

Journal of Clinical and Basic Cardiology

An Independent International Scientific Journal



Journal of Clinical and Basic Cardiology 2006; 9 (1-4), 37-40

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Blood Pressure Maxima in Humans

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The generation of pressure in the blood circulation is reconsidered. It is shown that there exists a maximum pressure in the blood circulation of any individual and that changes leading to this value being exceeded are likely contributory causes of heart attacks and strokes. The causes of increases and decreases in individual blood pressure values below this limit are discussed. J Clin Basic Cardiol 2006; 9 (online): 37–40.

Key words: blood pressure generation, blood pressure changes, related conditions

The origin of pressure in the blood circulation has been the subject of study for many decades and has given rise to various hypotheses and explanations [1–6]. From Pascal's Law the pressure in any column of liquid above ground level is uniform irrespective of any changes in the diameter of the column throughout the length. This pressure is known as hydrostatic pressure and is given by the formula

$$p = h \times r \times g,$$

where p is pressure, h is height, r is density, and g is the acceleration of gravity. The contraction of the heart is required to result in the generation of the pressure which equals or exceeds the hydrostatic pressure at the level of the head above ground level in the normal vertical stance. This pressure raises blood from the heart to the highest point of the head and from the feet to the heart level. This work demonstrates the calculation of the pressure capable of being generated by the heart plus the origin of changes in this pressure.

The Generation of Blood Pressure

For a human with a physical height of 169 cm (5 feet 6 inches), the downward pressure exerted by a column of blood at head height with a density equal to 1.025 g/ml (plasma density) is 126.3 mmHg. The downward pressure exerted by a column of the same liquid at the level of the heart at 110 cm above ground level in the same human is 82.15 mmHg. These calculated values are in good agreement with measured values for an individual with the characteristics used in the calculation e.g. systolic pressure 120 mmHg – diastolic 80 mmHg. The agreement is also good for individuals with other physical characteristics (Tab. 1). The heart is required to generate sufficient pressure to overcome the hydrostatic pressure at head level and raise the blood to this level and from the feet to the heart in order to create the circulation of blood. The pressure generated by the heart can be calculated using the Law of Laplace which states that the pressure expanding a membrane is related to the tension by the formula

$$p = 2T/r,$$

where p is pressure, T is tension, and r is the radius of the expanded membrane. The ascending aorta is the initial blood vessel of the circulation and any changes induced in the dimensions of this blood vessel by the operation of the heart are directly related to the expansion and contraction of the latter. A typical volume of blood injected into the ascending aorta by the heart (stroke volume) is 70 ml. At a pulse rate of 72 beats/min the injection occurs every 0.8 s. Typical dimensions of ascending aorta are 5 cm long and with a radius of 1.5 cm. Treating the ascending aorta as a cylinder gives a volume of 35.4 ml. Prior to the injection the ascending aorta is

filled with blood. The rapid injection of 70 ml of additional blood from the heart causes this vessel to expand momentarily giving a total volume of 105.4 ml. This means that the volume has increased by 2.97 times and the radius after injection is 2.59 cm. The radius has therefore increased 1.72 times on injection of the volume of 70 ml. This estimation makes the assumption that the cylindrical form is retained to a good approximation on injection of the volume of blood. The aorta is constructed from a flexible membrane and the expansion of this membrane results in the generation of a tension force (elastic recoil) which opposes the expansion. The factor T is the elastic force resisting the expansion of the aorta membrane. From this

$$r = 2T/p.$$

The expansion of the ascending aorta means that the value T has increased in proportion to the pressure generated by the injection of blood. The change in the value of the aorta radius is a measure of this increase in pressure. The injection of 70 ml of blood into the ascending aorta will produce a pressure at the heart level of 141.3 mmHg (1.72×82.15). This value demonstrates that the heart is capable of generating pressures in excess of that required to overcome the hydrostatic pressure at head level (126.3 mmHg). The pulmonary artery acts in the same manner. The result is confirmed by calculation of the maximum pressure which can be endured by most humans when subjected to increased pressure generated by increase in gravity. It is known from aviation research that a maximum increase of five times the force of gravity (5 g) can be endured by aviators before loss of consciousness. The heart level of an aviator with a physical height of 169 cm (5 feet 6 inches) is 40 cm above the seat level and the pressure at heart level is 29.2 mmHg. A gravity increase of five times is equal to a pressure of 145.9 mmHg at heart level. This exceeds the calculated pressure capable of being generated by the heart by such an individual as calculated above and confirms that the assumed expanded shape of the aorta is not unreasonable. The pressure generated at any instant is sensitive to the injected volume, e.g. a reduction in injected volume by 10 % to 63 ml changes the generated pressure from 141.3 mmHg to 137.1 mmHg and a 30 % reduction in injected volume changes the generated pressure from 141.3 mmHg to 126.9 mmHg. The measured values of blood pressure for an individual with the physical characteristics given above are less than the calculated maximum value. This demonstrates that the injected volume under normal circumstances is less than the maximum volume that the heart can deliver. The generated pressure produced in any individual is dependent on the injected volume, the volume of the ascending aorta and pulmonary artery and the actual height of the heart level.

