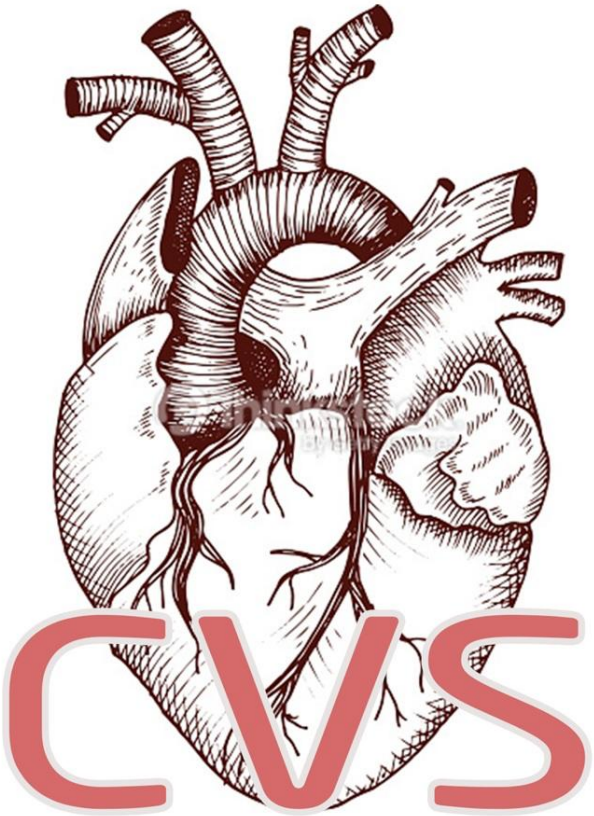

CVS

Physiology

♡ Slide Sheet ♥

Number:	15
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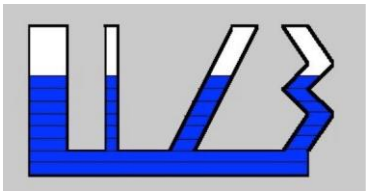
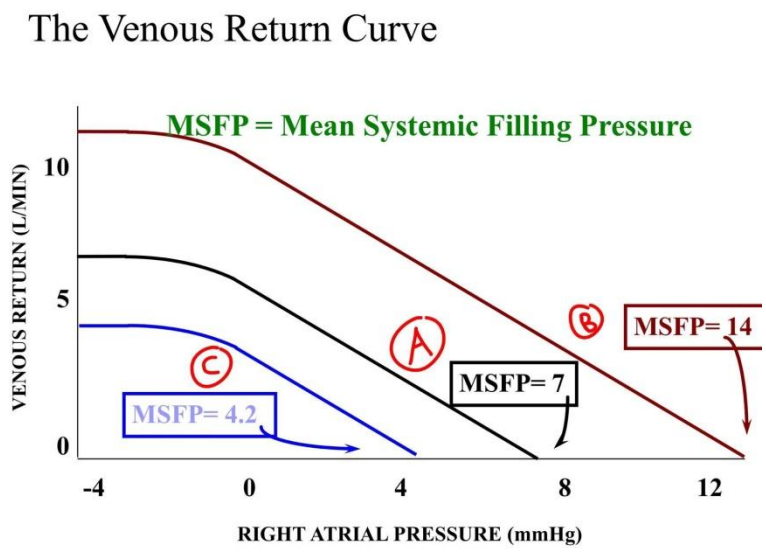
Preview

Last lecture, we started talking about the *venous return curve*. In this curve we relate the right atrial pressure with the vinous return (we put the right atrial pressure on the X axis and by that we can compare between this curve and the cardiac output curve which also has right atrial pressure values on the X axis). We said also if we stop the heart and equalize the pressure, the pressure will be the mean systemic pressure, because we are talking about the systemic circulation, now all over the circulation the pressure is the same, and it was measured by Guyton to be 7-8 mmHg.

