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ANNIVERSARY ADDRESS

Visceral Pain: Some Facts and Speculations

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According to rules and conventions the Anniversary Address of the President is generally on a subject of his speciality. For the past twenty years my interests have focussed largely on visceral mechanisms and so I decided to talk to you about the new hypothesis of visceral pain. All that I am going to present here has already been published earlier or more recently and I have therefore decided not to write up another review article that can serve as an Anniversary Address but rather to give an illustrated talk that will help you understand the reasons for advancing another hypothesis to explain visceral pain. Elsewhere, I have referred to this hypothesis as the inadequate parallel block hypothesis for visceral pain because this best describes what it is. But it is simplest to say that visceral pain occurs due to the absence of certain normal blocking mechanism. The two existing hypotheses of visceral pain, are the intensity hypothesis and the specificity hypothesis.

The new hypothesis arose from our studies on the J receptors. These receptors are located in the lungs. All sensory receptors are meant for signalling something—giving some kind of information to the brain (and other parts of the central nervous systems) which then interprets the information as a feeling of some kind and responds appropriately in the form of reflexes. So also the J receptors. These are stimulated during exercise by increased blood flow in the lungs which leads to increase in the fluid in the tissue in which the receptors lie. Thus, they signal the wetness of the tissue in which they lie. Actually this tissue is a sort of sponge that swells and the degree of swelling is signalled by the J receptors. The important point to keep in mind is that the J receptors are normally stimulated during exercise and the main reflex effect of these receptors is to terminate exrecise. From the reflex effects produced by them, the sensations produced by lobeline and the effects of high altitude pulmonary oedema (HAPO) on young soldiers, it was established that the J receptors not only produced breathlessness that had been suspected for several years (actually since 1955) but they also produced dry cough. This finding is of cardinal importance, especially in elderly people and those who have had coronary artery disease.

The J receptors not only produce the sensations of tickling in the throat leading to dry cough but also pain in the central part of chest i. e. substernal pain to be more precise. Now comes the main question. Why is it, that if the J receptors are stimulated effectively during exercise, most normal people do not experience any pain in the chest during exercise—why do dry cough and pain occur only if they are stimulated by lobeline and during HAPO? The answer is that no pain or dry cough occurs because during exercise these sensations are blocked by certain inputs from other parts of the body (e.g. muscles and also brain) that block the pathways that produce pain and irritation in the throat. Thus, if the J receptors are stimulated by any stimulus during rest you will get dry cough and also pain in the chest if the stimulus is strong enough. So this is the crux of the blocking hypothesis for the *absence* of visceral pain. My collaborators Dr Hans Raj and Dr V K Singh have shown experimentally that this is true by looking at the sensations produced by lobeline during rest and during exercise and so I think the validity of the hypothesis is established in the case of the nociceptive sensations produced by J receptors.

However, one of the sensations that is not blocked is the sensation of muscle weakness which everyone feels as a part of the complex of sensations involving breathlessness during moderate or more severe muscular exercise. This sensation of muscle weakness is the result of J receptors and so this reflex is called the J reflex. Man interprets this inhibition of muscles as a feeling of muscle weakness because man needs to put in more effort to overcome the inhibition (of the muscles) over which we have no control.

Let us come back to the new hypothesis. It is clearly valid in the case of the sensations produced by the J receptors. The question that now arises is: Is it also valid in the case of other types of visceral pain? The answer is may be in the case of pain originating from the heart. Firstly it may be noted that no pain occurs when the receptors in the heart are greatly stimulated during exercise because exercise generates inputs that block the pain pathways. Pain occurs when the same receptors are stimulated during rest when the blood supply to the heart mucle is reduced. This view has great practical importance because there are many people with past history of cardiac ischaemia (i.e. previous "heart attacks") who are advised to do exercise to improve the blood supply to the heart muscle. Many of these people get pain when they start exercise but they don't stop there. Instead they continue to do even more vigorous exercise and then find that the pain is less or gone. They think that this is so because jogging has imposed the blood supply to heart. May be this is so in some cases but in many others it is not. In the latter the reduction of pain is due to the operation of the exercise blocking mechanisms that block the pain pathways. So the jogger does not feel pain even though his heart is getting much less blood than it needs. This I think is the reason that has led to the occurrence of heart attacks during exercise in many people. One of the most noteworthy ones is that of astronaut James Irwin. The other is the equally well known "Running Guru" Jim Fix, the author of the "Complete book of running" who died of a heart attack at the age of 52 while jogging on a back road in Vermont. So the important message from this is that you can be fooled by the blocking mechanisms. Therefore, if you have had a heart attack before or evidence of reduced blood supply to the heart you must not continue with exercise (jogging, tennis, etc.) once you start feeling the familiar pain in the chest and left arm or elsewhere. If you must continue under such conditions you must first have an exercise test in the cardiologists laboratory who will tell you whether the pain disappears because the blood supply to your heart has actually improved.

Now I must tell you about the great significance of dry cough, breathlessness and music weakness. There is a tendency to dismiss dry cough as not being of much consequence because we have it so often and it is harmless. This is no doubt true because the commonest cause of cough is infection of the upper respiratory passages. But in the case of the elderly and people with evidence of left ventricular insufficiency it is of utmost importance if the dry cough occurs alongwith breathlessness during mild exertion or it is associated with muscle weakness. It is important for people especially chest physicians to realise that muscle weakness is a very important sympton in the case of the elderly especially if they have had serious heart or lung problems because it means that the J receptors are being continuously stimulated and so the lung tissue is getting wetter and if notice is not taken of this the person will die because of lack of oxygen.

So, an important message is: In the case of the elderly and those with previous history of heart and lung problems take notice of dry cough with breathlessness, or dry cough with muscle weakness, or breathlessness and muscle weakness. Less than a month ago one of my relations lost her husband because no one around him realised the importance of muscle weakness and the breathlessness that he had. And I know several others who have died prematurely in the same way.