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Will no one tell me what she sings?

Wordsworth's line was once used by a physician to depict his surgical colleague's approach to auscultation. Contemporary cardiologists are, perhaps, less arrogant, and they admit their doubts about exactly what produces heart sounds. Techniques such as intracardiac phonocardiography and echocardiography have helped enormously in elucidating cardiac murmurs, but the empirical physiology of the heart sounds is still not clear. Though this may seem surprising, it should not be altogether unexpected when viewed against the problems of interpretation of the findings from any new techniques, and echocardiography is no exception. Nevertheless, we have made a little progress.[1]

The origin of the first heart sound is controversial, and there are two schools of thought. The classic theory, as enunciated by Leatham,[2] attributes its major elements to vibrations from the closure of the atrioventricular valves: one burst of high frequency vibrations is caused by mitral valve closure and a second results from the tricuspid valve. This theory is opposed by Luisada[3] and his co-workers, who attribute the first high frequency vibrations to the early phase of rise of left ventricular pressure rather than to mitral valve closure and the second vibrations to a left-sided rather than a right-sided event—namely, ejection of blood into the root of the aorta.

Which of these conflicting views is correct? Echo-phonocardiographic studies in various cardiac abnormalities have strongly supported the classic view that the origin of the first heart sound depends on mitral and tricuspid closure. But the source of the sound is now attributed to sudden deceleration of a mass of blood within the ventricle rather than to actual contact of the valves on closure. These studies have also shown the origin of aortic and pulmonary ejection sounds to coincide precisely with the maximal opening of these valves.[1]

As to the second sound, Rouanet's hypothesis of 140 years' standing[4] attributed this to closure of the semilunar valves, with both an aortic and pulmonary component. This has gone unchallenged until the recent echo-phonocardiographic studies of Chandraratna *et al*.[5] These showed a delay between semilunar valve closure and the actual production of the second sound, suggesting that it may be a deceleration of a column of blood in the root of the aorta at the end of systole which causes the audible vibrations. The longer interval between pulmonary valve closure and sound production on the right side would be explained by the increased compliance of the pulmonary arteries. The matter remains unresolved but not uncontested; some workers still strongly support Rouanet's classical theory.[1]

Even the origin of the opening snap of the mitral valve (recognised by any medical student as the hallmark of the diagnosis of mitral stenosis) has been contested and attributed not to an opening event but in fact to reclosure of the valve.[6] There is, however, little support for this theory, though admittedly the snap occurs simultaneously with the termination of the opening of the valve rather than with the pressure crossover between atrium and ventricle.[1]

What are ordinary doctors to make of all this? Despite the confusions some generalisations may be made. The first and second sounds, ejection sounds, clicks, and opening snaps are all high-frequency sounds, which on auscultation are characteristically sharp and short. Their production depends on the deceleration of a mass of blood producing vibrations of the heart chambers, valves, and contents.[1] This concept does not include the lower frequency sounds—the third and fourth sounds—which are probably produced independently of valve closure and result from rapid ventricular filling and altered ventricular compliance respectively. We hope that familiarity with comparatively new non-invasive investigatory techniques will provide more and possibly final answers to the remaining questions.

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Ankylosing vertebral hyperostosis

In 1950 Jacques Forestier and Rotes-Querol[1] reported a condition predominantly affecting elderly men in which stiffness of the spine was associated with radiographic changes due to bony outgrowths arising from the anterolateral aspects of the vertebral bodies. Six years later a monograph[2] published in English emphasised the distinction of this syndrome from ankylosing spondylitis. Ankylosing vertebral hyperostosis, well recognised in Europe, differs from ankylosing spondylitis in affecting men over the age of 50, in its predilection for the dorsal spine, sparing the sacro-iliac joints, in the lack of pain and morning stiffness (it is frequently symptomless), and in the characteristic radiological picture. The spinal bony outgrowths may extend upwards, producing an appearance on the *x*-ray films like that of a candle flame, or downwards, giving a picture like that of candle wax dripping downwards.[1–3] The two may join to form a continuous sheet of bone anterior to the vertebral bodies. Laboratory findings are usually negative, with normal sedimentation rates and serum calcium, phosphate, and alkaline phosphatase concentrations, but diabetes mellitus may coexist. Forestier's disease is essentially a radiological rather than a clinical syndrome, to be distinguished from paraspinal ossification in psoriatic arthropathy,

the various ankylosing spondylarthropathies, and chronic fluorosis, and from the spinal changes in acromegaly, hyperparathyroidism, ochronosis, and simple degenerative and post-infective spondylarthropathies. Being relatively benign and often symptomless, its importance lies essentially in the diagnostic confusion it may cause with these other conditions and in its coexistence in some cases with diabetes mellitus. It is not an uncommon condition, Vernon-Roberts and his colleagues[4] finding it in 20 of about 500 necropsies.

