as much as they should. If it is right that when we give treatment and fail we should constantly review our methods and our skills, it is also true that when we avoid the responsibility of intervention we should be reminded of the results of our inactivity.

We can be sure that there will be deaths from surgical treatment but that these will diminish when patients are given a chance earlier, before all the large coronary arteries are extensively involved. Coronary angiography is likely to play an important part in the selection of such patients. If the history of anticoagulant therapy forms any sort of a parallel it will be a very long time before there is conclusive statistical support for surgical treatment. In the meantime it is important that patients should be considered for one or other form of treatment on the basis of the fullest possible investigation so that as numbers do become available they can be useful in statistical surveys.

Summary and Conclusion

Mechanical factors in arterial disease are discussed.

The part played by local arterial obstruction in the production of peripheral emboli is emphasized.

The application of thrombo-endarterectomy to the coronary vessels is described and case reports of six patients are given.

Coronary angiography has an important part to play in the detailed investigation of coronary artery disease. The early demonstration of stenosis or complete obstruction may well be of vital importance in assessing prognosis and in the selection of patients for surgical treatment. The most logical surgical treatment is removal of the arterial obstruction where this is possible.

References

Beck, C. S. (1929). *Arch. Surg.*, **18**, 1659.
—— (1935). *Ann. Surg.*, **102**, 801.
—— and Leighninger, D. S. (1960). *Ohio St. med. J.*, **56**, 809.
Burke, J. F., and Nardi, G. L. (1961). *J. thorac. cardiovasc. Surg.*, **42**, 65.
Cannon, J. A., Longmire, W. P., and Kattus, A. A. (1959). *Surgery*, **46**, 197.
Florey, H. W. (1925). *Brain*, **48**, 43.
—— (1960). *Brit. med. J.*, **2**, 1329.
Honey, G. E., and Truelove, S. C. (1957). *Lancet*, **1**, 1155.
Honour, A. J., and Ross Russell, R. W. (1962). In press.
May, A. M., and Bailey, C. P. (1958). *J. int. Coll. Surg.*, **29**, 160.
Nordenström, B. (1960). *Acta radiol. (Stockh.)*, Suppl. 200.
O'Shaughnessy, L. (1936). *Brit. J. Surg.*, **23**, 665.
Poole, J. C. F., and French, J. E. (1961). *J. atheroscler. Res.*, **1**, 251.
Sabiston, D. C., jun. (1960). *Amer. Surg.*, **26**, 217.
—— Fauteux, J. P., and Blalock, A. (1957). *Ann. Surg.*, **145**, 927.
Satinsky, V. P., Kuhn, R., Kompaniez, E. V., and Baum, R. N. (1959). *Dis. Chest*, **35**, 88.
Senning, Å. (1961). *J. thorac. cardiovasc. Surg.*, **41**, 542.
Sones, F. M., jun., Shirley, E. K., Proudfit, W. L., and Westcott, R. N. (1959). *Proceedings of 32nd Scientific Sessions, American Heart Association*, p. 773.
Vineberg, A. M. (1946). *Canad. med. Ass. J.*, **55**, 117.
Wharton-Jones, T. (1851). *Guy's Hosp. Rep.*, 2nd ser., **7**, 1.

The Chartered Society of Physiotherapy has sponsored a film, "Posture and Movement in Childbirth," which deals, in two parts, with "Posture in Pregnancy" and "Post-natal Rehabilitation." The film is directed by Mrs. M. Williams, M.C.S.P., T.M.A.O.T., in collaboration with the Obstetric Unit of Edgware General and Bushey Maternity Hospitals. It is a 35 mm. colour filmstrip and details may be obtained from the producers, Camera Talks, 23 Denmark Place, London W.C.2.

ABO BLOOD GROUPS IN RELATION TO ISCHAEMIC HEART DISEASE

BY

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The increasing incidence of ischaemic heart disease in certain countries has led many to the view that environmental influences are accelerating its development. Studies on groups have shown that Japanese who have come under the influence of the American mode of life in Hawaii and California have a greater susceptibility than Japanese in Japan (Keys *et al.*, 1958). There are similar group studies on recent immigrants into Israel (Toor *et al.*, 1957), and the differences in mortality dependent upon social class (Registrar-General, 1954) in England and Wales add to this view. Contrariwise, ischaemic heart disease has close associations with certain inherited disorders—for instance, essential xanthomatosis—and in clinical practice one is impressed by the high prevalence of the disease among members of the patient's family. Indeed, this impression is confirmed by the survey of Gertler and White (1954) on the frequency of the disease in parents and siblings of affected individuals. These authors, too, have emphasized the role of constitutional factors in the development of the disease by reporting the increased frequency of the disease in mesomorphic individuals as opposed to ectomorphs. Body build may be influenced by feeding in childhood, and likewise familial relationships could arise as readily from environmental causes. The differentiation of the role of genetic from environmental factors becomes an important issue, as environmental influences may be modified.

