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VAN DIE REDAKSIE

BELUISTERING BY MYTERKLEPVERNOUING

Van al die prosedures wat by die moderne diagnose van hartkwale beoefen word, is beluistering die waardevolste. Myterklepvernouing is 'n goeie voorbeeld van die waarde van hierdie tegniek, want hier word noukeurige beluistering met 'n magdom van inligting beloon.

By 'n tipiese geval van 'n baie nou stenose (sonder enige komplikasies) van die myterklep, sal beluistering by die hartpunt in elke siklus 5 liggaamlike tekens uitlok. Hierdie tekens kom in die volgende orde voor: 'n voorsistoliese geruis; 'n harde klap as 1ste hartklank; 'n normale 2de klank; 'n oopgaan-klapgeluid; en 'n lae, rammelende middel-diastoliese geluid. Hierdie volgorde is in 1862[1] deur Duroziez uitgeken, toe hy dit foneties as ‚ffout-tatarou' beskryf het. ‚Ffout' is die voorsistoliese geluid wat aangroei tot 'n harde 1ste hartklank; ‚tata' is die 2de klank waarop die oopgaan-klapgeluid volg; en ‚rou' is die middel-diastoliese geluid. Elke onderdeel van hierdie klassieke vyftal word op sy beurt bespreek.

Die *voor-sistoliese geluid* word veroorsaak wanneer die oorvergrote linker-voorkamer teen die einde van die ventrikulêre diastool saamtrek en die bloed deur die vernoude myteropening forseer. Dit is dus gedemp by voorkamertrilling, en word nie gedurende vroegtydige kamersistool gehoor nie. As daar ernstige hartversaking is, kan die vergrote linker-voorkamer nie die bloed sterk genoeg stoot om 'n hoorbare effek te hê nie, en dan verdwyn die voor-sistoliese geluid. Hierdie geluid verskyn ook nie wanneer kwaai drukverhoging in die longslagaar die bloedstuwing deur die linkerhart drasties verminder nie.[2] As 'n eersterangse hartblokkade teenwoordig is, kan hierdie geluid geskei word van die 1ste hartgeluid[3] en ietwat vroeër in die diastool voorkom. By algehele hartblokkade, met totale afsluiting tussen die voor- en die hartkamer, kom voorkamer-sistoolgeluide nog voor, maar hulle het dan geen konstante betrekking op die hartklanke nie.

Die *harde 1ste hartklank* ontstaan op die volgende manier: Aan die einde van diastool forseer die hoë druk in die linker-voorkamer die slippe van die klep diep in die linkerkamer in. Die daaropvolgende skielike sametrekking van die kamer druk die klepslippe teen

EDITORIAL

AUSCULTATION IN MITRAL STENOSIS

The most useful single procedure in modern cardiological diagnosis is auscultation. Its value is well illustrated by considering mitral stenosis, in which diligent auscultation is rewarded by a wealth of information.

In a typical case of uncomplicated, tight mitral stenosis, auscultation at the cardiac apex will elicit 5 physical signs in each cycle. These are, in sequence, a presystolic murmur, a loud, snapping 1st heart-sound, a normal 2nd sound, an opening snap, and a low-pitched, rumbling mid-diastolic murmur. This sequence was recognized by Duroziez in 1862,[1] when he characterized it phonetically as 'ffout-tatarou'. 'Ffout' is the presystolic murmur, rising in crescendo to a sharp 1st heart-sound; 'tata' is the 2nd sound followed by the opening snap, and 'rou' is the mid-diastolic rumble. Each component of this classical pentalogy will be considered in turn.

The *presystolic murmur* is produced by the hypertrophied left auricle when it contracts towards the end of ventricular diastole and forces blood through the narrowed mitral orifice. It is therefore abolished by auricular fibrillation and is not heard during ventricular premature systoles. When there is gross cardiac failure, the dilated left auricle is incapable of propelling the blood with sufficient force to produce an audible effect, and the presystolic murmur disappears. The murmur is also abolished when severe pulmonary hypertension drastically reduces the blood flow through the left heart.[2] In the presence of 1st-degree heart-block (i.e. prolonged auriculo-ventricular conduction) the murmur may be separated from the 1st heart-sound[3] and assume a position somewhat earlier in diastole. In complete heart-block, with total dissociation of auricular and ventricular activity, auricular systolic murmurs still occur but bear no constant relation to the heart sounds.

The *loud 1st heart-sound* is produced as follows: The high left auricular pressure forces the cusps of the mitral

mekaar, en omdat hulle ietwat verstyf is, gaan hulle met 'n kortaf klapgeluid toe—die ‚toegaan-klapgeluid van die myterklep'.[4] Hierdie teken toon dus aan dat die druk in die linker-voorkamer verhoog is, en dat die klepslippe, nieteenstaande hul ligte fibrose, nog soepel is met betreklik vry chordae tendineae. In so 'n geval behoort dit maklik te wees om die verbinding met die vingers te skei.[4] Die 1ste geluid verloor hierdie hoedanighede as die klep weens hewige fibrose of verkalking heeltemal styf word, en as die chordae tendineae baie littekens dra. Onder hierdie omstandighede kan 'n tegnies moeilike valvotomie verwag word. By eersterangse hartblokkade, waar die klepslippe by die aanvang betreklik naby mekaar is, en by myterklepverswakking, waar hulle nie behoorlik kan toegaan nie, is die 1ste klank kenmerkend sag. Aktiewe hartontsteking is geneig om die 1ste geluid te demp, en by voorkamertrilling varieer die sterkte daarvan volgens die kringloop van die hartslag.