Table 1. Calculation of diastolic and systolic blood pressures

Physical height		Atmosphere pressure		Pressure			
				Whole blood ⁺ at		Plasma [#] at	
				Heart level	Head level	Heart level	Head level
feet-ins	ins	cm	cm H ₂ O*	Diastolic mmHg	Systolic mmHg	Diastolic mmHg	Systolic mmHg
4' 6"	54	138.24	1041.4	69.51	106.93	66.33	103.40
4' 7"	55	140.80	1041.4	70.79	108.91	67.55	105.32
4' 8"	56	143.36	1041.4	72.08	110.89	68.78	107.23
4' 9"	57	145.92	1041.4	73.37	112.87	70.01	109.15
4' 10"	58	148.48	1041.4	74.65	114.86	71.24	111.06
4' 11"	59	151.04	1041.4	75.94	116.84	72.47	112.98
5' 0"	60	153.60	1041.4	77.23	118.82	73.70	114.89
5' 1"	61	156.16	1041.4	78.52	120.80	74.92	116.81
5' 2"	62	158.72	1041.4	79.80	122.78	76.15	118.72
5' 3"	63	161.28	1041.4	81.09	124.76	77.38	120.64
5' 4"	64	163.84	1041.4	82.38	126.74	78.61	122.55
5' 5"	65	166.40	1041.4	83.66	128.72	79.84	124.47
5' 6"	66	168.96	1041.4	84.95	130.70	81.07	126.38
5' 7"	67	171.52	1041.4	86.24	132.68	82.29	128.30
5' 8"	68	174.08	1041.4	87.53	134.66	83.52	130.21
5' 9"	69	176.64	1041.4	88.81	136.64	84.75	132.13
5' 10"	70	179.20	1041.4	90.10	138.62	85.98	134.04
5' 11"	71	181.76	1041.4	91.39	140.60	87.21	135.96
6' 0"	72	184.32	1041.4	92.68	142.58	88.44	137.87
6' 1"	73	186.88	1041.4	93.96	144.56	89.66	139.79
6' 2"	74	189.44	1041.4	95.25	146.54	90.89	141.70
6' 3"	75	192.00	1041.4	96.54	148.52	92.12	143.62
6' 4"	76	194.56	1041.4	97.82	150.50	93.35	145.53
6' 5"	77	197.12	1041.4	99.11	152.48	94.58	147.45
6' 6"	78	199.68	1041.4	100.40	154.46	95.81	149.36

*Density (water) = 1.00 g/ml; ⁺Density (whole blood) = 1.06 g/ml; [#]Density (plasma) = 1.025 g/ml

The volume of blood injected into the aorta can vary between 70 ml and 120 ml depending on the above factors in any individual. The latter value gives a total aorta volume on injection of 155.4 ml. Using the same aorta dimensions as used above, this represents a cylinder with a radius of 3.15 cm and the radius factor is 2.1. An individual with a physical height of 5 feet 6 inches thus gives a maximum generated pressure possible as 178.3 mmHg compared with 141.3 mmHg calculated above. The most relevant value of an individual blood pressure is the maximum possible generated pressure which represents the limiting blood pressure. This will be different for each individual.

Blood Flow and Blood Pressure Effects

The injection of blood into the aorta represents the initial pulse in a train of pulses which travel through the blood circulation. The advance of the pulse is controlled by the inertia of blood present in the vessels ahead of the pulse. The radius of blood vessels progressively decreases beyond the aorta as the result of continuous branching and progressively increases in the veins returning blood to the heart. In a section of the vessel ahead of the aorta, the decrease in radius means that a section of the same length as the aorta (5.0 cm) initially contains a smaller volume of blood. Assuming that this section has a radius of 1.3 cm (aorta radius 1.5 cm) the volume of blood present is 26.5 ml (aorta volume 35.4 ml). The injection of 70 ml of blood into this section gives a volume increase to 96.5 ml. This means that the volume has increased by a factor of 3.64 and the radius after injection is 2.47 cm.