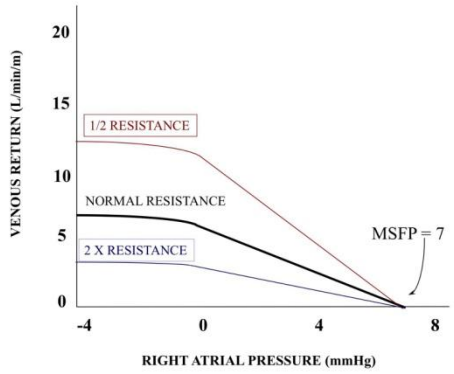


Now if the right atrial pressure equals this value, there will be no venous return (at point 0 of venous return we call it mean systemic filling pressure (MSFP)) . but if the right atrial pressure decreases then the blood will move to right atrium , until you reach 0 right atrial pressure the venous return at that point will be about 5 liters (look at curve A in the figure) , then when you become in negative pressure (which is actually vacuum) there will be collapse of large vessels , and they will open when the accumulated blood around the collapsed part of the vessels reaches a high enough pressure to open them to allow some blood to pass through them until they close again (they will open and close repeatedly) to the extent that we have almost plateau (in which the venous return will not increase significantly with decreasing right atrial pressure) .

Note that in MSFP : we say systemic because it is in the systematic circulation and filling because it causes filling of the heart .



Now how can you increase this MSFP ? By increasing blood volume (infusion, too much drinking …) , venoconstriction…, that all shift the curve right and upward (see curve B above) . But if you have bleeding , dehydration … that will shift the curve downward and to the left (curve C).

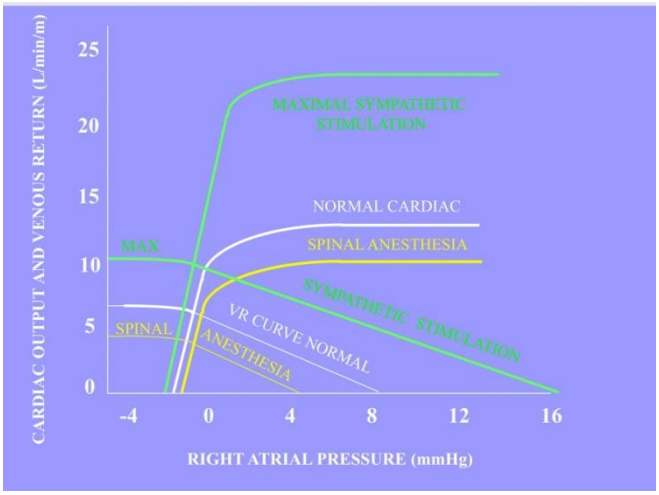


How does resistance affect MSFP ?

We will talk in upcoming lectures about the total peripheral resistance that lies mainly in arterioles. Arterial system doesn't have much blood, so changing the diameter of the arterioles (changing the total peripheral resistance) will change how these vessels will press on their blood content which is in this case a relatively small amount of the whole blood content, so it will not have any significant effect on the MSFP, while changing the diameter of the veins will affect a large amount of blood which will result in significant changes in MSFP, so applying this , if you increased the mean arterial *resistance* (not the resistance for venous return) the MSFP will be the same except the flow (venous return in this figure) will change (with decreased TPR the venous return will increase and vise versa) .

Factors that affect venous return :

- one condition is called *beriberi (thiamine deficiency)* having elastic arterial walls that aren't going to recoil so low resistance exists , then arterial dilation will occur and we will have more flow. (in slides we have decreased RVR , which means resistance to venous return not total peripheral resistance)
  NOTE that venous return = (MSFP- RAP)/ RVR … RAP: right atrial pressure.
- *AV fistula / AV shunt* = we connect arteries and veins, this is done in patients with renal failure, in this case you shortcut the circuit decreasing resistance ->increasing flow (this occurs acutely , then we will have balance in resistance)
- *Hyperthyroidism*→ increased metabolism (T3/T4 causes O2 consumption which causes releasing of vasodilators that ↓resistance ↑flow (RVR = resistance to venous return not total peripheral resistance)
- *Anemia* : decreasing in RBCs that decreases viscosity then ↓resistance then ↑flow
- Decreased blood volume leads to decreased MSFP which decreases the flow.
- Decreased venous compliance (as if we have venoconstriction) ↑MSFP and ↑flow
- Decreased sympathetic causes venodilation → decreasing venous return
- Obstruction (in femoral v. For example) of veins ↑resistance (like in abdominal tumors or -normally- in pregnancy that sometimes causes varicose veins which ends after delivery)



Here we have 2 curves together **(cardiac output & venous return) curves .**

The working cardiac output is the point of intersection between the 2 curves , where the cardiac output = venous return

Anything that shifts the curves will change the point of intersection (for example, sympathetic inhibition that happens in spinal anesthesia).

