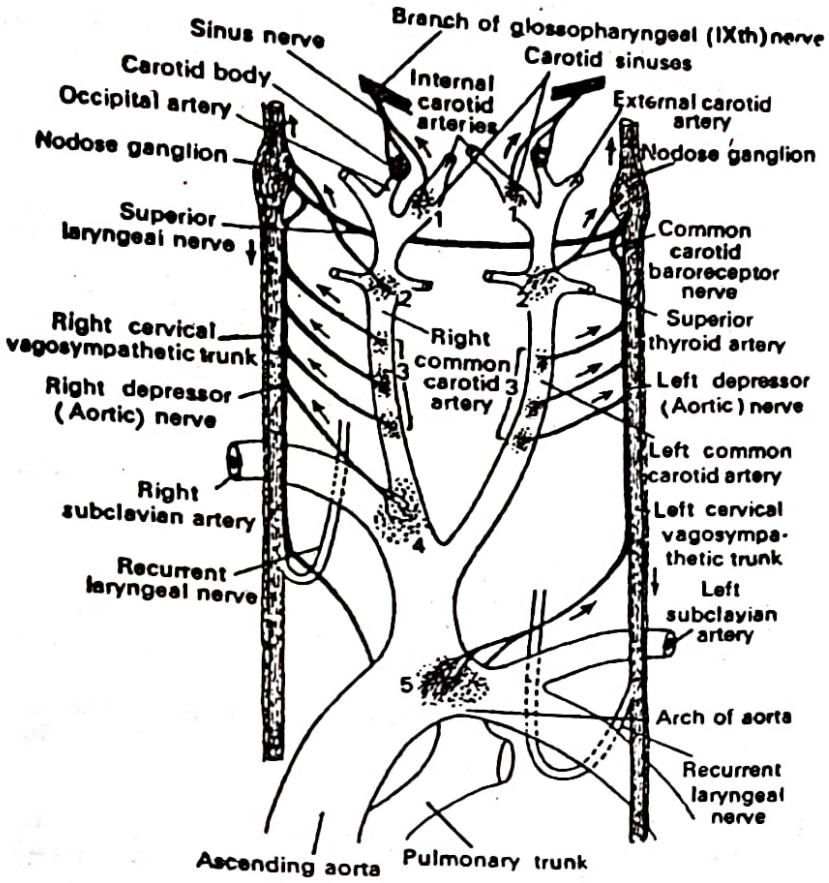

FIG. 218. Diagram represents the distribution of different baroreceptors on the walls of the blood vessels. 1 = carotid sinus. 2 = baroreceptors at the junction of the superior thyroid artery and common carotid artery. 3 = baroreceptor areas on the wall of the common carotid arteries. 4 = baroreceptors at the junction of the subclavian artery and common carotid artery. 5 = aortic arch baroreceptors.

in the carotid sinus—are also present in the adventitia of aortic arch, the roots of great vessels and even the adjoining parts of left ventricle. They serve the same function as the carotid sinus.

Aortic nerve. This nerve arises from the aortic body, the aortic arch and the basal part of left ventricle. It is a purely afferent nerve. Its course varies in different species but in human being it mostly passes in the vagus. Like the sinus nerve it ends in medulla being closely related to cardiac, vasomotor and respiratory centres.

(*b*) **CHEMORECEPTORS.** This includes *carotid* (G. *Karos* = sleep) *body* and *aortic bodies* (FIG. 220).

Carotid body (FIG. 219). It is a small nodule situated on the occipital artery, a branch of the external carotid artery very close to the carotid sinus. It consists of clumps of large polyhedral cells (*Glomus cells*), richly supplied with blood vessels and nerves. The vessels arise from the carotid artery. Some of the cells stain with chromic acid and belong to the chromaffin system but do not contain adrenaline. Numerous afferent nerve fibres surround the cell clumps and even the individual cells, and terminate in special *chemoreceptors*. They are sensitive to chemical changes in blood.

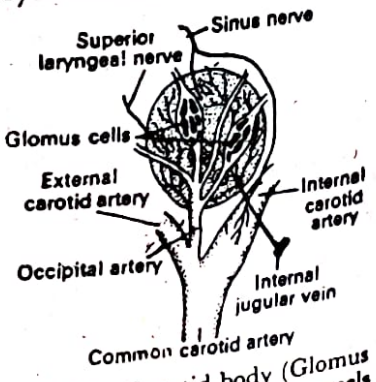

FIG. 219. Carotid body (Glomus caroticum) showing blood vessels and nerve supply.

