South African Medical Journal
Suid-Afrikaanse Tydskrif vir Geneeskunde
P.O. Box 643, Cape Town Posbus 643, Kaapstad

Cape Town, 26 February 1955 Vol. 29 No. 9 Kaapstad, 26 Februarie 1955
Weekly 2s. 6d. Weekliks 2s. 6d.

EDITORIAL

SERUM PROTEINS

This subject was discussed recently at the Royal Society of Medicine, London, and a series of interesting papers have been published.[1]

The exposure of serum proteins to the influence of an electric field under certain specific conditions causes the proteins to fractionate into groups which migrate towards one of the electrodes at different velocities. By the use of paper electrophoresis analysis of the proteins can be performed even in small laboratories, and a large literature is already available on this subject. Using a standardized technique Flynn [1] finds that adequate information can be obtained by simple visual assessment of the paper strip; and the form of the protein spectrum may yield information of special diagnostic significance; though in most cases the electrophoretic pattern shows only non-specific changes. In certain conditions the routine investigation by this method is of value, e.g. in obscure anaemias. He states in summary that as regards the changes in electrophoretic globulin fractions the alpha globulins are increased in high fever, tissue destruction and the nephrotic syndrome; the beta globulin in conditions in which the serum phospholipids and cholesterol are increased, and in some cases of myeloma; the gamma globulin in chronic infections, liver disease, collagen diseases, and in some cases of myeloma.

In a report on the changes in the serum and urinary protein which are found in the nephrotic syndrome Hardwicke [1] shows that in the serum the albumin is low and the α_2 globulin at a high level; and that the urine contains variable proportions of albumin and the various globulin fractions in different patients, the high-molecular α_2 globulin being virtually absent. He finds that the serum lipid is also much increased, but the urine is strikingly free from lipid. The author shows graphically the alterations in concentration of the globulin components of the plasma as the albumin level falls, and the relationship between serum albumin and the daily urinary protein loss.

In another paper Frazer [1] briefly reviews the present knowledge of the lipoproteins of human blood. These substances have been thought to play some part in the transport of lipids. Recent studies suggest that the levels

VAN DIE REDAKSIE

SERUM-PROTEÏENE

Hierdie onderwerp was onlangs by die *Royal Society of Medicine* in Londen bespreek, en 'n reeks interessante artikels is gepubliseer.

As serum-proteïene onder sekere spesifieke toestande aan 'n elektriese veld blootgestel word, breek die proteïene in groepe op wat teen verskillende snelhede na een van die elektrodes beweeg. Deur middel van papier-elektroforese kan die proteïene selfs in klein laboratoriums ontleed word en daar is baie literatuur oor hierdie onderwerp beskikbaar. Flynn [1] het 'n standaardtegniek gebruik en gevind dat voldoende inligting verkry kon word deur doodeenvoudig die papierstrook met die blote oog te bestudeer; uit die vorm van die proteïenspektrum mag inligting verkry word wat diagnosties van groot belang is, in die meeste gevalle dui die elektroforesepatroon egter net veranderings van 'n nie-spesifieke aard aan. In sekere gevalle is die roetine-ondersoek deur hierdie metode van nut, bv. met verborge bloedarmoede. Met betrekking tot veranderings in die elektroforetiese globuliendele verklaar hy opsommend dat alpha-globulien vermeerder wanneer weefsels tydens 'n hoë koors en die nefrotiese sindroom vernietig word; beta-globulien wanneer die serum-fosfolipiede en die cholesterol toeneem en ook in sommige gevalle van miëloom; die gamma-globulien in kroniese infeksies, lewerkwale, kollageensiektes en in sommige gevalle van miëloom.

In sy verslag oor die veranderings in serum- en urienproteïen wat by die nefrotiese sindroom te voorskyn kom, wys Hardwicke [1] daarop dat die albumien in die serum laag is en die α_2 globulien hoog; dat die verhouding van die albumien en die verskeie globuliendele in die urien van pasiënt tot pasiënt wissel en dat feitlik geen hoë molekulêre α_2 globulien aanwesig is nie. Hy vind ook dat die serumlipied baie toeneem maar dat die urien besonder lipied-vry is. Die skrywer verduidelik met grafieke hoe die konsentrasies globulienbestanddele in die plasma verander na gelang die albumien sak, ook toon hy die verwantskap aan tussen die serum-albumien en die daaglikse verlies van urienproteïen.

Frazer [1] gee 'n kort oorsig van die huidige kennis van die lipoproteïene in mensbloed. Die mening was dat hierdie stowwe 'n rol in die vervoer van die lipiede speel. Onlangse studies suggereer dat die bloedhoogtes 'n aanduiding is van die mate waartoe hul deur die lewer

in the blood reflect the degree of their production and utilization in the liver. The author considers that changes in the blood lipoproteins can be significantly correlated with atherogenesis, and that certain tissue changes may prove to be reversible. The precise significance of changes in the lipoprotein pattern in the blood has still to be elucidated. Most interesting information on the variations in lipoprotein under different conditions is concerned with the amounts of different-sized particles found on ultracentrifugation.