End of preview

Now starting in the first part of the physiology of circulation (ending "cardio" part and starting "vascular" part) we will talk about physical laws (ohm's law is a basic law , $F=\Delta p/R$)

Functions of circulation or blood flow to tissues :

-Delivery of O_2 and removal of CO_2 from tissue cells.

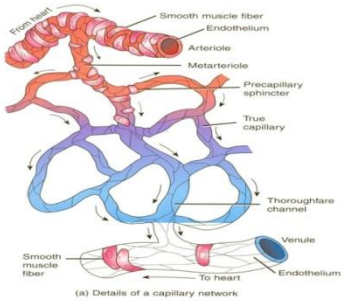
-Gas exchange in lungs.

-Absorption of nutrients from GIT.

-Urine formation in kidneys.

Systemic circulation starts from Aorta -> large then medium then small sized arteries-> then arterioles -> metarterioles-> capillaries -> venioles etc…

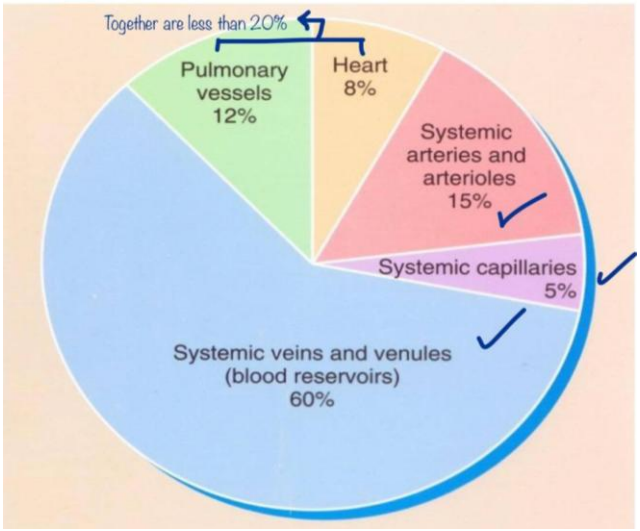
In capillaries we have microcirculation - where we have arterial and venous sides where filtration and reabsorption happen respectively, and we won't talk about it because we talked about it in first year .



Distribution of blood volume :

Around 2/3 of our blood volume lies in veins (the largest amount of blood compared to other components) that's why we call veins the capacitance vessels (because they have the largest capacity)

Doctor faisal read what is written in this figure



-Total peripheral resistance (TPR) lies in arterioles and if you constrict them they aren't going to affect the MSFP .

- Spleen is a reservoir of blood (we need it in case of hemorrhage).

- Blood flow to tissues is controlled in relation to tissue needs. So more need, brings more blood to the tissue (auto-regulation) .

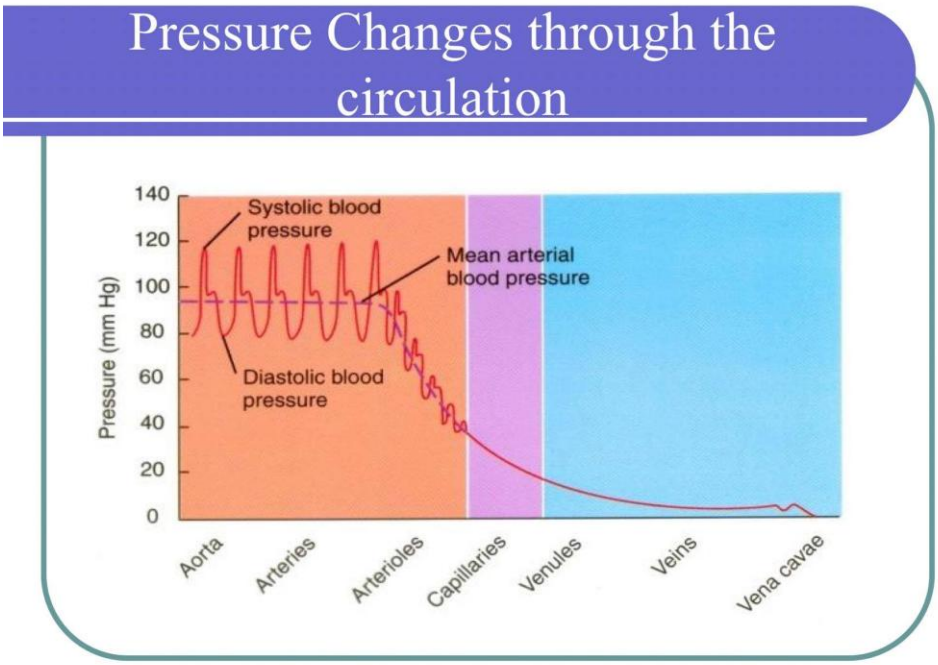
-Cardiac output is the sum of blood flow to the tissues .

