CLINICAL DIAGNOSIS OF POST-OPERATIVE CIRCULATORY DISTURBANCES

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THE development of biochemistry and biophysics have in later years given us important tools for estimating the efficiency of circulation and ventilation; we can estimate the oxygenation saturation or the oxygen tension, the pH, bicarbonate and the carbon dioxide tension by punctioning arteries and veins. We can also measure the blood pressure in arteries and veins with accuracy by direct punction and determine the circulating blood volume. We still do not possess instruments for clinical use which can give us reliable informations of the flow through arteries. Again existing instruments are not always available to us day and night and we may find ourselves in situations where they are not available at all; also by using these instruments valuable time may be lost—and that may even prove fatal to the patient. Therefore exact clinical observation and reasoning is still of the greatest importance and, if properly executed, may lead us far on our way to the right diagnosis and treatment.

Some time ago I happened to walk through our receiving station for emergency cases together with an anæsthetist. We found an unconscious man in obvious respiratory distress, lying on a stretcher on the floor, while a perspiring, slightly cyanosed intern was lying on his knees besides the man trying to puncture his cubital vein in order to obtain some exact information. The anæsthetist immediately lifted the stretcher with the patient on to a table, intubated the man, aspirated the fluid which obstructed his bronchi and ventilated him with dramatic effect.

I would like to present the problem I am speaking about today in the following way: You are called some time in the early morning—let us say between 3 and 7 a.m. by a nurse speaking to you over the telephone. You are on duty and she tells you that the patient who was operated upon the day before or two days before has suddenly become ill. She can hardly feel his pulse and she is not sure that she can get the blood pressure. You are, of course, not suggesting any treatment by telephone, but you jump out of your bed and rush over to the department, quickly rehearsing the most probable causes of this sudden change in the patient's health.

Let me first make the comment that it is not very common that a patient gets ill suddenly, and that if it does happen, then it usually is between 11 p.m. and 6 to 7 a.m., because this is the time when the patients are not as closely observed as at other times; not in your hospital, of course, not in mine, but in a hospital elsewhere. If the patient's general condition, his pulse and his blood pressure are observed at regular intervals, sudden changes rarely take place. Such things do, of course, exist as internal bleeding from a major artery, pulmonary embolism, and acute coronary thrombosis, but they are not common as post-operative conditions.

I would mention as the more common causes of the conditions I am referring to: internal bleeding, post-operative shock, heart failure, gastric distension, massive atelectasis, and pulmonary embolism, and I will try to point out the clinical signs which are characteristic of these conditions.

When I speak to my students on this question and ask them what they will do to make the diagnosis, one will suggest to take an electrocardiogram, another one to take the blood pressure, a third one to use a stethoscope, but I would like to emphasize that you must first use your eyes and get all the information you can by that before touching the patient.

Psychic Behaviour.

As soon as you are close enough to see the patient you must notice his psychic behaviour. A bleeding patient is sometimes very quiet and sometimes in great anxiety—even screaming. For a patient in shock it is characteristic that he is in a state of apathy. In heart failure we must distinguish between right-sided heart failure and left-sided heart failure. In right-sided heart failure the patient is dyspnœic, fighting for breath, perhaps sitting up in bed to make the best use of his auxiliary respiratory muscles. In left-sided heart failure, as, for instance, in acute coronary thrombosis, he is quiet, but at the same time in great anxiety, lung œdema may develop with respiratory distress. A patient with gastric distension shows practically all the same clinical signs as a patient in shock. A patient with massive atelectasis is also in a state of anxiety and so is a patient with pulmonary embolism. You will see that the most characteristic observation regarding the psychic condition of the patients is the apathy of a patient in shock.

Respiration.

Next, without touching the patient, you can observe the respiration. A patient with severe bleeding has a quick gasping respiration and a feeling of air hunger. The patient in shock has a quick shallow respiration. A patient with a right-sided heart failure is fighting for air and, as already described, often sitting up, fighting for breath. In left-sided heart failure the respiration will resemble the respiration in severe bleeding and, if lung œdema develops, he is in respiratory distress. A patient with atelectasis has a quick shallow breathing, and very characteristic is the so-called frustraneous cough which may tell you the diagnosis as soon as you

enter the room. The patient with pulmonary embolus is fighting for air and trying to sit up, if he is not too ill to do that.

Skin.