Aortic bodies. Four groups of aortic bodies have been shown in cat (Howe, 1956). These are small nodular structures, supplied by a special blood vessel and situated (*i*) on the thorax between the pulmonary trunk and

ascending aorta, (*ii*) on the ventral surface of the root of the right subclavian artery, (*iii*) on the ventral surface of the root of the left subclavian artery, and also (*iv*) on the ventral surface of the aortic arch (FIG. 220). Afferent pathways from these chemoreceptor areas are lying in the aortic nerves and vagi. Their structures, nerve endings and functions are similar to those of carotid body.

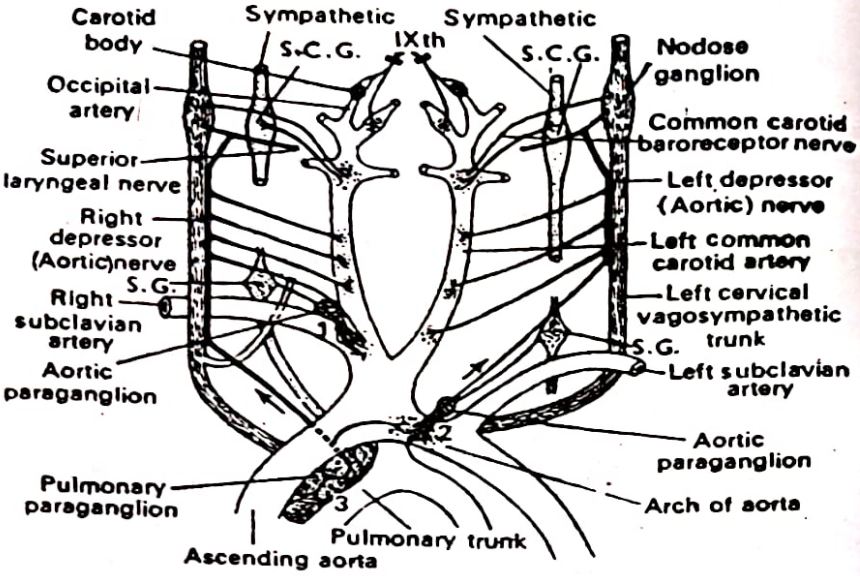

FIG. 220. Diagram represents the distribution of chemoreceptors at areas of the blood vessels. 1, 2, 3 are the chemoreceptor areas.

By perfusion experiments, the effects of chemical changes in blood, as brought about through the sino-aortic chemoreceptors, have also been studied in detail. It is seen that CO_2 excess, O_2 lack and increased H-ion concentration stimulate respiration (mainly), increase heart rate, produce vasoconstriction and raise blood pressure. After haemorrhage or in enfeebled circulation a rhythmic blood pressure wave (**vasomotor wave**) is often encountered. These vasomotor waves are due to chemoreceptor activities under such state. These waves were observed by Mayer (1876) and known as **Mayer's wave.** Following inactivation of chemoreceptors, these waves disappear completely.

Mechanisms of stimulation of chemoreceptors. It has been claimed that for the stimulation of chemoreceptor nerve endings, the liberation of acetylcholine plays as the chemical intermediary (*vide* Chemical regulation of respiration).

Experimental observations. To study the functions of the sino-aortic nerves various experiments have been performed. For instance, (1) *Section and stimulation* of the sinus and carotid nerves. (2) *Perfusion experiments* in which the carotid sinus region is isolated and perfused with blood or other fluids whose pressure and composition can be varied at will. (3) *Electrophysiological study* shows that in normal arterial pressure the sinus nerves discharge impulses, the frequency of which rises with systolic pressure and diminishes with diastolic pressure. Rise in systolic pressure increases the frequency of impulse discharge. (4) *Cross-circulation experiments*—Heymans and his associates have studied the functions of the carotid sinus and sinus nerve by cross-circulation experiments (FIG. 221). The carotid sinus of the second dog B was isolated (the nerve supply remaining intact) and perfused with the blood of the first dog A. When the arterial pressure of the dog A was raised the arterial pressure of the dog B was diminished. Again when the arterial pressure of the dog A was lowered, the arterial pressure of the dog B was increased by secretion of adrenaline as evidenced from splenic contraction in dog C which got adrenaline from

dog B through anastomoses of the suprarenal vein and the jugular vein. The followings are the complete observations:

On raising the pressure in the carotid sinus, the following reflex effects are produced: (1) Slowing of the heart rate. (2) Peripheral vasodilatation preferably in the splanchnic bed so as to increase the total vascular capacity. (3) Fall of blood pressure. (4) Diminished adrenaline secretion. (5) Slowing or stoppage of respiration. (6) Diminished tone in voluntary muscles. (7) Various changes in the viscera, viz., increased volume and movement of the stomach, decreased tone of urinary bladder, etc., caused by disturbed activity of the autonomic system. Fall of sinus pressure or section of the sinus nerve produces opposite effects. Stimulation of the central cut end of the sinus nerve also produces similar effects.