Die *2de hartgeluid* word veroorsaak deur die toegaan van die halfmaankleppe. Die 2de of longslagaarklepgedeelte van hierdie geluid word harder namate die druk in die longslagaar styg. By myterklepvernouing is die tweede geluid nie abnormaal verdeel nie; 'n lang tussenpose tussen die samestellende klanke beteken dat daar belemmering in His se bondel is.

Die *oopgaan-klapgeluid van die myterklep*[5] kom omtrent 0·08 sekondes na die aanvang van die 2de geluid voor. Dit ontstaan wanneer die ietwat verstyfde klepslippe oopgegooi word deur die vinnige instroming van bloed in die linkerkamer vroeg in die diastool. Dit is 'n hoë klank, en hoewel dit die beste gehoor kan word by die onderste rand van die borsbeen of by die hartpunt, is dit dikwels wyd versprei oor die hele hartstreek. Dit is 'n waardevolle teken omdat dit blykbaar kenmerkend is van myterklepvernouing, en omdat dit nie voorkom by aandoenings wat myterklepvernouing naboots met middel-diastoliese en voor-sistoliese geluide nie. Dit is dus afwesig by ventrikulêre tussenskotdefek, by oop ductus arteriosus, skildkliervergiftiging, bloedarmoede, en by 'n gewas in die linkervoorkamer. Ook vergesel dit nie die Austin Flint-geluid nie. As hierdie teken wel voorkom, beteken dit gevestigde myterklepstenose met soepel klepslippe en minimale terugvloeiing. Net soos die harde 1ste klank, kom dit nie voor as die klepslippe vol littekens om met kalk aangeslaan is nie.

Die *rammelende middel-diastoliese geluid* word veroorsaak as die bloed uit die voorkamer deur die vernoude myterklep in die kamer vloei. Die klank word die beste opgeneem met 'n ‚klok'-tipe gehoorpyp net by die hartpunt met die pasiënt links gedraai. Liggaamsoefening kan dit beklemtoon. Die Valsalva-maneuwer kan hierdie geluid uitwis,[2] en dikwels word dit uitgeskakel by groot hartversaking of by hoë druk in die longslagaar.[3] Dit is nie waarneembaar by gevalle waar die myterklep aansienlik onbekwaam is nie.

Benewens hierdie 5 tekens, kan sekere *bykomende* tekens onder bepaalde omstandighede ontwikkel. As ernstige drukverhoging in die longslagaar ontwikkel, sit hierdie slagaar uit, en dan kan 'n vroeë sistoliese klikgeluid[7] in die longstreek gehoor word. Die buitengewoon hoë druk in die longslagaar kan terugvloeiing by die longslagaarklep veroorsaak, as gevolg waarvan

valve deep into the left ventricle at the end of diastole. Then the ventricle contracts, suddenly forcing the cusps together and, because of their slight rigidity, they close with an abrupt snap—'the closing snap of the mitral valve'.[4] This sign thus indicates that the left auricular pressure is elevated and that the cusps, though somewhat fibrotic, are still pliant, with relatively free chordae tendineae. In such a case digital separation of the commissure should be readily accomplished.[4] The 1st sound loses these qualities if the valve is made rigid by excessive fibrosis or calcification or if there is much cicatrization of the chordae tendineae; in these circumstances a technically difficult valvotomy must be anticipated. In 1st-degree heart-block, where the cusps are relatively close together at the onset of systole, and in mitral incompetence, where they cannot shut properly, the 1st sound is characteristically soft. Active carditis tends to subdue the 1st sound and in auricular fibrillation its intensity will vary with cycle length.

The *2nd heart-sound* is produced by closure of the semilunar valves. The 2nd or pulmonary-valve component of this sound increases in intensity as the pressure in the pulmonary artery increases. The 2nd sound is not abnormally split in mitral stenosis; wide separation of its components indicates bundle branch block.

The *opening snap of the mitral valve*[5] occurs about 0·08 seconds after the beginning of the 2nd sound. It is produced when the somewhat stiffened cusps are flung open by the rapid inflow of blood into the left ventricle during early diastole. It is a high-pitched sound and although best heard at the lower left sternal border or at the cardiac apex, it is often widely conducted over the praecordium. It is a valuable sign because it seems to be pathognomonic of mitral stenosis, and does not occur in conditions which simulate mitral stenosis by producing mid-diastolic and presystolic murmurs. Thus, it is absent in ventricular septal defect, patent ductus arteriosus, thyrotoxicosis, anaemia and left auricular tumour, and does not accompany the murmur of Austin Flint. Its presence indicates established mitral stenosis with pliant cusps and minimal regurgitation. Like the loud 1st sound, it is absent when the cusps are heavily scarred or plastered with calcium.[6]

The *mid-diastolic, rumbling murmur* is produced by the flow of blood from auricle to ventricle through the stenosed mitral orifice. It is best heard through a bell-type stethescope just medial to the apex, with the patient inclined to his left side. It may be accentuated by exercise. The Valsalva manoeuvre may obliterate this murmur[2] and it is often abolished by gross cardiac failure or by severe pulmonary hypertension.[3] It is not heard in cases with considerable mitral incompetence.