The radius has increased by a factor of 1.9. The pressure generated in this instance would be 156.1 mmHg and is higher than the maximum pressure generated at the aorta of 141.3 mmHg. From Pascal's Law the former pressure change is transmitted equally in all directions and the pressure is transmitted undiminished. As the pulse continues through the system under these conditions the pressure generated would increase opposing the pressure generated at the aorta. In order that the pressure in any part of the circulation system remains below that generated by the injection of blood into the aorta the radius ratio in the next section is required to be 1.72, and, for the above radius, the length of the section of radius 1.3 cm is 6.73 cm with a volume of 35.4 ml, i.e. the pulse always involves a volume of blood vessel equal to that of the aorta before injection of blood from the heart. For this to occur a longer length of blood vessel with a reduced diameter is involved. This shows that as the pulse moves through the blood circulation the length of blood vessel involved increases e.g. in the case of a blood vessel of radius 0.8 cm the length involved is

17.8 cm. This differentiates the flexible tube cardiovascular system from a rigid tube system where narrowing of a tube results in a change in flow rate and does not change the applied pressure giving rise to flow due to the absence of the radius factor. These conditions hold when the blood flow from a single vessel is divided into many reduced diameter blood vessels.

Observations have indicated that blood pressure oscillations are diminished (damped) by the vascular beds. Progressive reduction in the blood vessel radii will result in progressive diminution of the pressure signals from individual vessels as the length of blood vessel over which the aorta pulse spreads increases. This gives rise to the decay in signal strength at the approach and entry into the vascular beds. The pressure signals will tend to increase after passage through a vascular bed as the blood vessel radii commence to increase.

Increase in Individual Blood Pressure

Increased blood pressure in any individual is generally defined as 90 mmHg diastolic – 140 mmHg systolic pressure. High blood pressure is generally defined as 100 mmHg diastolic – 160 mmHg systolic pressure or above. Other variations are also known, e.g. high systolic pressure only (170/70 mmHg) and high diastolic pressure only (130/104 mmHg). There are several possible origins of observed increases in diastolic and systolic blood pressures above known individual measured values and calculated values derived as above from the physical characteristics of the individual. This applies to both systolic and diastolic values changing together and independ-

ently. The systolic pressure is dependent on both the blood density and the aorta radius factor. The systolic blood pressure of any individual is observed to rise under particular conditions, e.g. exertion or stress, and to return to normal on removal of the cause. This effect can arise from one of two sources. As demonstrated above, an increase in pressure can be generated remote from the aorta. In the first instance, this occurs with an increase in the volume of air inhaled under exertion giving rise to pressurisation of the air inhaled. The increase in pressure is transmitted to the blood in the pulmonary artery giving rise to an increased rate of flow in this blood vessel causing an increase in the volume of blood entering the heart in a given time period with a resultant increase in the volume of blood injected into the aorta. The latter change in turn results in an increase in pressure generated at the aorta. These conditions are accompanied by an increase in pulse rate. The increased pulse rate indicates that the heart contracts when the increasing volume resulting from the filling with blood attains at particular value (activating volume). The increased rate of filling described above means that this value is attained in a shorter time period, e.g. at a pulse rate of 72 beats/min 70 ml will be injected in 0.8 s and at 120 beats/min 70 ml will be injected in 0.48 s. The observed increase in blood pressure under conditions of exertion indicates that the activating volume in any individual is progressive and can increase without triggering heart contraction over a range of volumes up to a limit which is the blood pressure maximum. In the case of stress, the origin of the change is the same as that for reduction in blood vessel diameter above except that the reduction in blood vessel diameter is the result of metabolic chemical changes. The factor T in the Equation of Laplace is the tension induced in an expanded membrane which is generally taken to be an inactive body. The material enclosing large blood vessels is, in part, muscle tissue which is not inactive and whose function is controlled by the nervous system. This means that the factor T can vary and there is a relationship between the factor T and a factor describing the change induced in the muscle tension characteristics by the operation of the nervous system and/or associated chemical compounds (neurohumeral effects). Designating this factor as N then T is related to N as

$$T = (T^* + N)$$

where T^* is the tension displayed by the entire tissue of the blood vessel with no muscular action. Any change in the value of N results in tension force altering. The factor N is presently considered to be mainly controlled by electric signals transmitted through the nerve system (cardiovascular system). The regulation of these signals is considered to involve ions (Ca^{2+}) and various metabolic chemical compounds such as renin-angiotensin, catecholamines, insulin and oestrogen. In situations involving change in individual blood pressure (arterial hypertension, diabetes, heart failure, arterial stiffening with increasing age), a change is usually noted in the nature (sympathetic tone) of tissue of blood vessels indicating a change in the factor T^* . The effect of stiffening of the change in the factor T^* for the aorta is that the injected pulse of blood induces less expansion of the aorta and an increased expansion of the next section of blood vessel. The latter section has different dimensions from the aorta and the radius ratio changes. As described above, this leads to an increase in blood pressure. Similarly, an increase in the systolic blood pressure of any individual can occur when pressure above that at head level is generated at another point in the circulation. As described, blood vessel diameter decrease means that a longer length of blood vessel is required to maintain the blood pressure the same as that generated by the injection of blood into the aorta. Decrease in a blood ves-

