THE MINERAL AND LIPID COMPOSITION OF THE ARTERIES OF WHITE AND BANTU CHILDREN

B. J. MEYER,* D.SC., M.D.; A. C. MEYER,† M.SC., D.SC.; W. J. PEPLER,‡ D.SC., M.D.; AND J. J. THERON,† M.B., CH.B., M.SC., D.SC.

The coronary arteries of Whites are affected sooner and more severely by atherosclerosis and thrombosis than those of Bantu.[1-5] This racial difference is not present, however, in the case of the cerebral arteries.[5-7] These conclusions are based on comparative studies which included clinical investigations and macroscopic and microscopic postmortem examination of arteries.

*Department of Physiology, University of Pretoria.
†National Nutrition Research Institute.
‡Institute of Pathology, University of Pretoria.

Various explanations have been offered for the racial differences found in the one set of arteries and their absence in the others, but none accounts satisfactorily for all the known facts. It was considered that a comparison of the mineral and lipid composition of the arteries of newborn and older children of the two races might supply information of importance to the understanding of the factors which determine the development of atherosclerosis, and such a comparative study, involving the determination of ash, calcium, total lipids, cholesterol, phospholipids and triglycerides, was therefore carried out.

MATERIAL AND METHODS

The arteries of 51 White and 124 Bantu children between the ages of 1 day and 9 years, who had died of natural causes in the Pretoria General Hospital, were collected as described in an earlier publication.[5] The aortas and coronary arteries were found to be surrounded by a rather well-developed layer of fat. The adventitial layer of the aorta and coronary arteries, and where possible of the cerebral arteries also, was stripped. The same procedure was rigidly applied in every case.

Drying of material. The material was dried to constant weight in a vacuum desiccator over concentrated sulphuric acid. This procedure removed about 95% of the moisture. The vacuum-dried material was then ground in a Wiley mill and stored at −2°C in glass containers if not analysed immediately. Before analysis the 5% of moisture remaining after vacuum dehydration was removed by exposing the material to a temperature of 50°C until the weight remained constant.

The dry material obtained from individual vessels was insufficient for complete analysis and the material of different vessels had to be pooled.

Ash and calcium. A weighed portion of the dried material was ashed at 600°C for 5 hours and any remaining organic material was oxidized with nitric acid. The cooled sample was weighed and the calcium concentration determined by a microceric method based on Vogel's macromethod.[8]

Total lipids. Total lipids were determined according to the method of Böttcher[9] and calculated as a percentage of the dry material. The extracted lipid was then dissolved in chloroform and made up to volume. Aliquots of this solution were used for the determination of cholesterol, phospholipids and triglycerides.

Cholesterol. Total cholesterol was determined by the method of Zlatkis *et al.*[10] as modified by Rosenthal *et al.*[11] Free cholesterol was determined by the method of Zak *et al.*[12]

Phospholipids. The method of Brown[13] was employed for the phospholipid studies.

Triglycerides. Triglycerides and cholesterol were separated chromatographically from phospholipids on a silicic acid column.[14-16] After saponification, the glycerol was oxidized to formaldehyde, which gives a red colour with chromotropic acid.[14,17,18]

RESULTS

The mean values for the different constituents studied are presented in Table I. As material obtained from different individuals had to be pooled, the data were not suitable for statistical analysis.

Vascular specificity. Quantitative differences in the lipid composition of the different vascular beds were already present during the first 6 months of life and were maintained in the older age groups (Table I). Attention is drawn in particular to the very high triglyceride concentration of the coronary arteries and the high phospholipid concentration of the cerebral arteries.

Race. No differences worthy of note were found between corresponding vessels of the two races.

