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## SUBARACHNOID HAEMORRHAGE

In a thoughtful Lumleian lecture Dr. Elkington[1] has included spontaneous subarachnoid haemorrhage in his review. Conservative treatment of this malady carries a high mortality of 45-60%. After the age of 40 the mortality increases with age. In an attempt to reduce this mortality Elkington advocates that bilateral carotid angiography should be carried out as soon as possible to demonstrate the cause of the haemorrhage and allow surgical treatment. There is by no means complete agreement on the question of exactly when this should be done. McKissock and Walsh[2] hold that it should be performed immediately, provided each common carotid artery in turn can be compressed without ill effect. Of over 400 cases in which angiography was performed during the acute phase[3,4] arterial (berry) aneurysms were shown to be the cause of haemorrhage in 54%, arteriovenous malformations in 11·5%, primary intracerebral or intracerebellar haemorrhage in 7·5%, while no lesions was demonstrated in 27%. It should be noted that post-mortem evidence in any large series indicates that cases with berry aneurysms constitute about 75% of the total. These authors emphasize the need for demonstrating the precise cause of the bleeding so that it can be dealt with surgically, either directly at the site of the defect itself or by some form of carotid ligation.

The anatomical site is also of importance in prognosis, as was shown by Logue[5] in aneurysms of the anterior cerebral and anterior communicating arteries. In these arteries the first bleed seldom kills, but there is a tendency to bleed again with fatal outcome, especially in the second week. The time factor is the factor of major importance.

McKissock and Walsh[2] operated on 52 patients who were in danger of dying from the haemorrhage which caused their admission, and these included all cases in coma and semicoma and those with severe neurological signs. Their mortality in this group was 65·5% whereas in 43 cases of the same grade of severity, treated conservatively, the mortality was 90%. Those who were admitted within 8 weeks of the haemorrhage and were recovering from it, and were therefore in danger of recurrent haemorrhage, formed their second group. In 72 such cases treated surgically the mortality was 12·5% while it was 37·5% in 48 cases treated conservatively. If a period of more than 8 weeks has elapsed since the initial bleed[6] there is only a 20% risk of recurrent haemorrhage, and in 17 cases treated surgically the mortality was 23·5%, while with conservative treatment it was 6%.

These figures all apply to demonstrated arterial aneurysms and it seems that if more that 8 weeks have elapsed since the haemorrhage, conservative treatment is preferable, but that surgery should be undertaken if the period is less than this, provided an aneurysm is demonstrated in a suitable site. Not all would agree to operate during the phase of acute haemorrhage; Elkington points out that with the use of artificial hypothermia and improved surgical techniques early operation will become more frequent. Using artificial hypothermia Botterell *et al.*[7] report a mortality of only 30% in 64 patients operated on within 7 days of subarachnoid haemorrhage.

Prognosis in the angiomatous group is good and surgical treatment is likely to result in cure since the tendency to bleed again is small. It also seems likely that the prognosis is better in the group in which no lesion is demonstrated by angiography.

1. Elkington, J. St. C. (1958): Lancet, **2**, 327.
2. McKissock, W. and Walsh, L. (1956): Brit. Med. J., **2**, 559.
3. McKissock, W. (1956): Ann. Roy. Coll. Surg. Engl., **19**, 361.
4. Walsh, L. S. (1956): Acta radiol. (Stockh.), **46**, 321.
5. Logue, V. (1956): Brit. Med. J., **1**, 473.
6. Hyland, H. H. (1950): Arch. Neurol. Psychiat., **63**, 61.
7. Botterell, E. H. (1958): J. Neurosurg., **15**, 4.

## BLOEDBESINKING

Die ondersoek van bloedbesinking is seker een van die eenvoudigste spesiale ondersoeke in algemene gebruik, en ewe akkuraat uit te voer in die navorsingsinrigting en die verafgeleë spreekkamer in die platteland. Die geskiedenis van die toets is interessant; sy diagnostiese waarde is gering maar sy praktiese waarde is groot.