-Arterial pressure is affected by many factors that's why it is almost kept constant.

Now look at this figure below and note that we take the components of each part together, for example large arteries (we talk about all large arteries as one unit) and this is applied on capillaries, arterioles … etc

(where the flow in 12 billion capillaries = cardiac output)

- Blood pressure in Aorta is pulsatile (ranges between systolic and diastolic 120/80), and between them we have mean arterial pressure (MAP= 1/3 systolic p. + 2/3 diastolic p.) note that the MAP is closer to the diastolic p.



- Cardiac output(CO) at all these areas is the same.
- Flow through each part (arteries or capillaries or…) = CO
  LET'S REPEAT THAT WE ARE NOT TALKING ABOUT ONE ARTRY ALONE , but about all arteries together.
  CO= flow = Δp / R

Now keep your eyes with the curve above. the upcoming numbers are approximate numbers:

Let's assume that the MAP in Aorta = 100 (actually it's not 100 , 120/80 gives MAP = 93-95) And if the MAP in arteries is 95 -for example- the Δp will be 5 , then to keep a constant CO (to say 1 L/min) , resistance (R) must be 5 in order to maintain the same CO . <u>We can see that the drop in pressure isn't high because there is no high resistance in arteries .</u>

At the beginning of arterioles the MAP = 85 and at the end = 35 , then Δp = 50 and R must be 50 to have CO=1, we have too much pressure drop in arterioles because we have the largest resistance (so arterioles are called major *resistance vessels*) .

Another difference that at the end of arterioles no pulsation is found(damping of pulsation occurs) , so pulsation in capillaries will be considered abnormal.

In capillaries, at the beginning MAP is 35 and at the end 15, why is it lower at the end ? We must have lower p so that blood would flow according to the difference in pressure (Δp) .

We have Continues pressure drop (pressure gradient) as we go from Aorta to right atrium .

Main pressure drop is in the arterioles (arteriolar capillary junction) .

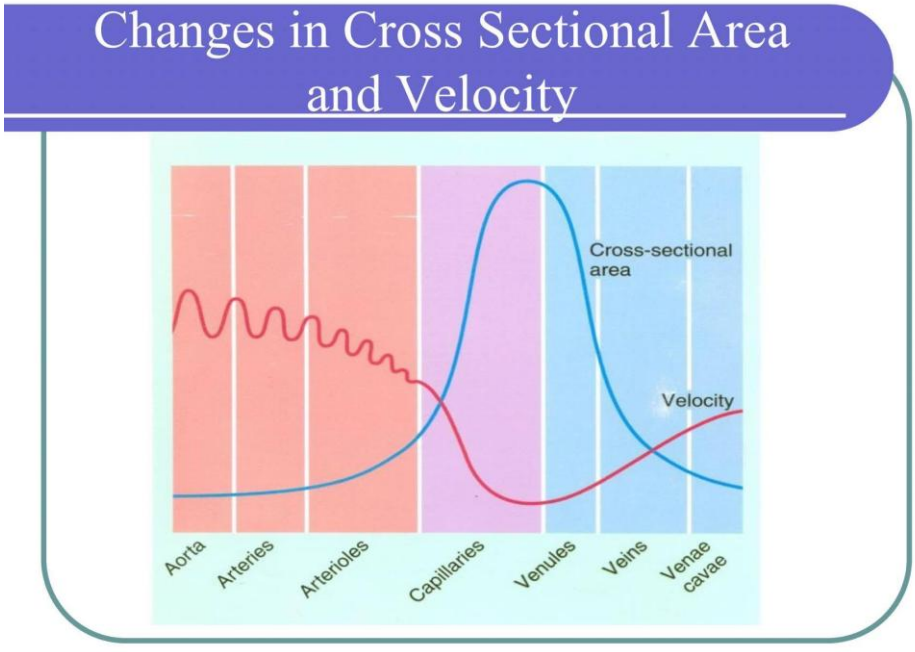
Does vasoconstriction in arterioles cause hypertension? Yes , vasoconstriction of arterioles could lead to hypertension , if it increased resistance, MAP would increase (MAP = CO*TPR)

In any vessel, flow = area * velocity = πR2 * velocity

Flow through each part is constant and equals = CO .