Age. The general trend with age was an initial decrease in the mean concentration of the various components determined, followed by a progressive increase. In order to shed more light on the initial decrease in these constituents, the aortas of 24 of the White and 60 of the Bantu children between the ages of 1 day and 8 years were evaluated more critically in respect of age (Tables II and III). Most of the constituents proved to be present in high

TABLE I. MEAN VALUES EXPRESSED AS GRAM PER CENT OF THE DRY MATERIAL

Age in years	*Race*	*No. of cases*	*Index*	*Ash*	*Calcium*	*Total lipid*	*Total chol.*	*Free chol.*	*Ester chol.*	*Phospholipid*	*Triglyceride*
						Aorta					
0–½	W	29	0	2·860	0·075	3·745	0·618	0·385	0·232	1·635	0·456
	B	64	0	2·484	0·092	3·780	0·639	0·394	0·245	1·646	0·462
½–2	W	10	0	1·691	0·070	2·606	0·398	0·269	0·129	1·109	0·314
	B	34	0	1·700	0·072	2·588	0·382	0·264	0·119	1·054	0·309
2–9	W	10	0	1·740	0·057	2·889	0·446	0·274	0·172	1·118	0·357
	B	24	0	1·662	0·058	2·867	0·433	0·300	0·133	1·173	0·334
0–9	W	49	0	2·393	0·070	3·338	0·538	0·339	0·199	1·422	0·407
	B	122	0	2·104	0·080	3·260	0·527	0·339	0·188	1·388	0·394
						Coronary arteries					
0–½	W	31	0	2·757	0·035	6·032	0·739	0·501	0·238	1·634	2·724
	B	66	0	2·447	0·042	5·941	0·756	0·496	0·260	1·874	2·453
½–2	W	10	0	1·706	0·047	6·306	0·742	0·477	0·266	1·612	2·989
	B	34	0	1·621	0·046	5·733	0·629	0·318	0·311	1·500	2·667
2–9	W	10	0	2·261	0·047	6·700	0·659	0·403	0·256	1·392	2·699
	B	24	0	1·780	0·054	5·780	0·645	0·363	0·282	1·497	2·735
0–9	W	51	0	2·454	0·040	6·021	0·724	0·477	0·247	1·582	2·771
	B	124	0	2·091	0·046	5·853	0·700	0·421	0·278	1·699	2·566
						Cerebral arteries					
0–½	W	18	0	3·404	0·056	6·550	1·194	0·804	0·389	3·076	0·995
	B	53	0	2·590	0·049	6·375	1·153	0·724	0·429	2·825	0·376
½–2	W	9	0	2·281	0·064	5·008	0·919	0·635	0·284	2·380	0·346
	B	31	0	2·735	0·051	5·085	0·963	0·651	0·311	2·162	0·306
2–9	W	9	0	3·074	0·077	4·970	0·833	0·569	0·264	2·321	0·338
	B	22	0	2·570	0·056	4·811	0·858	0·586	0·272	2·061	0·296
0–9	W	36	0	3·041	0·063	5·769	1·035	0·703	0·332	2·713	0·369
	B	106	0	2·628	0·051	5·673	1·036	0·674	0·362	2·472	0·339

Abbreviations: W = White subjects (both sexes), B = Bantu subjects (both sexes).

TABLE II. AORTAS OF WHITE SUBJECTS: MEAN VALUES EXPRESSED AS GRAM PER CENT OF THE DRY MATERIAL

Age	No. of cases	Ash	Calcium	Total lipid	Total chol.	Free chol.	Ester chol.	Phospho-lipid	Trigly-ceride
0–1 day	7	3·820	0·080	3·790	0·675	0·430	0·245	1·886	0·401
1 day–1 month	4	3·130	0·068	3·660	0·617	0·389	0·228	1·895	0·377
1–6 months	3	3·000	0·072	2·750	0·502	0·300	0·202	1·305	0·346
7–12 months	2	1·464	0·062	2·564	0·385	0·273	0·112	1·225	0·278
13–18 months	2	1·503	0·066	2·163	0·356	0·244	0·112	1·060	0·225
19–23 months	1	1·465	0·067	2·138	0·351	0·239	0·112	1·116	0·242
2–4 years	3	2·027	0·057	2·517	0·446	0·278	0·168	1·094	0·287
5–7 years	2	2·048	0·057	2·618	0·426	0·245	0·156	1·157	0·296