The increasing use of the blood-group distribution in the study of diseases has prompted us to use this technique in an attempt to assess the role of genetic factors in the development of ischaemic heart disease.

The Population of the Cape Peninsula

In a previous report (Bronte-Stewart *et al.*, 1955) a description was given of the three main racial groups resident in the Cape Peninsula—namely, Bantu, Cape Coloured, and White (European)—which was felt to be pertinent to their differing prevalence of ischaemic heart disease. In that report the Whites were treated as a homogeneous group, but for the purposes of the present study it became necessary to divide the Whites into English-speaking, Afrikaans-speaking, Jewish, and other groups and define the measure whereby this separation was achieved. There are distinct differences between these groups. Apart from home language, there are differences in outlook, occupation, temperament, dietary customs, and religious faith, but the environmental differences are not as marked nor are they dictated to the same degree by economic factors as between Bantu, Coloured, and White groups.

Four magisterial districts constitute this area, and population estimates are based on the 1951 census. No analysis of census statistics is available on the basis of home language, but this would not effectively divide the English-speaking and Jewish groups. Population according to religious faith is analysed. Professed religious faith also appears among the particulars recorded on the hospital admission of each in-patient or out-patient. Professed religious faith was therefore used as a means of subdivision for both the patients and the controls.

The Afrikaans-speaking group in general attend the Nederduitse Gereformeerde, Hervormde, and Gereformeerde Kerke (Dutch Reformed and related churches), where Afrikaans is the language medium of the service. Most of the English-speaking group belong to the Church of England, Methodist, Presbyterian, and Congregational Churches. The former has its roots in a relatively small stock transplanted from Holland and France during the seventeenth and eighteenth centuries and there has been little admixture since. The latter originated from British immigrants mainly in the nineteenth and twentieth centuries. Nearly all the 20,652 Jews here are Ashkenazi Jews originating from Central and Eastern Europe, the main immigration occurring about the turn of the present century.

While the Jewish group is fairly well defined, separation between English- and Afrikaans-speaking groups is less clear-cut genetically owing to a small degree of intermarriage during the last two generations. Nevertheless, utilizing professed religious faith as the means of subdivision, distinct differences in the ABO blood groups emerge between English- and Afrikaans-speaking groups. The Jewish and Afrikaans- and English-speaking groups constitute just over 93% of the White population in the proportion of 10:25:65 respectively. The remaining Whites belonging to the Roman Catholic, Lutheran, and other churches were treated separately as they were a heterogeneous collection of Irish, Portuguese, Italians, Scandinavians, etc.

Control Series.—As is the custom in blood-group studies (Aird *et al.*, 1960), the control series were formed from new donors in this area subdivided on their professed religious faith. The paucity of Jewish new donors necessitated a separate survey to obtain suitable controls by grouping all children attending a Jewish day-school; school attendances being compulsory. It will be seen from Table I that the blood-group pattern conforms to that expected from the country of origin (Mourant *et al.*, 1958).

TABLE I.—*Control Series Used*

Racial Group	Blood Group				Total
	O	A	B	AB	
English ..	913	857	211	99	2,080
Afrikaans ..	876	714	215	97	1,902
Jewish ..	403	440	185	87	1,115
Other Whites ..	251	212	68	36	567
Coloured ..	276	220	149	57	702

Patients Studied

From the outset it must be emphasized that our patients with ischaemic heart disease were sampled from anticoagulant clinics. In obtaining our sample in this way the material is selected in so far that it represents mainly patients who have survived long enough to receive anticoagulant therapy. It is the accepted policy of physicians practising in this area to prescribe anticoagulant therapy as an integral part of the treatment of ischaemic heart disease, particularly in those who have had myocardial infarction or who have severe angina pectoris. The material was restricted to these two disorders. The study began at Groote Schuur Hospital, the teaching hospital of the University of Capetown. This hospital serves an area wherein the proportion of English-speaking to Afrikaans-speaking to Jewish Whites is 8:2:1. Later, the sample was enlarged by incorporating patients at the Karl Bremer Hospital, the teaching hospital of the University of Stellenbosch, a medical school where Afrikaans is the language of instruction. This hospital serves a predominantly Afrikaans-speaking area with the proportion of English-speaking to Afrikaans-speaking to Jewish being 22:42:1. Finally, the survey was extended to include private patients attending clinical pathologists in private practice. We believe that in this way we have drawn on the vast majority of subjects with ischaemic heart disease in this area.