Remember Boyle's Law that states V1*p1=V2*p2, which describes the pressure-volume relationship of a gas (where V stands for volume and p stands for pressure), a similar law that concerns us here is the continuity equation which states that at a fixed volume flow rate through a certain tube, an increase in cross sectional area results in decreased velocity to maintain the constant flow and vice versa, i.e A1*v1= A2*v2 (where A stands for cross sectional area, and v stands for velocity).

Here in flow we have the same concept, flow is constant and (area* velocity) must be constant , so if we have ↑ area then velocity will ↓, and vise versa …



We have Higher velocity in Aorta and lower in arteries because of lower area in aorta.

In capillaries, large area causes slow velocity so the flow and the CO will still be constant. (don't forget again that we are talking about all capillaries in the body .actually they are more than 12 billions capillaries) .

Slow flow gives capillaries an advantage, so they have enough time for exchange of gases and nutrients, also the large area increases the exchange (faster and higher diffusion)

Velocity in vena cavae is lower than velocity in Aorta , because we have 2 vena cavae (larger area) and because its more flexible than Aorta (see this figure)

	cm
Aorta	**2.5**
Small Arterioles	**20**
Arterioles	**40**
Capillaries	**2500**
Venules	**250**
Small Veins	**80**
Venae Cavae	**8**

capillaries have the largest total cross sectional area in the circulation .

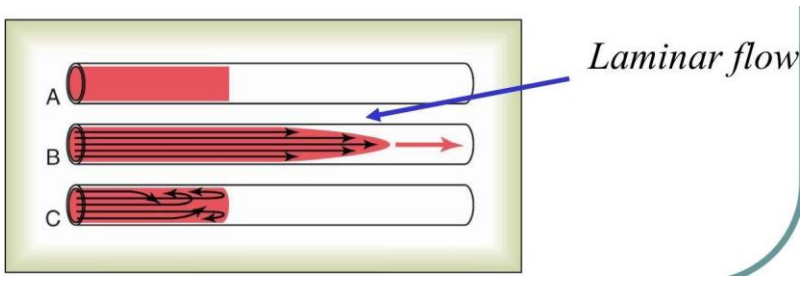
Flow is constant . velocity in Aorta >Arterioles > Small veins >Capillaries , and is inversely related to cross sectional Area .

Now let's talk in details about blood flow: Blood flow or ‘‘F‘‘ =the quantity of blood that passes a given point in the circulation in a given period of time (mL/Sec)or (mL/Min)or (mL/Hour)…

These laws of flow are applied only on laminar flow (or stream line flow) , blood in “laminar flow” flows in layers . On the other hand, Turbulent flow (called "eddy" currants like حوامات الماء) occurs when we have constriction and it's not an efficient flow (only laminar is efficient) .

Note that we talked previously about “turbulence” in heart sounds , where S1&S2 are caused by turbulence of blood around closed valves .

Blood doesn’t flow as plugs (tube 'A' below) .Instead it goes as in tube 'B' , where the blood in the center has the highest velocity that decreases at the sides because of increasing resistance near the wall. This gives what we call “ parabolic “ shape of flow (زي منحنى أو شكل قطع مكافئ).



Again Laminar means in layers (laminae) with uniform speeds at certain distances from the wall . If the flow rate increases, then the trend for turbulence will increase.

To distinguish between laminar and turbulent flow , we calculate reynold’s No. (Re)

$$\text{Reynold's No (Re)} = \frac{v.d.\rho}{\eta}$$ If Re is > 400 then Turbulent flow

v=velocity , d= diameter of vessel , ρ(Rho)=density of blood, η(ita)=viscosity of fluid

If (Re) is > 1000, then the flow is turbulent, if it is < 400, then the flow is laminar, if it is 400-1000, the type of flow depends on the situation (for example, if there is constriction or atherosclerosis there will be turbulence).

