

**Course Name :An Overview on Maternal Health Antenatal, Intranatal and Postnatal Care**

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### **Physiological changes in pregnancy ( Part 2 )**

Good morning students. I welcome you all to the next session of our NPTEL online certified course on the topic and overview on maternal health, the antenatal, intranatal and postnatal care. I am Dr. Barnali Ghosh, an obstetrician and gynecologist working as assistant professor at B.C.Roy Medical College and Research Center, IIT, Kharagpur. Today, our topic of discussion is the physiological changes or the maternal adaptations occurring during pregnancy. This is the continuation of the previous class discussion where we had a discussion regarding the reproductive tract changes, the hematological changes and the respiratory system changes and a little bit regarding the skin and the breast changes occurring during pregnancy.

Today, we will be discussing with the cardiovascular system changes, right. So what are the changes occurring in the CVS? Starting you know right from the beginning, what happens in the early stages in the first trimester of pregnancy, there is vasodilatation. So the arteries, the peripheral arteries, the systemic arteries, they undergo vasodilatation starting right from 5 to 6 weeks of gestation, right. And this is brought about by you know estrogen as well as the circulating prostaglandins.

They will be causing this vasodilatation by nitric oxide. This nitric oxide will lead to vasodilatation, right. And what happens because of this? There is decrease in peripheral vascular resistance. There is also decrease in systemic vascular resistance, right. So due to this, as a result of this, the blood pressure will also decrease.

The arteries are increasing in diameter. There is vasodilatation which will lead to decrease in blood pressure. As a compensatory mechanism, the heart, this is the heart, the four chambers of the heart and this heart will be pumping more blood into the circulation for the maintenance of blood pressure. Thus the cardiac output, cardiac output will increase, right. So cardiac output increases during pregnancy by 40% whereas systemic vascular resistance decrease by 25%, right.

What is cardiac output? Cardiac output is nothing but it is heart rate multiplied by stroke

volume. So during pregnancy, heart rate also increases and the stroke volume increase is you know much, much higher. There is appreciable increase in the stroke volume. How come this happens? Why there is appreciable increase in stroke volume? See this heart, the heart muscles, right, muscles of the ventricles, they undergo hypertrophy and hyperplasia. So number one, if I draw this heart here, the muscles of the ventricles, they will undergo hypertrophy and hyperplasia, right.

So there is increase in ventricular muscle mass, number one. So this will lead to increased ventricular contractility, right. So this is number one. Another thing is this ventricular cavity, there is dilatation, physiological dilatation of ventricular, left ventricular cavity, right. So the cavity enlarges and due to this left ventricular cavity and due to this what happens? Increase in end diastolic volume, right.

So first increase in ventricular muscle mass causing increased myocardial contractility and secondly increase in the ventricular cavity, that is a physiological dilatation occurring in the ventricles during pregnancy will lead to increased blood accumulating in the ventricles. That means there is increase in the end diastolic volume. These two together will result in increase in stroke volume, right. So this is what happens in CVS. Another thing to note here is this ventricular apex, there is shifting due to increase in this muscle mass.

This is increasing, increasing, increasing and the ventricular apex will be shifted outwards laterally where it was present initially in the non-pregnant state, the apex of the ventricle shifts laterally and somewhat upwards. So there is shift in the apex bit during pregnancy, right. So coming to the theory, yes there is peripheral vasodilatation starting from 5 to 6 weeks of pregnancy which is mediated mainly by estrogen and the circulating prostaglandins via the increase in you know nitric oxide and thereby resulting in fall in systemic vascular resistance by approximately 25 to 30 percent which results in fall in blood pressure and as a compensatory mechanism there is rise in cardiac output. Cardiac output is nothing but heart rate into stroke volume. Heart rate increases by 10 to 15 beats per minute and stroke volume will increase appreciably and we have discussed it is due to number one the ventricular muscle mass increasing thereby leading to increase in myocardial contractility and secondly it is due to physiological dilatation of the ventricles of the heart leading to increase in end diastolic volume, right.

So ultimately the heart beat increases, the blood being pumped out of the heart also increases. Cardiac output increases, right and these are all physiological these are normal in pregnancy. During the pregnancy the cardiac output increases by 40 percent compared to the pre-pregnant values. Now coming to the shifting of apex beat, right. So this apex beat this is the you know it is felt by the palm of the examiner or the examining doctor when he or she places the palm over the apex the ventricular apex he can feel the apex beat on the palm.