Martin [1] reports some experiences with the transfusion of human albumin and balance studies in patients with chronic liver damage. It was found that after intravenous injection in health and disease relatively rapid passage of albumin may take place through the body compartments and perhaps to the cells themselves. Another point stressed by this author is the ability of albumin as a highly charged ampholyte to interact with small molecules, thus making albumin important in the control of the disposal of small molecules.

Some rare protein disorders have lately been described, as for example agammaglobulinaemia, in which there is complete absence of gamma-globulin; and macroglobulinaemia, in which there is a very dense band in the gamma-globulin, a high percentage of protein in the serum being of high molecular weight.

1. Proc. Roy. Soc. Med. (1954): **47,** 827-838.

vervaardig en gebruik word. Die skrywer is van mening dat verandering in bloedlipoproteïene met die ontstaan van slagaarvervetting gekorreleer kan word en dat sekere weefselveranderings moontlik herroepbaar is. Die juiste betekenis van veranderings in die bloed lipoproteïenpatroon wag nog op verduideliking. Besonder interessante inligting oor variasies in die lipoproteïene onder verskillende omstandighede is betrokke by die hoeveelhede deeltjies, verskillend in grootte, wat met uitermate sentrifugering gevind word.

Martin [1] gee 'n verslag oor ondervindings i.v.m. die oortap van mens-albumien en balansstudies in pasiënte wat aan kroniese lewerbeskadiging ly. Nadat gesonde of siek persone binneaars ingespuit was, was bevind dat die albumien betreklik vinnig deur die liggaam se kompartemente trek, moontlik tot in die selle self. Die skrywer beklemtoon ook die punt dat albumien (as 'n hooggelaaide amfoliet) en die klein molekules op mekaar reageer en dat albumien derhalwe invloed uitoefen op die reëling van klein molekules.

Onlangs is seldsame proteïen-ongesteldhede beskrywe soos bv. agammaglobulieneem, wanneer die gammaglobulien geheel en al afwesig is; makroglobulieneem, wanneer die gamma-globulien 'n baie digte band het, en 'n hoë persentasie serum-proteïen 'n hoë molekulêre gewig besit.

1. Proc. Roy. Soc. Med. (1954): **47,** 827-838.

BLOOD GROUPS AND THE CLINICIAN

Children, it has been said, should choose their parents with care. Everyone knows that the Rh groups of parents are of importance; but until quite recently it seemed to matter little to anyone whether he belonged to Group A, B, AB or O unless he had just received an incompatible transfusion. Haemolytic disease of the newborn, it is true, may be due to ABO incompatibility, but this is rare. In 1950 the authors of a standard work [1] could still observe that, maternal-foetal differences apart, there was no evidence that those of any given blood group were particularly susceptible to any disease. They added that some differences in susceptibility would surely be found. This would be expected on theoretical grounds, since a balanced polymorphism of the kind exemplified by the blood groups needs such selective advantages if it is to be maintained.

Several recent papers have suggested some of the possible advantages and limitations of belonging to particular ABO groups. Aird and his colleagues [2] showed in 1953 that subjects of group A seemed to be significantly more liable to carcinoma of the stomach than those of group O. But the tribulations of the O subjects are not confined to blood-giving, and in the following year Aird's team [3] showed that bearers of group O were apparently very appreciably more prone to peptic ulcer than those of other groups. Pike and Dickens [4] brought forward evidence to suggest that this was also true of toxaemia of pregnancy. Aird *et al.*[3] found no relation between the ABO groups and the incidence of carcinoma of the colon, rectum, bronchus and breast.

Further work will clearly be necessary. Statistical investigations of the kind needed are full of pitfalls, but there seems no doubt that real differences in disease incidence between those of different blood groups have been demonstrated, and that more will be found. From the genetic point of view, the mechanisms of selective advantage and disadvantage are still obscure. Carcinoma of the stomach, for example, usually attacks those who are at or near the end of reproductive life, so that its effect on the A frequency of coming generations must be very small. Probably it is only one of many factors which act in this way. Human population genetics is a most complicated subject, and one of which we know little. The blood-group studies now being undertaken are of obvious clinical interest; but they promise also to contribute something, however indirectly, to our understanding of the mechanism of human evolution.

1. Race, R. R. and Sanger, R. (1950): *Blood Groups in Man,* 1st ed., p. 241. Oxford: Blackwell Scientific Publications.
2. Aird, I., Bentall, H. H. and Roberts, J. A. F. (1953): Brit. Med. J., **1,** 799.
3. Aird, I., Bentall, H. H., Mehigan, J. A. and Roberts, J. A. F. (1954): *Ibid.*, **2,** 315.
4. Pike, L. A. and Dickens, A. M. (1954): *Ibid.*, **2,** 321.