In addition to these 5 signs, certain *additional* signs may develop under special circumstances. When severe pulmonary hypertension develops, the pulmonary artery dilates and an early systolic click[7] may be heard in the pul-

Graham Steell se vroeë, blasende diastoolgeluid voorkom. Die 3de hartgeluid is nie opvallend by myterklepvernouing sonder komplikasies nie; as dit voorkom, beteken dit moontlik aktiewe ontsteking in die hart of onbekwaamheid van die myterklep. Dit kan onderskei word van die oopgaan-klapgeluid omdat dit laer gestem is en baie later in die diastool voorkom. In so 'n geval is myter-valvotomie nie raadsaam nie.[3]

Ten laaste kan daar 'n *sistoliese geluid* wees wat groot versigtigheid by vertolking verg. As dit sag is en slegs tot 'n gedeelte van die sistool beperk is, is dit waarskynlik van min belang. As dit egter gedurende die hele sistool en selfs verby die hartpunt hoorbaar is, dui dit myterkleponbekwaamheid aan.[8] By 'n nou stenose van die myterklep kan sommige pansistoliese geluide ook by die hartpunt voorkom; hulle ontstaan gewoonlik as die drieslippige kleppe weens verhoogde druk in die longslagaar gebrekkig funksioneer. Hierdie geluide word kenmerkend harder by inaseming[9] en word vergesel van voelbare oorvergroting van die regterkamer, sistoliese polsing in die lewer, en 'n merkbare sistoolgolf in die nek-aarpols. So 'n sistoliese geluid is nie 'n teenaanwysing vir 'n myterklep-operasie nie—dit verg dit eerder.

1. Duroziez, P. (1862): Aangehaal deur Ongley *et al.*, *loc. cit.*
2. Wood, P. (1954): Brit. Med. J., **1**, 1051 en 1113.
3. Ongley, P. A., Sprague, H. B. en Rappaport, M.A. (1955): New Engl. J. Med., **253**, 1049.
4. Holmes Sellors, T., Evan Bedford, D. en Somerville, W. (1953): *Ibid.*, **2**, 1059.
5. Mounsey, P. (1953): Brit. Heart J., **15**, 135.
6. Wynn, A. (1953): *Ibid.*, **15**, 214.
7. Leatham, A. en Vogelpoel, L. A. (1954): *Ibid.*, **16**, 21.
8. Brigden, W. en Leatham, A. (1953): *Ibid.*, **15**, 55.
9. Muller, O. en Shillingford, J. (1954): *Ibid.*, **16**, 195.

monary area. The unusually high pressure in the pulmonary artery may produce regurgitation at the pulmonary valve, resulting in the early, blowing diastolic murmur of Graham Steell. The 3rd heart-sound is not obvious in uncomplicated mitral stenosis; its occurrence suggests active carditis or mitral incompetence, and it may be distinguished from the opening snap by its lower pitch and because it occurs much later in diastole. In its presence, mitral valvotomy is generally not advisable.[3]

Finally, there may be a *systolic murmur*, and its interpretation requires careful judgment. If it is soft and confined to part of systole only, it is probably of little consequence. If, on the other hand, it occupies all of systole and is conducted beyond the apex, mitral incompetence is indicated.[8] Some loud pansystolic murmurs may, however, occur at the apex in tight mitral stenosis; these usually arise from tricuspid valves which are functionally incompetent because of pulmonary hypertension. Such murmurs characteristically increase with inspiration[9] and are accompanied by palpable right ventricular hypertrophy, by systolic pulsation of the liver, and by a prominent systolic wave in the jugular venous pulse. Such a systolic murmur does not contraindicate mitral valvotomy, but rather calls for it.

1. Duroziez, P. (1862): Quoted by Ongley *et al.*, *loc. cit.*
2. Wood, P. (1954): Brit. Med. J., **1**, 1051 and 1113.
3. Ongley, P. A., Sprague, H. B. and Rappaport, M. A. (1955): New Engl. J. Med., **253**, 1049.
4. Holmes Sellors, T., Evan Bedford, D. and Somerville, W. (1953): *Ibid.*, **2**, 1059.
5. Mounsey, P. (1953): Brit. Heart J., **15**, 135.
6. Wynn, A. (1953): *Ibid.*, **15**, 214.
7. Leatham, A. and Vogelpoel, L. A. (1954): *Ibid.*, **16**, 21.
8. Brigden, W. and Leatham, A. (1953): *Ibid.*, **15**, 55.
9. Muller, O. and Shillingford, J. (1954): *Ibid.*, **16**, 195.