Your visual informations are far from ended here. You will still have to look at the skin. In bleeding the colour of the skin is white as a sheet, or yellowish, like old ivory as it is poetically described in most text books. In shock the skin colour is pale, cyanotic, and wet. The reason for this difference in the colour of the skin I shall mention when we come to the pathology. In right-sided heart failure the skin is blushing red and cyanotic. In left-sided heart failure it is pale. In atelectasis the skin is more or less cyanotic, and so it is in pulmonary embolism.

Veins.

Let us still let the blood pressure apparatus alone for a while and look at the veins. In a patient with severe bleeding the veins are contracted and so are the arteries and capillaries. The veins are actually contracted and may be seen furrowing the surface of the skin as dried-up river-beds when the volar side of the forearm is observed aslant. In shock the veins are also contracted and the blood is pooled in the dilated capillaries. In right-sided heart failure the veins are distended, and this may be best seen on the neck, but also on the hands. In left-sided heart failure the veins are contracted. In atelectasis it depends on the degree of the condition, and in pulmonary embolism they will often be distended because of right-sided heart failure.

By using your eyes alone you have already made many important observations, and if you know what to look for, and you have trained yourself in observing patients, all this—which it has taken me a long time to go through—is a matter of seconds.

Pulse.

Now it is time to feel the pulse, but it will not give you much information, unless the patient has coronary disease, because in practically all these conditions the pulse will be quick and small, but in coronary disease it will usually also be irregular.

Blood Pressure.

The blood pressure is of importance if you follow it regularly, but in the present condition it will be of very little help to you in making the diagnosis. If the patient is bleeding the blood pressure will remain normal for a long time, because most arteries and arterioles will contract in order to keep a sufficient flow through the brain and the coronary circulation. You may have ischemia of the kidneys with a normal blood pressure. We know that a healthy animal can lose more than one-third of its blood and keep a normal blood pressure. When the pressure drops it usually drops quickly. In post-operative shock it is my experience that if you follow the blood pressure at regular intervals you will find that it is falling gradually, and that it is much more uncommon to see it drop quickly as in the bleeding patient. It is extremely dangerous if a patient in shock

has a blood pressure of 70 or below for more than 20-30 minutes. Following this you can often bring the blood pressure up again by giving sufficient transfusion, but you will often find that irreparable changes have developed in the kidneys, liver or lungs from which the patient will ultimately die.

In right-sided heart failure the blood pressure may remain normal, but in left-sided failure it will usually be low. This is serious because the coronary flow will decrease and therefore the blood pressure should be kept as high as possible with vasopressors. In atelectasis the blood pressure varies with the seriousness of the condition. In pulmonary embolism it will be low.

Pathology.

The treatment of these conditions depends on an understanding of the pathology. Time will not permit me to go very deep into this, but I would like to make a few comments.

In bleeding—as already mentioned—the peripheral arteries contract and the tissues suffer from anoxia. Blood transfusion is the obvious treatment and should be started as soon as possible and continued during the operation to stop the bleeding. If blood is not immediately available some sort of plasma expander should be used until blood is available.

Post-operative shock is primarily due to loss of blood and plasma and is characterized by a decrease of circulating blood, capillary dilatation, and dehydration. In this form of shock blood transfusion is the best treatment.

Sufficient blood should be transfused to restore the circulating blood volume. Clinically this is indicated by a normal filling of the veins and the hands and feet becoming warm, dry, and regaining the normal colour of the skin.

The circulating blood volume can now be determined, using the radio-iodinated human serum albumin (1_{131}) as tracer substance. The method is based upon an isotope dilution method and an apparatus, called Volemetron, automatically makes all measurements and computions, and gives the answer in twenty minutes with an accuracy of ± 5 per cent. In open-heart operations, when the blood loss during the operation is sometimes difficult to estimate, this method has been of great help to us.