So normally it is present in the fourth intercostal space but during pregnancy this apex beat it was present in the mid clavicular line at fourth intercostal space. This was the previous position during non-pregnant state. In pregnant state it shifts outwards, it moves outwards 2.5 centimeter away from the mid clavicular line. Previously it was at the mid clavicular line but during pregnancy it shifts 2.

5 centimeter away from the mid clavicular line and there is a little bit upward shift and it reaches the fourth intercostal space. Previously it was in the fifth intercostal space during pregnancy or at term pregnancy it is palpated at fourth intercostal space 2.5 centimeter away from the mid clavicular line, right. Now coming to a very important phenomenon occurring during pregnancy which is called as the supine hypotension syndrome. This is why this occurs.

As the uterus is growing first it was a pelvic organ up till 12 weeks and after 12 weeks it grows into the abdomen being an abdominal organ. As the pregnancy progresses the uterus enlarges in size and this enlarging uterus will be you know putting pressure over the IVC which is behind the uterus. It will compress the IVC and thereby leading to venous stasis which will ultimately lead to decrease in venous return to the right side of the heart. Thus there is less blood coming to the heart, less blood coming out of the heart. So the cardiac output momentarily decreases due to the pressure of the uterus on the IVC which will decrease the blood pressure, right.

So this compression of the IVC by the uterus is more when the patient is in supine, is in lying down position, right. And this phenomenon of decrease in blood pressure during the lying down position it is seen somewhat later in pregnancy. Now starting from the second trimester around 20 weeks. During this time after 5 weeks of pregnancy, 5 months of pregnancy, you know we tell the patient that you should be lying down on the left side or the right side in lateral position more preferably on the left lateral position towards the third trimester because this will you know help the uterus to shift towards the, towards one side, towards the left iliac fossa. So as she moves in the left lateral position, the uterus will fall in the left iliac fossa thereby decreasing the compression of the, over the IVC and thereby the venous return to the right heart will be maintained thus maintaining the blood pressure.

So what is the supine hypotension syndrome? Yes it starts from second trimester approximately around 20 weeks and you know this in lying down position, in supine position of the mother there is compression of the IVC by the dextrorotated pregnant uterus which will lead to decrease in venous return to the right heart thereby causing decrease in blood pressure or hypotension called as supine hypotension syndrome. And very important to note here that turning the pregnant mother from supine to left lateral position will increase the cardiac output by 25 percent and vice versa when she turns from the left lateral position to the supine position

it will result in decrease in cardiac output by 25 percent right. So in case of you know operation or in LUCS when the mother is in operation theatre table she is in supine position and we have a specialized you know pillow which is to be kept below the back of the mother this is known as Crawford's wedge right it has an inclination it has an inclination right you can see here. So it is kept at the back of the mother so that the mother gets you know the back gets tilted the pelvis of the mother gets tilted and it is mother gets towards the left lateral position right. So mother is in supine but keeping this wedge below the back of the mother will help to rotate the pelvis of the mother towards the left side which will help in you know shifting the uterus towards the left iliac fossa right.

So the uterus drops to the side and it will be relieving the compression over the IVC right so the uterus drops to the side and off the IVC and thus cardiac output and utero placental flow are optimized they are maintained and you know after the baby is delivered during caesarean section we keep this wedge below the back of the mother after the baby gets delivered then we remove the wedge because now the uterus you know has decreased in size and the compression of the IVC will automatically go right. So this is a clinical implication of the supine hypotension syndrome. Now coming to the changes in the CVS as a whole cardiac output yes cardiac output increase by 40 percent stroke volume we have discussed there is appreciable increase in stroke volume heart rate increase by 10 to 15 beats per minute blood pressure in the early stages of pregnancy it decreases right in first second trimester blood pressure previously decreases there is vasodilatation peripheral vasodilatation leading to decrease in blood pressure but you know in third trimester there may be increase in blood pressure. CVP central venous pressure there is no change right pulmonary capillary wedge pressure there is no change it remains unchanged during pregnancy. Pulmonary capillary wedge pressure is actually an indirect measure of the left heart function right.

So this remains unchanged during pregnancy jugular venous pressure will remain unchanged during pregnancy all these three does not undergo any change. Peripheral vascular resistance or systemic vascular resistance yes we know there is peripheral vasodilatation and the resistance will decrease by 25 to 30 percent. Coming to the serum colloid osmotic pressure we have already discussed in hematological changes that there is increase in blood volume in pregnancy where the blood plasma increase is more compared to the blood cell increase. So this leads to you know hemodilution in pregnancy. So the colloid osmotic pressure which is the which will decrease because there is hemodilution so this decreases in pregnancy right.

