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VAN DIE REDAKSIE

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Die siektebeheer van akute verstopping van die hart is grotendeels toegespits op die voorkoming en behandeling van die komplikasies. Omtrent die helfte van die pasiënte wat die ramp self oorleef, sal rustig herstel. By die ander word die herstel bemoeilik deur talle komplikasies—hartversaking, aritmie, propvorming, hartbreuk en skok. Die gebruiklike behandeling vir hartversaking is met digitalis, soutonthouding en diuretika. Terloops kan dit weer beklemtoon word dat matige dosisse digitalis *wel* by hartverstopping gebruik kan word. Die aritmies word volgens hulle oorsprong behandel; die meeste boesem-aritmies word doeltreffend met digitalis behandel, terwyl dié wat in die kamers ontstaan met chinidien of procaine amied beheer word. Procaine amied mag slegs in noodgevalle binneaars gegee word, en dan moet dit met groot versigtigheid geskied omdat dit die bloeddruk verlaag.

Daar is geen bepaalde behandeling vir die komplikasies van propvorming en hartbreuk nie, maar hulle voorkoms kan verminder word met geskikte voorbehoedende behandeling. Middels teen bloedstolling is aangewese vir die voorkoming van klontvorming. Hoewel die doeltreffendheid van hierdie behandeling grondig bevestig is, kan dit nogtans op sigself verantwoordelik wees vir 1·7 persent van die sterftes weens bloeding.[1] Baie gesaghebbendes behandel dus nie hulle ligte gevalle, by wie propvorming seldsaam is, met koaguleringverhoedmiddels nie. Hulle beperk die gebruik daarvan tot die ‚kwaai gevalle'[2] wat gekenmerk word deur 'n geskiedenis van vorige verstopping, of wat gepaard gaan met onbeheerbare pyn, hartversaking of skok, waar propvorming betreklik dikwels voorkom.

Die gevaar van hartbreuk kan verminder word as die genesing van die wond in die hartspier vergemaklik word. 'n Streng ruskuur moet toegepas word gedurende die eerste 2 weke, wanneer nekrose van die spiere en akute ontstekingsinsyfering in die selle die weefselbeeld oorheers, en wanneer hartbreuk die meeste voorkom. Teen die einde van die tweede week, wanneer die bindweefsel-vesels al goed herstel, word die pasiënt toegelaat om homself te voed en self sy ligte toilet te ver-

EDITORIAL

SHOCK IN ACUTE CARDIAC INFARCTION

The management of acute cardiac infarction is largely concerned with the prevention and treatment of its complications. Of the patients who survive the immediate catastrophe, about half will recover uneventfully. In the others, the road to recovery is beset with complications—heart failure, arrhythmias, thrombo-embolism, cardiac rupture and shock. Cardiac failure is treated conventionally with digitalis, salt restriction and diuretics. In passing it is well to emphasize again that digitalis in moderate dose is *not* contra-indicated in cardiac infarction. The treatment of the arrhythmias will vary with their site of origin: most supraventricular arrhythmias are effectively treated with digitalis; those arising in the ventricles are controlled by quinidine or procaine amide. Intravenous procaine amide should be given only in emergency and with due caution because of its hypotensive action.

Thrombo-embolic complications and cardiac rupture have no specific treatment, but their incidence may be reduced by the appropriate prophylactic management. Anticoagulants are indicated for the prevention of thrombo-embolism. Although the efficacy of this treatment has been clearly substantiated, it may itself be responsible for a 1·7% mortality from haemorrhage.[1] Many authorities, therefore, do not administer anticoagulants to mild cases, in which thrombo-embolism is rare, but restrict their use to those 'bad cases',[2] characterized by a history of previous infarction, or accompanied by intractable pain, cardiac failure or shock, in which thrombo-embolism is relatively common.

The danger of cardiac rupture may be lessened by facilitating adequate healing of the myocardial wound. In the first 2 weeks, when muscle necrosis and acute inflammatory-cell infiltration dominate the histological scene and cardiac rupture is most common,[3] strict rest is enforced. At the end of the second week when

sorg. Na 4 weke, wanneer die meeste pasiënte mag opstaan, is baie lymstof reeds neergelê, maar die pasiënte moet hulle ten minste nog 2 maande lank baie stil hou terwyl die littekenweefsel stewig aangroei.[4]