Sometimes it looks as if the arterioles and capillaries are not able to contract and great amounts of blood are still pooled in the periphery. In such cases a permanent infusion of contracting agents, like aramin or nor-adrenalin, may have a good effect. In other cases it seems that the arterioles are in a sort of spasm. You will then find a patient with white goose skin, cold hands and feet, and hyperthermia. The temperature in the rectum may be 39-40° C. and the skin temperature on the feet around 20° C. In such cases the use of chloropromazine may have a dramatic effect, but you must be ready to pump in blood or plasma expander when the vessels open up, follow the blood pressure, the filling of the neck veins and the temperature, and if necessary put the patient in the Trendelenburg position. This form of treatment has also had a dramatic effect in patients who have been overtreated with nor-adrenalin or if this treatment has been given

on a wrong foundation. By treating patients with shock and hyperthermia with ganglion blockers you will see the patient's skin become red and warm, the skin temperature on the feet go up and the temperature in the rectum fall. The climatic surroundings are important. Observe how the patient is covered, the temperature of the room and the circulation of air. Many problems connected with this have only recently been studied scientifically.

Right-sided heart failure is usually due to increased resistance in the pulmonary circulation. The pulmonary blood flow is still difficult to measure, but with the introduction of right-sided heart catheterization and mediastinal puncture we have at least in recent years learned much about pulmonary pressure and resistance. Many conditions will increase pulmonary resistance and pressure, for example, failure of the left ventricle, mitral stenosis, cardio-vascular changes in the lungs in left to right shunt, pulmonary fibrosis, and emphysema. Such condition may be found both in young and old patients. The old-fashioned treatment was venesection, morphia, and oxygen.

Increased pulmonary resistance with or without right-sided heart failure will reduce the pulmonary ventilation and produce cyanosis (hypoxia) and CO_2-accumulation.

If oxygen is given the cyanosis may disappear, but the respiration becomes superficial and CO_2-retention will increase. The clinical signs of CO_2-retention are: a rise in blood pressure, sweating and drowsiness, ending in coma.

Arterial puncture with determination of the oxygen saturation percentage and the carbon dioxide pressure gives an accurate picture of the inefficient ventilation.

Today we have at our disposal a rational treatment, which has saved many lives. That is early tracheostomy and artificial respiration, either manual or using a respirator, which usually is more efficient and, if necessary, may be continued for days or weeks.

In left-sided heart failure oxygenation of the heart muscle is the main objective. As already mentioned, the blood pressure must not be allowed to drop. Oxygen treatment is important. A new and promising treatment is now in the experimental stage, namely, left-sided by-pass through a heart and lung machine or an arterial pumping device as introduced by Harken in Boston.

The prevention and treatment of atelectasis is today known to everybody, thanks to the pioneering work done in Great Britain.

The conservative treatment of pulmonary embolism is to put the patient up in half-sitting position, administering oxygen and euphyllin.

Pulmonary embolectomy has been taken up again with the aid of extracorporeal circulation. I have very little personal experience. Fortunately pulmonary embolism has become rare, but let me say only that a condition for success is that a patient with severe pulmonary embolism is immediately transferred to the operation room or an adjacent recovery ward, where the doctors who will eventually perform the operation can decide when it is time to do it. It is useless to call the thoracic surgeon when the patient is close to death. Today

the operation should be performed by the aid of extracorporeal circulation and a pump-oxygenator must be ready. If the operation should succeed, experience shows that it should immediately be followed by a ligation of the inferior caval vein below the renal veins to prevent new emboli.

I don't think that I have told you anything that you have not heard before, but I hope to have said it in a slightly different way which may be of use to some of you.

	Bleeding	Shock	Heart Failure (right)	Heart Failure (left)	Stomach Distension	Atelectasis	Pulmonary Embolism
Psyche	anxiety or quiet	apathy	anxiety	anxiety	apathy	anxiety	anxiety
Respiration	quick, snapping air hunger	quick shallow	sitting fight for air	quick shallow	shallow	quick shallow frustration cough	sitting up fight for air
Skin	white, yellow	pale bluish wet	red-cyanotic	pale	pale bluish wet	cyanotic	cyanotic sweating
Veins	contracted	contracted	distended	contracted		normal	distended
Pulse	small, quick	small, quick	fair	small, irregular	small	quick	small, quick
Blood Pressure			normal	low	low	normal	low
Pathology	lack of circulating blood dehydration	capillary dilatation	high pulmonary resistance	coronary disease	reflex?	obstructed airways	pulmonary arterial spasm
Therapy	blood transfusion plasma expander hæmostasis	blood transfusion ganglion block. agent oxygen	morphia venesection oxygen sitting position trache-ostomy and respirator	oxygen nitroglycerin left by-pass vaso-pressor	aspiration	bronchial aspiration physiotherapy	oxygen sitting up euphyllin embo-lectomy