Biernaki (1891) was die eerste om die verskynsel in siektetoestande te bestudeer.[1] Fåhreus het in 1918 sy waarnemings oor die besinking van eritrosiete gepubliseer, soos gevind in swangerskap,[2] en daarna het hy sy belangstelling uitgebrei na mediese geskiedkunde, en die aandag gevestig op die *crusta inflammatoria* of *phlogistica* van die ou Griekse geneeshere. Die 4 ‚humors' van die oergeneeskunde is in die besinkingsbuis te besigtig, nl: Die flegma, of witlagie, wat as die oorsaak van die siekteproses beskou is, die bloed, en heel onder die ‚swart gal'—die massa gepakte rooibloedselle. Die serum kom ooreen met die ‚geel gal'. Die verhouding van die 4 humors het die basis van die patologie en die patogenese van die outyd gevorm, en verklaar die populariteit van bloedlating oor 'n tydperk van ruim 2,000 jaar.[2]

Die suspensie-stabiliteit van die bloed is afhanklik van die vermoë tot *rouleaux* formasie van die rooibloedselle, 'n meganisme wat nog nie ten volle verklaar is nie. Waarskynlik speel fibrinogeen 'n belangrike rol en om dié rede beïnvloed die fibrinogeen-gehalte die besinking merkwaardig. Die proteïen-fraksies in die bloed is ewe belangrik, indien nie meer belangrik nie. Fibrinogeen, globulien en albumien

het in hierdie volgorde effek op die hoogte van die lesing—albumien het in werklikheid 'n negatiewe korrelasie.[2] 'n Formule bestaan waarvolgens die besinking vanaf genoemde 3 proteïenwaardes, bereken kan word met 'n akkuraatheid van binne 20% deviasie. Die grootte van die rooiselle, die pH en oksigenasie van die bloed, asook die aantal rooiselle, beïnvloed die besinking. Die temperatuur en die lesitien-cholesterol verhouding is ook belangrik.[1,2]

Die besinking in anemie is wisselvallig en moet gesien word as die totaal van verskeie faktore: (1) Soos die rooiselle verminder, so styg die besinking—'n basiese fisiese eienskap. (2) Faktore bestaan in anemie wat die effek volkome kan neutraliseer, bv. in 'n mikrositiese anemie sal die kleinheid van die selle hulle stadiger laat sink. Verdunningseffekte verdun insgelyks die plasmaproteïene en vertraag die besinking. Gewoonlik egter is die besinking effe hoër in anemie, maar hierdie faktore kan tot 'n onverwagte hoë òf lae besinking in gevalle met anemie lei. 'n Hoë besinking in 'n anemiese pasiënt is dus nie *noodwendig* die gevolg van die anemie nie.[2]

'n Ander toestand waarin die besinking ook versigtig beoordeel moet word, is in gevalle met sianose. Die verhoogde koolsuurgas-inhoud lei tot swelling van die rooiselle en vertraag die besinking. Om hierdie rede is die besinking dikwels laag in hartversaking en sianose, hoewel in die eersgenoemde geval die lewerstuwing 'n effek op die proteïene mag hê, en dus deels verantwoordelik is vir dié verskynsel.

In akute infeksies word die temperatuurstyging eerste gevind en daarna 'n styging in die witseltelling. Die besinking is selde in die eerste 24 uur verhoog. Hoewel klinies volkome genesing teenwoordig mag wees, bereik die besinking sy piek as die temperatuur eers normaal is en mag dit nog etlike weke verhoog bly as gevolg van metaboliese versteurings in die proteïen-metabolisme. In toestande met weefselnekrose styg die besinking ook, en dan gewoonlik langsamer as in akute infeksies bv. in gevalle met miokardiale infarksie. Die grootste belang van hierdie toets skuil in sy gebruik in gewrigstoestande—'n verhoging dui op aktiwiteit van die proses of op komplikasies.[2] In tuberkulose sal 'n buitengewone snel besinking op 'n komplikasie soos akute pleurale of renale aantasting dui. In aktiewe tuberkulose is die besinking matig versnel, hoewel 'n normale besinking in 2·1% van gevalle van ope tuberkulose gevind is.[2]

Hoewel hierdie toets dus nie spesifiek is nie, behalwe in een geval, het hy die toets van die tyd deurstaan as 'n nuttige prosedure by alle pasiënte. As die geneesheer dus bewus is van die beperkings en die faktore wat dit beïnvloed, styg die waarde van die toets dienooreenkomstig. Die geval waarin die toets spesifiek is, is in sekelselanemie, waar 'n verskil van meer as 20 mm. in die besinking van veneuse en geoksigeneerde bloed gevind word.[3]

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2. Wintrobe, M. M. (1956): *Clinical Haematology*, 4e Uitgawe. Londen: Henry Kimpton.
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