‚Hartskok' ná hartverstopping is 'n toestand waarby die sistoliese bloeddruk gedurig laer as 80 mm. Hg is, hoewel daar nie ander bloeddrukverlagende omstandighede soos vinnige hartslagonreëlmatigheid, longverstopping, diabetiese suurvergiftiging of 'n beskadiging van die harsingbloedvate ens.[5] is nie. Dit moet onderskei word van die kortstondige bloeddrukverlaging wat tydens die eintlike verstopping plaasvind en wat verbygaan sodra die pyn deur morfien of 'n soortegelyke middel verlig word. Die patogenese van hartskok word nog nie duidelik verstaan nie. Dit word hoofsaaklik veroorsaak deur die onvermoë van die sentrale pompwerking van die beskadigde hartspier om genoeg bloed uit die hart te stoot. Dit word dikwels vererger deur die afwesigheid van 'n vergoedende vermeerdering in die weerstand van die periferale bloedvate, d.w.s. deur 'n instorting van die periferale bloedsomloop. Dit kan ook verder vererger word deur oormatige toediening van morfien, of as die pasiënt in 'n regop houding met die bene laag verpleeg word. Hoe laer die bloeddruk, hoe swakker is die bloedsomloop in die kransslagaar en hoe groter is die verspreiding van die verstopping; 'n noodlottige kringloop word dus ingestel wat die hart se kragvermoë meer en meer benadeel. Vroegtydige en kragdadige behandeling is onontbeerlik as die gewone 80-90 persent kans op die dood vermy wil word.

Behandeling is eerstens gemik op die beperking van die omvang van die hartspier se beskadiging. Hierdie doel word bereik deur die oordeelkundige gebruik van suurstof om die plaaslike bloedloosheid van die hartspier te verbeter; deur die vroegtydige gebruik van binneaarse heparien om die propvormingproses te stuit, en deur die spoedige herstelling van 'n bevredigende diastoliese bloeddruk. Tweedens word daar deur die binneaarse toediening van herhaalde klein dosisse van 'n vinnig-werkende digitalis-glikosied (bv. Ouabain),[6] probeer om die hart se uitstootvermoë te vermeerder. Op hierdie manier word die saamtrekbaarheid van die hartspier vermeerder, en as die digitalis in klein dosisse toegedien word, is daar min kans dat die hartspier te prikkelbaar sal word. Binneaarse[7] of binneslagaarse[8] oortappings van volle bloed om die hart se werkvolume te vermeerder het ook hul voorstanders, en 'n paar dramatiese verbeterings is reeds te boek gestel. Maar by hartversaking met bloedstuwing, wat dikwels 'n verdere komplikasie by hartskok is, kan die oortappings nie in groot genoeg hoeveelhede of teen 'n bevredigende snelheid gegee word nie, en op sigself het hulle nie juis veel gedoen om die sterftesyfer te verminder nie.

Ten laaste kan die behandeling daarop gemik wees om die periferale weerstand te vermeerder, en dit is blykbaar die doeltreffendste van die beskikbare prosedures. Noradrenalien of een van die verwante sintetiese amien-simpatikomimetika word vir hierdie doel gebruik. Hierdie stowwe het hoofsaaklik 'n periferale bloeddrukverhogende werking, maar Sarnoff en sy medewerkers[9] het onlangs aan die hand gegee dat

fibroblastic repair is well under way, the patient is permitted to feed himself and to attend to minor toilet procedures. Much collagen has already been laid after 4 weeks, when most cases are allowed up, but restriction of activity for at least 2 more months is required while sound scarring is established.[4]

'Cardiogenic shock' after cardiac infarction is a state in which the systolic blood pressure is persistently below 80 mm. Hg in the absence of other hypotensive circumstances such as rapid cardiac arrhythmia, pulmonary infarction, diabetic acidosis, cerebral vascular accident, etc.[5] It is to be distinguished from the transient fall in blood pressure which occurs at the time of infarction and which passes when morphine or a similar drug relieves the pain. The pathogenesis of cardiogenic shock is not clearly understood. It is mainly due to failure of the central pumping action of the damaged myocardium to maintain an adequate cardiac output. It is often aggravated by the absence of a compensatory increase in peripheral vascular resistance; that is, by peripheral circulatory collapse. It may be further aggravated by the administration of too much morphine, or by nursing the patient in an upright position with the legs down. The lower the blood pressure, the poorer is the coronary circulation and the greater the spread of the infarction; thus a vicious circle is established to the progressive detriment of the cardiac output. Early and vigorous treatment is essential if the usual 80-90% chance of death is to be avoided.