Criteria for Selection.—With few exceptions all patients had been hospitalized. The records of each patient were carefully scrutinized so far as possible by one observer, to reduce observer error. Strict criteria were followed in the selection, and cases with incomplete records and atypical clinical features were excluded. Patients with coexistent diseases such as aortic-valve disease, diabetes mellitus, polycythaemia, hypothyroidism, etc., were excluded. This reduced the sample to 855 patients, of whom 695 had survived myocardial infarction and 160 had angina pectoris. In 63 of the 695 patients with a history of having had a myocardial infarction no electrocardiographic records at the time of the attack were available. They, too, were excluded from the study, as the clinicopathological study reported by Paton (1957) revealed that the diagnosis of myocardial infarction on clinical grounds alone was grossly inadequate. The remaining 632 patients were further subdivided on the presence or absence of significant Q-wave electrocardiographic patterns according to the criteria laid down by Schrire (1959).

Of the 160 subjects with angina pectoris, 113 had abnormal electrocardiographs (E.C.G.) at rest or on effort. Effort E.C.G.s were not performed in the remaining 47; but they were not excluded, in view of a typical clinical history with central chest pain bearing a relationship to effort and relief by nitrites.

The race, age, sex, and professed religious faith of each patient were recorded for the purpose of subsequent analysis. The final sample consisted of 694 White and 98 Cape Coloured patients, of whom 77% were drawn from the Groote Schuur Hospital, 11% from the Karl Bremer Hospital, and 12% from the clinical pathologists in private practice. Only one Bantu with ischaemic heart disease was encountered. He had diabetes mellitus and was not included in the series.

Blood-grouping.—All the controls and patients sampled were grouped by the same individual using the standard tube technique.

Method of Calculation.—As the study proceeded it became obvious that we were confronted not only with a heterogeneous population but also with possible differences dependent upon the clinical presentation of the disease group. As our sample was too small for the extensive subdivision that the standard statistical procedures required we have followed the advice of Fraser Roberts (1957), using the technique that allows pooling of the data. In this way we have obtained a combined weighted estimate of the strength of the association. Of the two techniques suggested we have followed that of Woolf (1955), as this can be used for combining and comparing populations with widely differing blood-group frequencies. Furthermore, as Fraser Roberts states, this has the advantage that the results, expressed as a relative incidence, come out in a form with a simple and direct physical meaning; it being natural to think of diseases in terms of incidence.

Results

The controls and the basic data are submitted in Tables I and II; the patients being subdivided into race as defined, and into the manner of clinical and electrocardiographic presentation. The combined weighted estimate of the association is seen by comparing all the 792 cases against 6,366 controls in Table III; weighted in the sense that the proportion with which each race is represented in the control series is identical with that in the patient series. This merely serves to indicate that there is a deficiency of group O individuals among the patients with ischaemic heart disease and that the degree of excess of groups A and B is similar. Expressing these deviations in the form of relative incidence shows that these ratios, A:O (1.34) and A+B:O (1.34) and A+B+AB:O (1.31), are essentially similar, too. This observed excess of A and B and a deficiency of O in the patients over that expected from the controls is slightly greater in the females. For example, the relative incidence of A:O for the 617 males is 1.32 (P<0.001) and for the 175 females is 1.40 (0.1>P>0.05), while the female relative incidence of A+B:O is 1.51 (0.02>P>0.01) and the male 1.30 (P<0.001). Subdivision of the sample into decades of age greatly reduced sample size, and for this reason detailed statistical analyses were not undertaken, but at each decade of age the A and B predominance over O is maintained with the exception of the over 70's, comprising only 41 patients, where there is a predominance of group O.

TABLE II.—*Basic Data: Blood Groups of Patients in Series by Manner of Presentation and by Race*

Racial Group		Blood Group O	A	B	AB	Total
English	Myocardial infarction:					
	With Q waves	57	87	24	8	176
	Without Q waves	20	32	10	2	64
	Angina pectoris	17	21	6	3	47
Afrikaans	Myocardial infarction:					
	With Q waves	54	41	10	3	108
	Without Q waves	9	11	2	2	24
	Angina pectoris	23	14	3	4	44
Jewish	Myocardial infarction:					
	With Q waves	27	42	14	8	91
	Without Q waves	11	12	10	0	33
	Angina pectoris	13	9	6	1	29
Other White	Myocardial infarction:					
	With Q waves	14	24	6	2	46
	Without Q waves	5	4	6	0	15
	Angina pectoris	7	8	1	1	17
Coloured	Myocardial infarction:					
	With Q waves	13	31	15	4	63
	Without Q waves	5	6	1	0	12
	Angina pectoris	9	5	6	3	23

TABLE III.—*Percentage Group Frequencies*

Blood Group	Controls*	Total Series (792 Cases)	Controls*	M.I. With Q Waves (484 Cases)	Controls*	M.I. Without Q Waves (148 Cases)	Controls*	Angina Pectoris (160 Cases)
O	42·3	35·8	42·4	34·1	41·9	33·8	42·5	43·1
A	38·5	43·7	38·5	46·5	38·9	43·9	38·1	35·6
B	13·2	15·1	13·1	14·3	13·2	19·6	13·4	13·8
AB	6·0	5·4	6·0	5·2	6·0	2·7	6·0	7·5

* Controls weighted according to the proportion of the different ethnic groups among the patients.
Note similarity between controls and cases with angina pectoris.