sel diameter can occur by deposits or the action of metabolic or applied chemicals. Many non-metabolic natural and synthetic chemical compounds have been known for decades to affect blood pressure. Where the length of any given blood vessel is fixed and a longer length is not available to accommodate the required change in length the result will be an increase in blood pressure as described above. Unlike narrowing of blood vessels as described complete blockage of blood vessels does not lead to a change in systolic blood pressure as there is no passage of volume pulse and therefore no effect on the radius factor. The effect is to deprive the cells served by the vessel of the supply of chemicals required for normal functioning plus an increase of blood flow through associated blood vessels. The effect of partial blockage is dependent on the position of the blockage in the vessel. Blockage at the entrance of a blood vessel has the same effect as complete blockage. Partial blockage at a point along the vessel has the same effect as narrowing as described above. A decrease in systolic blood pressure occurs when there is a reduction in the value of the radius factor arising from reduced expansion of the aorta. This occurs when the injected volume is reduced by incomplete contraction of the heart. A change in both the measured diastolic alone or in both diastolic and systolic blood pressures indicates the presence of the above changes plus a change in blood density. The diastolic pressure is dependent solely on the blood density (ρ) and the height of the heart above ground level (h). A change in the value of diastolic pressure will only occur with a blood density increase. Venous blood density decreases when body position changes from vertical to seated and to supine [7]. In the latter positions, the value of height (h) has decreased and the hydrostatic pressure has also decreased. The normal conditions heart will generate the same pressure in all stances and in the seated and supine positions, the generated pressure exceeds the hydrostatic pressure. This change is controlled by a change in the density of blood fluid. The major component of blood fluid is water (90 %). The density of water is increased by the concentration of dissolved substances. In order for the density of blood to decrease the concentration of dissolved material is required to decrease. This occurs either by substances leaving blood plasma (precipitation) or pure water entering blood plasma, each resulting in a decrease in the concentration of the dissolved materials. In the seated and supine body positions, the change in blood density is the result of water entering the blood plasma from cells by osmotic or electro-osmotic transfer. Under these circumstances, the excess-generated pressure enhances this transfer of water resulting in the dilution of the blood fluid and giving rise to the change of density observed. This change occurs regularly during the sleeping period. The effect of a change in blood density on systolic pressure is demonstrated by the difference in the calculated values for systolic pressure for a human with a physical height of 169 cm (5 feet 6 inches) using the density of plasma (1.025 g/ml) and the density of whole blood (1.06 g/ml) (Tab. 1), namely 81.07 mmHg, and 84.95 mmHg respectively. An increase in blood density can be caused by an increase in the concentration of either the dissolved inorganic ions, proteins or suspended material. The dominant ionic compound in blood is sodium chloride with a concentration of 6.19 mg/ml (0.106 moles/l). A concentration of 350 mg/ml of sodium chloride is required to increase the density of pure water to a value of 1.025 g/l [8]. This indicates that a change in ion concentration is not the principle cause of a change in density. Blood plasma containing 76 mg of protein/ml has a density of 1.025 g/ml [8]. To change this value by 4 % requires that the protein concentration of blood has to be increased to 210 mg/ml [8]. Such an increase has not been observed as far as is known. An increase

in whole blood density of any individual is therefore a more likely cause of a change in the systolic pressure. This requires an increase in the concentration of suspended matter such as an increase in suspended particles of cholesterol. Diuretics are known to control blood pressure and function by inducing removal of excess water and sodium from the body. These compounds affect blood pressure through density change. Blood pressure is known to change throughout the day. The results above indicate that the reason for this variation is the change in blood density.

Conclusions

It is shown that a maximum generated blood pressure exists for each individual which can be calculated from knowledge of the maximum injected volume, the aorta dimensions and the height of the heart above level ground. The value will be specific to each individual and a high measured systolic blood pressure in any given individual need not indicate the presence of any deleterious metabolic condition. Only the third of the required factors can be readily determined. A measure of the maximum generated blood pressure could be obtained by the use of a mercury sphygmomanometer modified to record in the same manner as a maximum-minimum thermometer with a magnet-controlled indicator within the mercury tube. The blood pressure under seated rest conditions is measured and recorded by the position of the indicator. This is followed by measurement of the maximum blood pressure obtained under exertion generated by deep breathing in the

seated position. Any change being shown by a difference in the indicator position. The latter value will be an approximation to the maximum value. The generation of pressure in excess of the a maximum value in regions remote from the aorta as described will interfere with the operation of the heart and could cause rupture of blood vessels. These effects are like contributory causes of heart attacks and strokes. The causes of changes in measured systolic and diastolic blood pressures are shown to be different although these causes can operate in conjunction. A change in the systolic blood pressure alone means that there has been a change in the blood density. The results demonstrate that changes in blood density are one of the factors leading to change in blood pressure.

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