Treatment first aims at limiting the extent of the myocardial damage. This is achieved by the judicious use of oxygen to reduce the myocardial ischaemia; by the early use of intravenous heparin to halt the thrombolic process and by the early restoration of an adequate diastolic blood-pressure. Secondly an attempt is made to increase the cardiac output by giving repeated small doses of a quick-acting digitalis glycoside (e.g. Ouabain) intravenously.[6] In this way the contractility of the myocardium is increased and if the digitalis is given in small doses the danger of increasing myocardial irritability is minimal. Intravenous[7] or intra-arterial[8] transfusions of whole blood to increase cardiac output have their advocates and some dramatic responses to these procedures are on record. But transfusions cannot be given in effective volume or at adequate rate in the presence of congestive heart failure, which often complicates cardiogenic shock, and on their own they have done little to lower the mortality rate.

Finally, treatment may aim at elevating the peripheral resistance, and this seems to be the most effective of the available procedures. Nor-adrenaline, or a related synthetic sympathicomimetic amine is used for the purpose. These substances have a predominantly peripheral vasopressor action, but Sarnoff and his

hulle in 'n mindere mate ook direk die saamtrekbaarheid van die hartspier kan stimuleer. Hierdie middels vermeerder nie die hartspier se prikkelbaarheid nie, en die hart se bloedvolume word vermeerder sonder dat die kransslagaar meer bloed per eenheid arbeid moet lewer. Noradrenalien self is die kragtigste van hierdie bloeddrukverhogende middels; dit word as 'n stadige binneaarse inspuiting van 5 persent dekstrose in water bevattende 'n 4 mg.-noradrenalienbasis gegee. Dit is noodsaaklik dat die bloeddruk noukeurig dopgehou word gedurende die inspuiting, want as dit te veel of te vinnig verhoog word, kan longedeem intree. Dit is 'n vereiste dat hierdie prosedure met die grootste noulettendheid uitgevoer word, want selfs 'n geringe lek uit die bloedvat kan ernstige weefselversterwing veroorsaak.[10]

Weens hierdie gevare is die sintetiese drukverhogende amiene ingevoer; hulle werking is baie minder drasties, en hulle kan in herhaaldelike spier- en aarinspuitings toegedien word. Hulle sluit die volgende middels in: fenielefrien waterstofchloried (‚neo-synephrine'), hidroksiamfetamien (‚paredrine'), mefentermien (‚wyamine') en isopropielnoradrenalien (‚isuprel'). Laasgenoemde is veral waardevol by gevalle waar hartblokkade ook voorkom.[5] Mits die skoktoestand kort tevore eers ingetree het, is dit in die praktyk gerieflik om eers die werking van die sintetiese drukverhogende middels uit te toets. As daar egter nie 'n dadelike en blywende reaksie is nie, moet 'n noradrenaliendrup dadelik ingestel word. Die spoed van toediening sal bepaal word deur die gevolglike drukverhoging, maar oor die algemeen kan dit aangeneem word dat die uiteindelike kans op oorlewing gering is[5] as méér as 1 mg. noradrenalien per uur nodig is om 'n uitwerking te hê. Nieteenstaande sy beperkings en gevare, is hierdie soort terapie ongetwyfeld waardevol, en die gebruik daarvan het oor die algemeen die hartskok-sterftesyfer met 20 persent verminder.[11]

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associates[9] have recently suggested that to a lesser extent they may also stimulate myocardial contractility directly. These drugs do not increase myocardial irritability and the cardiac output is enhanced without demanding increased coronary flow per unit work. Noradrenaline itself is the most potent of these pressor drugs; it is given by slow intravenous infusion of 5% dextrose in water, containing 4 mg. of nor-adrenaline base. Careful monitoring of the blood pressure is essential during the infusion because, if it is elevated too far or too fast, pulmonary oedema may supervene. A scrupulous technique is also necessary because even slight extravascular leak may cause intense tissue necrosis.[10]

Because of these hazards the synthetic pressor amines have been introduced; they are far less drastic in their effects and may be given by repeated intramuscular or intravenous injection. They include phenylephrine hydrochloride ('neo-synephrine'), hydroxyamphetamine ('paredrine'), mephentermine ('wyamine'), and isopropylnoradrenaline ('isuprel'). The latter is of particular value in cases associated with heart block.[5] In practice, provided the shock is of recent onset, it is convenient to try the effect of the synthetic pressor drugs first. Unless there is a prompt and sustained response, however, a nor-adrenaline drip should be instituted immediately. The speed of infusion will depend on its pressor effect, but in general, if more than 1 mg. of nor-adrenaline per hour is required to obtain an effect, the ultimate chances of survival are slight.[5] Despite its limitations and dangers, this form of therapy is undoubtedly valuable and an over-all reduction of 20% in the mortality from cardiogenic shock has been achieved by its use.[11]

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