Causes of turbulent:

-constriction

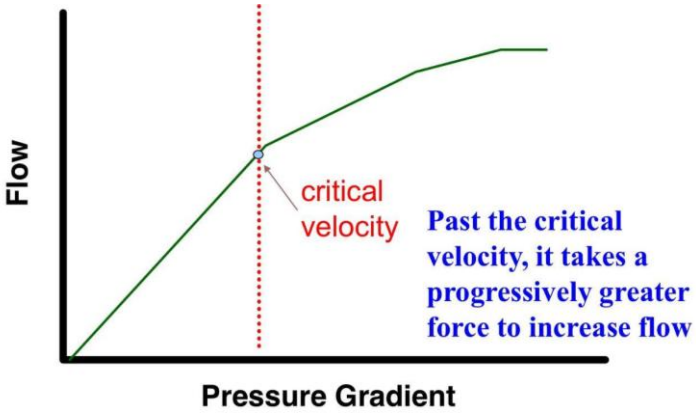
-rapid narrowing (that disturbs flow) .ex: atherosclerosis

Again Laminar means in layers (laminae) with uniform speed . If the flow rate increases , the trend for turbulence will increase.

-sharp turns/curves in circulation . ex:axillary with subclavian arteries.

-high velocity

Critical velocity: above this velocity we have turbulent flow

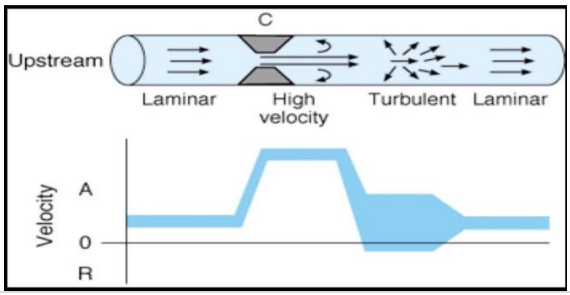


-rough surfaces in the circulation.

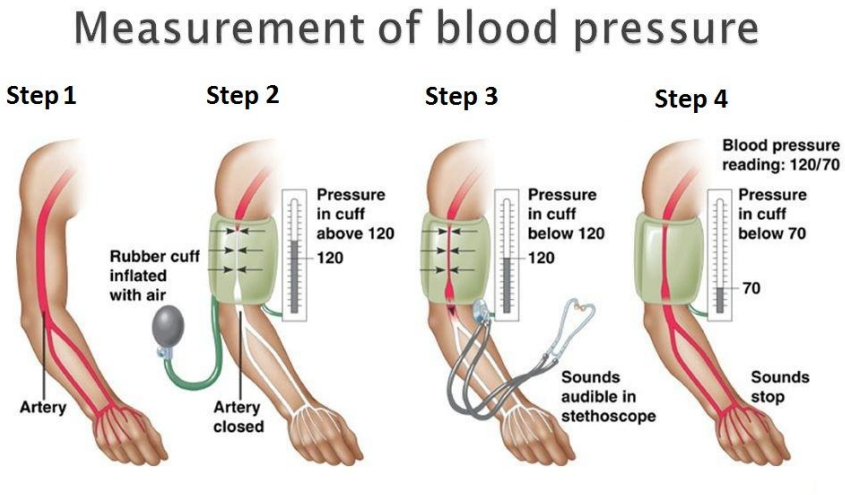
Turbulence predisposes to the formation of thrombi, because turbulent flow actually isn't going to flow (it stops everywhere) .

Laminar flow has no sound, but turbulent flow has sounds. So because of these features, when we hear sounds in blood pressure measuring procedure, the sound is produced by turbulence of blood.

To illustrate this point, actually we increase the pressure in the cuff until the flow stops (so the pressure we put is higher than systolic p.) . Then we decrease the pressure slowly until we reach the systolic pressure where the flow resumes in



a turbulent way so a sound will result from the flow of blood. we keep lowering the pressure in the cuff until the sound finally disappears because we have now laminar flow (and the pressure becomes lesser than diastolic p.)



A student's question: Does AV block cause turbulence? Yes, it does.

Abnormal sounds of flow are called murmurs / bruits (it's a French word , where "t" is silent and not pronounced) that you can hear on heart sounds. Murmurs or bruits are important in diagnosing vessels stenosis, vessel shunts, and cardiac valvular lesions.

If the bruit was in vessel then it is due to constriction, but if it was in heart then it is due to regurgitation or stenosis.