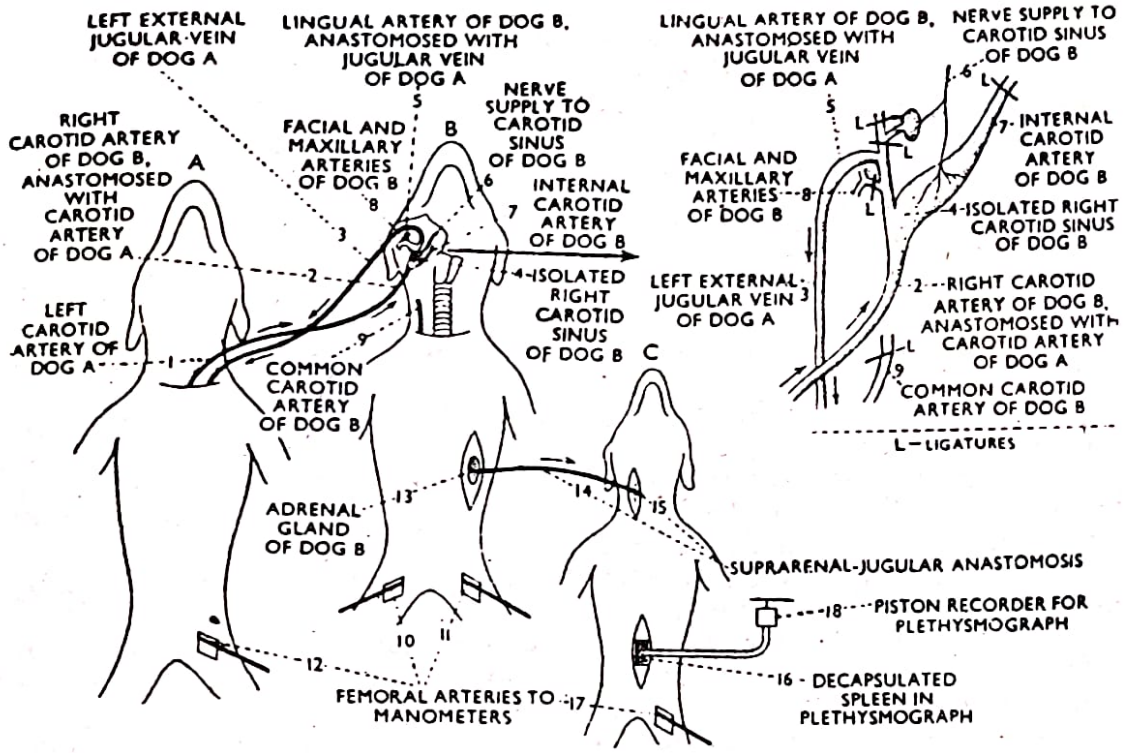

FIG. 221. Scheme of perfusion of the isolated carotid sinus of dog B, by dog A, and an anastomosis between the suprarenal vein of dog B and the jugular vein of dog C. The blood from dog A flows through the carotid sinus of dog B and back to dog A *via* anastomosis between the lingual artery of dog B and the external jugular vein of dog A (after *Heymans*).

Raising the aortic pressure causes the following effects: (1) Slowing of the heart rate—mainly due to the stimulation of cardio-inhibitory centre and partly to the inhibition of accelerator centre. (2) Inhibition of the vasomotor centre causing vasodilatation. (3) Depressed adrenaline secretion. (4) Fall of blood pressure. (5) Depressed respiration. Fall of aortic pressure or section of the aortic nerve—produces reverse effects. Stimulation of the central cut end of the aortic nerve produces similar effects. Sometimes stimulation of the central cut end of vagus or aortic nerve may raise blood pressure by reflex cardiac acceleration and vasoconstriction. This proves that these nerves also carry some pressor fibres.