TABLE III. AORTAS OF BANTU SUBJECTS: MEAN VALUES EXPRESSED AS GRAM PER CENT OF THE DRY MATERIAL

Age	No. of cases	Ash	Calcium	Total lipid	Total chol.	Free chol.	Ester chol.	Phospho-lipid	Trigly-ceride
0–1 day	20	2·965	0·091	4·075	0·719	0·434	0·285	1·969	0·486
1 day–1 month	7	3·050	0·096	3·880	0·611	0·374	0·237	1·911	0·467
1–6 months	6	2·150	0·082	2·845	0·492	0·314	0·178	1·333	0·344
7–12 months	5	1·815	0·072	2·615	0·410	0·282	0·129	1·222	0·324
13–18 months	7	1·958	0·073	2·281	0·347	0·238	0·109	1·059	0·267
19–23 months	3	2·090	0·077	2·345	0·363	0·248	0·116	1·090	0·273
2–4 years	8	1·858	0·058	2·610	0·396	0·267	0·129	1·128	0·305
5–8 years	4	1·908	0·056	2·858	0·444	0·295	0·150	1·270	0·330

concentration at birth, and the decreases usually started shortly after birth.

DISCUSSION

The two elements involved in atherogenesis are: (*a*) the blood-vessel wall and (*b*) the blood in the lumen of the vessel.

In the newborn, the different vascular beds show both histological[19] and chemical differences (Table I). One is tempted to see a link between these inborn differences and the anatomical selectivity of atherosclerosis. However, it is noteworthy that although the aortas and coronary arteries of Whites are much more prone to develop atherosclerosis than those of Bantu,[1-5] the present findings for newborn infants differed little in the two races. It thus appears unlikely that inborn differences in vascular composition can be the primary factor which determines the site of development of atherosclerosis.

Another possibility which must be considered is that the site of development of atherosclerosis may be conditioned by the haemodynamics of the different vascular beds. The forceful rhythmic stretching of the aorta and the rhythmic flexion of the coronaries due to the action of the heart may in some way render these vessels especially susceptible to atherogenesis. The cerebral arteries, which are subjected to less mechanical stress than the aortas and coronaries, are less susceptible to atherogenesis.

Since histological structure, chemical composition and haemodynamic activity appear to be similar in corresponding vascular beds of newborn Whites and Bantu, an explanation outside of the above factors must be sought for the greater liability of Whites to develop aortic and coronary atherosclerosis. Differences in the chemical composition of the blood of the two races may be the answer. Chemical derangements in the blood can be precipitated by a multitude of factors including physical activity, endocrine activity and diet, and racial differences in the chemical composition of the blood have been described.[2,20]

According to Moulton[21] the mammalian body is characterized by a rapid decrease in water content and an increase in protein and ash content from birth onwards, until a stage of 'chemical maturity' is reached. He also states that the most striking change in the composition of the mammalian body that occurs during growth is an increase in fat content. His views on the changes in water, ash and protein content are based on an analysis of the fat-free tissues of the mammalian body. Weisberg[22] has drawn attention to the high water content of the body of infants. At the same time the blood plasma of babies has been stated to contain higher concentrations of sodium, potassium, phosphorus, sulphur, organic acids and proteinate than that of subjects aged from 5 to 25.[22] According to Fomon and Bartels[23] all fat fractions in the blood are very much lower at birth than they are in the adult, but the concentrations are doubled within 14 days. In rats the total lipid and the total neutral fat content of the body has been reported to increase progressively from birth to the age of 70 days. However, free cholesterol, ester cholesterol and phospholipid content were found to be higher in newborn rats than in rats of either 15 or 45 days.[24] Lansing[25] analysed the media of human aortas and found higher elastin and calcium concentrations in the 0 - 10 years age group than in the 11 - 20 years group. The high water content of the newborn child and its gradual decrease with advancing age may partially explain the findings of the present study.