Now coming to another important concept colloidal osmotic pressure it decreases we have just now read that colloidal osmotic pressure decreases by 10 to 15 percent. So if these are the blood capillaries right so the colloidal osmotic pressure inside the capillaries this decreases there is more fluid there is hemodilution and the concentration of proteins decreases and so the colloidal osmotic pressure decreases. This leads to you know more chance of extravasation of fluid by the

process of osmosis the osmolarity inside is low and in the extracellular matrix which is outside here the osmolarity is high and there will be a tendency for extravasation of fluids and water into the extracellular matrix right. So what happens there is fluid extravasation from the capillaries. So there will be more fluid accumulation in the extracellular matrix.

In lungs in the pulmonary capillaries the osmotic colloidal osmotic pressure also decreases and blood from the capillaries goes into the extracellular matrix thereby making the mother more prone to pulmonary edema. This ratio is the measure the colloidal osmotic pressure by pulmonary capillary wage pressure. This ratio gives us the measure of you know the extent of fluid extravasation and there is a decrease of 30 percent this ratio decreases and thus the pregnant women are more susceptible to pulmonary edema. Very important concept because they are more prone and we need to check we need to auscultate the chest every time she complains of breathlessness, she complains of cough whether there is any underlying pulmonary edema you know going.

So we need to intervene right. Okay now coming to the next concept that is hyperdynamic circulation. Blood volume increases in the capillaries, cardiac output increases, heart rate increases which results in a hyperdynamic circulation during pregnancy. So what happens S3, S4 they become audible during pregnancy and these findings are normal right. So what is S3? S1 is due to closure of the valves. S1 closure of mitral and tricuspid valve.

S2 is due to the closure of pulmonary and the aortic valves. S3 is due to blood draining into the right atrium and the right ventricle right. So when there is hyperdynamic circulation more blood coming to the right to the atrium and then passing to the ventricles which will result in a S3 or S3 is present during the pregnancy. S4 can also be audible.

S4 is due to atrial contraction. Systolic murmur. Yes, systolic murmur sometimes are present in pregnancy, but they are without thrill. Thrill is nothing but a palpable murmur. So the systolic murmur which is due to also due to the hyperdynamic circulation they may be present in pregnancy, but they are without thrill. So when the systolic murmur is present with a thrill it becomes an abnormal finding right.

So without thrill systolic murmur is normal in pregnancy. Another concept is S1 split. This is also present may be present during pregnancy. So S1 split is nothing but a difference. You know S1 is due to mitral and tricuspid valve closure and these two valves do not close at the same time.

So we can hear sometimes the mitral valve and the tricuspid valve closure separately and that is called as S1 split which can be present during pregnancy right. So this was all during the antenatal period. What was going on during the pregnancy right. Now coming to the

intrapartum period when the labor when a onset happens the mother goes into labor what happens during that time there is further increase in cardiac output right. So cardiac output I will you know go to the discussion part first what happens cardiac output starts increasing, increasing, increasing, increasing.

It will increase throughout the pregnancy and reach a peak at around say 32 weeks of gestation. This is a very important point at 32 weeks there is you know peak in cardiac output in the antenatal period. So any heart disease patient when she becomes pregnant you need to counsel her that yes you have an underlying heart disease and during pregnancy there is increase in cardiac output in and more the heart has to pump more blood and so more chance of heart failure which is maximum at 32 weeks. So sometimes we advise patients of heart disease for admission around 32 weeks because you know for observation because at this point the cardiac output is highest and there is more chance of heart failure and then we need to intervene.

So this was during your antenatal period. Now what happens after this it remains the same then labor onset starts labor starts right she goes into labor now again there is increase first stage labor is divided into four stages in first stage there is further increase of cardiac output by 15% in the second stage of labor it increases by 50% right in the third stage there is delivery of the baby and the placenta right. Now coming to the fourth stage following the delivery fourth stage following the delivery of the baby and the placenta the baby is outside the mother's womb with the placenta which has already been separated. Now the uterus will contract uterus will contract right physiological after the expulsion of the baby and the placenta the uterus will contract and help prevent blood loss from the uterine site right. So this contraction of the uterus causes blood to be recirculated or auto-transfusion of blood from the uterus into the circulation into maternal circulation right and thus there is increase in blood in heart can you understand. So the uterus is contracting following the delivery there is auto-transfusion every contraction of uterus will you know give extra amount of 300 to 500 ml of blood into circulation with every contraction right.