Recently a study has been reported from the United States of America by Utsinger, Resnick, and Shapiro.[5] Of their 30 patients 25 were men and 5 women, their ages ranging from 50 to 85 and averaging 67 years. In 23 the main complaint was of stiffness in the thoracolumbar spine of many years' duration, usually mild, intermittent, and non-radiating. This spinal stiffness was most apparent first thing in the morning and faded within an hour with simple movements. Normal spinal flexion, extension, and lateral rotation were present in all patients. Eleven of their patients complained of peripheral bone and joint pain, and in seven this was the main or only complaint, the areas affected being shoulder, hip, knee, and ankle; five had acute synovitis of a single joint. Eight had pain in the heels and in five it was the chief complaint. Laboratory investigations were essentially negative, and no evidence was found of abnormality of serum growth or parathyroid hormones; 132 samples of drinking water consumed by 11 patients showed no fluoride concentration levels above 2 parts per million. Only five patients had fasting hyperglycaemia; three others had gout. Every one of the 30 patients had hyperostosis of the thoracic spine and in 21 it affected the whole spine. A radiolucency was often seen between the newly deposited bone and the underlying vertebral body, and extraspinal manifestations included irregular new bone "whiskering" of the pelvis, large bony spurs, mostly on olecranon and calcaneus, and obvious calcification of sacrotuberous, iliolumbar, and patellar ligaments.

As Forestier and Rotes-Querol[1] stated in their original paper the mild nature of the symptoms gives little indication for active treatment: the purpose of their publication was to distinguish the condition from ankylosing spondylitis and spinal osteoarthrosis. It is referred to in Hollander and McCarty's textbook on arthritis only in the radiological section[6]; most general textbooks, with a few exceptions,[7] do not mention the condition. Pathological findings are also scantily reported. The association with diabetes mellitus and obesity has been noted,[8–11] and Harris and her co-workers[12] found 4 diabetics in a series of 34 patients with ankylosing hyperostosis. Pain in some region of the back was found in 29 of their cases, and, like Utsinger and his colleagues,[5] they found peripheral joint complaints not uncommon, 24 of their 34 patients having such symptoms. The diagnosis centres essentially on the spinal radiological findings, but the American workers found the extraspinal features so common that they suggested the title "diffuse idiopathic skeletal hyperostosis." The shorter title suggested by Bywaters and Forestier[13] at the 6th European Congress of Rheumatology, ankylosing hyperostosis, seems preferable.

Utsinger *et al* described their recent paper[5] as "diffuse skeletal abnormalities in Forestier disease"—a title which underlines the debt rheumatology and, indeed, general medicine, owes to this brilliant Frenchman. From his early studies with Sicard on radiographic techniques with iodised oil in the 1920s, his extensive work later on the place of gold salts in the treatment of rheumatoid arthritis, his pioneer research on ankylosing spondylitis, and many other contributions over the years, Jacques Forestier has won the highest place in the esteem and affection of his colleagues. His life history shows that great clinicians can still extend and improve medical knowledge and understanding. Forestier's original description of senile ankylosing hyperostosis of the spine is worth rereading as an example of clarity in the description of clinical and radiological findings.

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Cannabis psychosis

Psychotic reactions associated with the use of cannabis may be acute or chronic. As long ago as 1843 O'Shaughnessy[1] described acute cataleptic reactions among his patients who were prescribed cannabis, and the next year Jacques Moreau[2] drew attention to hallucinatory phenomena. Since then a series of reports have documented similar and other acute undesirable reactions to cannabis, either self-administered or taken for research purposes.[3–10] These acute psychotoxic reactions generally last a few hours but very occasionally as long as seven days. The risk of a reaction seems to be dose-related, though novices may experience one from quite small amounts—after smoking a single cigarette. The main features have been described[11] as including "paranoid ideas, illusions, hallucinations, depersonalisation, delusions, confusion, restlessness and excitement. In occasional instances there may be additional features of delirium, disorientation, and marked clouding of consciousness." Severe panic may also be prominent.

While this acute toxic reaction may legitimately be classified as a cannabis psychosis the term is more usually reserved for mental disturbances developing in the setting of long-term use, and there is a great deal of controversy about the part played by the drug. These doubts are partly due to the control the chronic user learns over the effects of the drug but also theoretical and methodological.[12 13] Briefly (and apart from the question of what constitutes psychosis) there is little evidence to suggest that adverse reactions in long-term users occur more often than in other comparable populations. Most reports have paid too little attention to factors other than use of cannabis, such as heredity, environment, personality features, and previous psychiatric history, all of which may affect the vulnerability of the individuals concerned. Furthermore, there does not seem to be any consistent relationship between