SUMMARY

1. Different vascular beds show quantitative differences in chemical composition at birth.

2. The corresponding vascular beds of newborn White and Bantu subjects differ only slightly in chemical composition.

3. The concentrations of the different chemical substances determined in the present study show an initial decrease before starting to rise.

4. The data do not support the concept that racial differences in susceptibility to atherosclerosis are due to inborn quantitative differences in the chemical composition of the blood-vessels, but seem rather to emphasize the importance of differences in the chemical composition of the blood. The greater susceptibility of certain vascular beds to atherogenesis is for this reason more likely to be due to haemodynamic

factors than to the chemical differences present in the blood-vessels.

We are greatly indebted to Dr. M. L. Neser for her critical reading of the manuscript.

REFERENCES

1. Becker, B. J. P. (1946): S. Afr. J. Med. Sci., **11**, 97.
2. Higginson, J. and Pepler, W. J. (1955): J. Clin. Invest., **33**, 1366.
3. Strong, J. P., Wainwright, J. and McGill, H. C. (1959): Circulation, **20**, 118.
4. Sacks, M. I. (1960): *Ibid.*, **22**, 96.
5. Meyer, B. J., Pepler, W. J., Meyer, A. C. and Theron, J. J. (1964): *Ibid.*, **29**, 415.
6. Laurie, W. and Woods, J. D. (1958): Lancet, **1**, 231.
7. Walker, A. R. P. and Grusin, H. (1959): Amer. J. Clin. Nutr., **7**, 264.
8. Vogel, A. I. (1955): *Textbook of Quantitative Inorganic Analysis*, 2nd ed. London: Longmans, Green & Co.
9. Böttcher, C. J. F. (1959): Nature (Lond.), **183**, 47.
10. Zlatkis, A., Zak, B. and Boyle, A. J. (1953): J. Lab. Clin. Med., **41**, 486.
11. Rosenthal, H. L., Pfluke, M. L. and Buscaglia, S. (1957): *Ibid.*, **50**, 318.
12. Zak, B., Dickenman, R. C., White, E. G., Burnett, H. and Cherney, P. J. (1954): Amer. J. Clin. Path., **24**, 1307.
13. Brown, W. D. (1954): Aust. J. Exp. Biol. Med. Sci., **32**, 677.
14. Carlson, L. A. and Wadström, L. B. (1959): Clin. chim. Acta, **4**, 197.
15. Randup, A. (1960): Scand. J. Clin. Lab. Invest., **12**, 1.
16. Van Handel, E. (1961): Clin. Chim. Acta, **7**, 249.
17. Van Handel, E. and Zilversmit, D. B. (1957): J. Lab. Clin. Med., **50**, 152.
18. Antonis, A. and Behrson, I. (1960): Lancet, **1**, 998.
19. Van Möolendorff, W. (1930): *Handbuch der Mikroskopischen Anatomie des Menschen*. Berlin: Julius Springer.
20. Walker, A. R. P. (1958): Ann. N.Y. Acad. Sci., **69**, 989.
21. Moulton, C. R. *in* Deuel, H. J. jnr. ed. (1955): *The Lipids*, vol. 2, p. 575. New York: Interscience Publishers.
22. Weisberg, H. F. (1962): *Water, Electrolyte and Acid-base Balance*, 2nd ed. Baltimore: Williams & Wilkins.
23. Fomon, S. J. and Bartels, O. J. (1960): Amer. J. Dis. Child., **99**, 27.
24. Williams, H. H., Galbraith, H., Kaucher, M., Moyer, E. Z., Richards, A. J. and Macay, I. G. (1945): J. Biol. Chem., **161**, 475.
25. Lansing, A. I. ed. (1959): *The Arterial Wall*. Baltimore: Williams & Wilkins.