Another thing is the compression on the IVC the IVC is behind it is going like this and this compression will be relieved as the baby is expelled out uterus decreases in height and the compression of the IVC will be relieved and so the venous return will also increase and cardiac output will also increase. So in the fourth stage there is maximum increase increase by 60 to 80 percent very important. In this stage the mother is most prone to heart failure we have to be very very you know most prone to heart failure very very cautious, meticulous because we need to monitor the mother and this increase remains for you know one hour it remains for one hour. After one hour what happens so after one hour after one hour of delivery the cardiac output returns to pre-pregnant levels pre sorry pre-labor levels. So this one hour following delivery is very vital and has the highest chance to you know to undergo heart failure.

So that is what is written here there is 15 percent increase in the first stage of labor then 50

percent then in the second stage then immediately after delivery 60 to 80 percent increase both due to number 1 uterine contraction leading to no more blood into maternal circulation and also the IVC compression has gone away and thus it will increase in venous return and will increase in cardiac output both these two things will lead to increase in cardiac output by 60 to 80 percent right. So uterine contraction every uterine contraction lead to auto transfusion of 300 to 500 ml of blood back to circulation and there is also rapid decline to pre-labor values within one hour of delivery pre-labor values what right now coming to the cardiac output when it reaches to the pre-pregnancy values pre-pregnancy values are reached by 4 weeks postpartum ok. So now that is all about the intrapartum and postpartum changes in the cardiac output. Now summing up the normal findings which are physiological which are not abnormal of the CVS in pregnancy are number 1 bounding and collapsing pulse hyperdynamic circulation increase in heart rate leading to bounding pulse. Yes ejection systolic murmur is normal without thrill if systolic murmur present with thrill then it is abnormal S1 is loud again due to hyperdynamic circulation S1 split is also normal relative sinus tachycardia S3 may be audible there may be some ectopic beats and there may be peripheral edema due to decreased colloidal osmotic pressure and due to venous stasis in the lower limbs ok.

Now coming to the normal ECG findings in pregnancy there may be some small atrial or ventricular ectopics left axis deviation the left heart goes undergoes hypertrophy hyperplasia increase in muscle mass causing left axis deviation Q wave or inverted T wave may be present in late 3 there may be ST depression or T wave inversion in the inferior and lateral leads right. So, this was all regarding the cardiovascular system changes. Now coming to the blood coagulation profile changes what happens during pregnancy as a whole it is a hypercoagulable state why because there is increase in the blood clotting factors there is decrease in the inhibitors of coagulation the proteins or the factors which will prevent coagulation their you know concentration decreases during pregnancy and thirdly there is decrease in the fibrinolytic activity right. So, coming to the clotting factors all the clotting factors increase during pregnancy except factor 11 and 13 these two decrease in pregnancy and factor 2 5 and 12 these remain unchanged right. So, you also need to know this the names of the clotting factors now there is an extrinsic pathway intrinsic pathway which you have you know went through in the physiology of blood clot coagulation.

Now just to remember that these factors some fact all factors increase, but 11 and 13 and it decrease and 2 5 12 will have no change. So, what are the names factor 2 is prothrombin factor 5 is labile factor and factor 12 is hageman factors these will have no change in pregnancy whereas, factor 11 is plasma thromboplastin antecedent and factor 13 is fibrin stabilizing factor which will decrease in pregnancy ok. Now coming to a virchow's triad I you all know that you know this is the virchow's triad which is explaining the mechanism of blood coagulation what is the triad number 1 is endothelial injury number 2 is hypercoagulability and number 3 is venous stasis when these three combine there the blood coagulation cascade gets activated and this is

called as the virchow's triad. In pregnancy endothelial injury happens yes due to over stretching of the blood capillaries there may be injury to the endothelial lining or following delivery minor trauma trivial trauma can lead to endothelial injury. Number 2 hypercoagulability just now we have read that there is increase in clotting factors.

So, it is a hypercoagulable state and number 3 venous stasis due to compression of the IVC by the uterus thus it you know increases the chances of blood coagulation or you know clot formation during pregnancy right. So, among the coagulation factors all the coagulation factors as except 11 and 13 will increase, but maximum increase is seen in case of fibrinogen which increases by 50 percent normally in non pregnant state the normal range is 200 to 400 you know on an average 300 milligram per dl which during pregnancy increases to 300 to 600 on an average 450 milligram per dl right. So, there is a 50 percent increase and this will ultimately lead to increase in ESR by 4 fold and also increase in C reactive protein right. Now coagulation factors increases yes we know now decrease in the inhibitors of coagulation or anticoagulation what are the factors coming into play number 1 is protein S. Protein S what happens protein C is another factor which is activated by protein S it gets activated protein C with the help of protein S right and this protein C will you know inhibit it will inhibit inhibit factor 5 A and factor 8 A these are all blood coagulation factors helping in extrinsic and intrinsic pathway of blood coagulation.

So, in pregnancy what happens this protein S decreases thereby the protein C though the amount is same remains unchanged, but activated protein C formation decreases in pregnancy there is activated protein C resistance and thus the inhibition of these 2 factors will also go away factor 5 A activated factor 5 A and activated factor 8 A will not be inhibited in pregnancy. So, ultimately it will be increased or hyper coagulable state right this will lead to hypercoagulable state. Another point is the anticoagulation factor antithrombin 3 this also decreases in pregnancy antithrombin 3 has 2 attachment side this is the heparin binding site right and this is thrombin binding factor 9 and factor 10 binding site. So, this antithrombin 3 concentration also decreases in pregnancy right. So, anticoagulation profile yes protein C decreases formation of protein C this remains unchanged, but activated protein C will decrease sorry it will be decrease very sorry for this it will be decrease because protein C activation cannot happen in absence of protein S.

So, activated protein C will decrease and antithrombin 3 also decreases. So, ultimately leading to a hypercoagulable state. Now fibrinolytic activity it also decreases in pregnancy and these 3 together causes the pregnancy as a whole a hypercoagulable state right. So, the blood tests for in vitro blood test which we do that is the blood bleeding time, clotting time, prothrombin time and activated thromboplastin time these remain unchanged in pregnancy, but as there is a more tendency for blood coagulation there is more risk of venous thromboembolism. VTE risk in pregnancy increases by 6 times and this increase starts right from the first trimester till 6 weeks postpartum.

Greatest risk is following delivery in the pure peril period right. So, if you know divide this into antenatal period and postnatal period the during the antenatal period increase the risk increases you know is 50 percent and postnatal period another 50 percent increase. Postnatal period is of 6 weeks antenatal period is of 40 weeks. So, all throughout this 40 weeks the risk increases 50 percent and after delivery just 6 weeks there is another 50 percent increase.

So, more risk period is the postnatal period right ok. Now as the pregnancy progresses first trimester, second trimester, third trimester the VTE risk gradually increases it is 10 percent another 10 percent in second trimester approximately 30 percent in third trimester. So, as pregnancy progresses the VTE risk also progresses right. So, this is what I have already mentioned. So, now coming to the pregnancy complication there is more coagulation may be forming a clot or you know blood coagulation, fibrin clot in the placental circulation that may lead to placental abruption. It may lead to decrease in utero placental circulation causing still birth there may be hypertensive vasospasm difficulty you know defective angiogenesis leading to hypertensive disorders in pregnancy gestational diabetes and preterm birth.

These are all associated with high cardiovascular risk or patients with heart disease who become pregnant right. So, now coming to another concept which is called as May thurner syndrome what happens the IVC see this is the aorta, aorta dividing into the common iliac arteries and IVC is towards the right of the aorta right. So, IVC with the two common iliac veins. So, here to note the left common iliac vein is you know compressed by the right common iliac artery right.

So, this is anatomy this is normal it is present. So, this left common iliac vein is compressed by the right common iliac artery. Now during pregnancy there is venous stress there is more chance of you know we have already told that there is more chance of hyper coagulation and this can lead to thrombosis formation and this thrombosis in the deep veins mostly occurs in this left side. So, left sided venous thromboembolism is more than the right side this is one point another is you know proximal venous thromboembolism is more common than distal venous thromboembolism which means that iliofemoral venous thromboembolism you know in the embolism is for dislodgement of this thrombus and thrombus in this iliofemoral veins are more common than the popliteofemoral veins right. And what is the ratio 9 is to 1, left is to right is 9 is to 1, 85 percent versus 15 percent in pregnancy, left side is more common on the left side ok. Now levels of coagulation factors they return to normal there is increase in coagulation factors during pregnancy and they return to normal after delivery by 2 weeks.

What other things occur at you know by 2 weeks following delivery? Number 1 just amalgamation of data so that you can you know just recollect very easily HCG normalizes by 2 weeks, coagulation factors normalizes by 2 weeks and number 3 uterus becomes a pelvic organ

by 2 weeks after delivery. It was an abdominal organ by 2 weeks it becomes a pelvic organ and it returns to the pre pregnant state by 6 weeks right. So, these are the events occurring by 2 weeks of delivery. So, this was all regarding the blood coagulation profile and the CVS changes occurring during pregnancy.

This is the end of today's discussion. Next we will carry forward with the other system changes in our next class. Thank you.