Subdivision on Clinical and Electrocardiographic Criteria

The 160 cases with angina pectoris and no past history of myocardial infarction have a blood-group distribution no different from their weighted controls (Table III). This can also be shown, indirectly, by removal of these 160 cases with angina pectoris from the total series, by which means the mean weighted A+B:O relative incidence rises from 1.34 to 1.47 (Table IV). This effect is seen in each race, although in the English and Other White groups there remains a preponderance of A and B over O individuals, but in neither instance is this statistically significant. The mean weighted relative incidence A+B:O for these 160 cases is 0.95. Among these are 113 cases with electrocardiographic abnormalities, but their A+B:O relative incidence is similar, 0.94.

There appears to be little if any gain by subdividing the remaining 632 cases with myocardial infarction on the basis of presence or absence of Q waves on the electrocardiograph. An indication of this can be seen in Table III, where the deficiency with which group O individuals are represented in these two groups of patients is almost identical when compared with their

TABLE IV.—*Incidence in Groups A and B Relative to Incidence in Group O in Relation to Ethnic Grouping and Manner of Presentation*

Racial Group	All Patients (792) No.	Relative Incidence A+B : O	χ^2	All Myocardial Infarctions (632) No.	Relative Incidence A+B : O	χ^2	Myocardial Infarction with Q Waves Only (484) No.	Relative Incidence A+B : O	χ^2
English	287	1·64	13·25	240	1·70	13·14	176	1·67	9·10
Afrikaans	176	0·89	0·54	132	0·96	0·06	108	0·89	0·33
Jewish	153	1·18	0·76	124	1·32	1·73	91	1·33	1·34
Other White	78	1·69	4·14	61	1·89	4·76	46	1·92	3·82
Coloured	98	1·77	5·59	75	2·20	7·74	63	2·64	9·04
Mean weighted relative incidence		1·34			1·47			1·44	
χ^2 Total			24·28			27·43			23·63
χ^2 Diff. from unity. D. of F.=1			13·16			17·92			12·79
χ^2 Heterogeneity. D. of F.=4			11·12			9·51			10·84
P Heterogeneity			0·05>P>0·02			0·05>P>0·02			0·05>P>0·02

Note that relative incidence rises on removal of patients with angina pectoris from series.

controls. There are differences in groups A and B, but as the myocardial infarction series without Q waves constitutes only 148 cases the significance of this is doubtful. Certainly in Table IV there are only minor differences between the A+B:O relative incidence in the 632 cases of myocardial infarction with and without Q waves, when compared with the 484 cases of myocardial infarction with Q waves only. In these cases the ABO distribution was the same whether the infarction was anterior or posterior.

Subdivision on Race and Religious Faith

Major differences do occur when the cases are analysed according to race, with the Afrikaans-speaking White South African and the Jewish cases showing no significant deviations from their controls. In all the other races, however, the A+B:O relative incidence is high (Table IV). These ethnic differences were noticable even in the pilot study, and increasing the sample size has not altered this trend. It is unlikely that these ethnic differences represent fluctuations occurring merely by chance. Chi-square analysis for heterogeneity (Table IV) makes this unlikely. In addition, analysis of the Groote Schuur Hospital sample, the Karl Bremer Hospital sample, or even the private patients separately, on the basis of religious faith reveals the same trends. Because a difference has been shown between whether the patient presents as a case of myocardial infarction as opposed to angina pectoris, Table IV contains as well the racial distribution of cases with myocardial infarction only. The similarity of trends does not support an explanation of the racial differences as being due to an undue weighting with cases of myocardial infarction in one group or angina pectoris in another.

On the other hand, there appears to be an inverse or negative correlation between the predominance of A and B over O and the prevalence of the disease in the different racial groups (Table V). These estimates of prevalence can only be regarded as rough, as the latest census figures are not to hand, and for this reason also they have not been age-standardized. There has been a

TABLE V.—*Association Between Myocardial Infarction and ABO Blood Groups in Relation to Prevalence*

Racial Group	Myocardial Infarctions	
	Prevalence per 1,000	Relative Incidence A+B : O
Jewish	21·1	1·32
Afrikaans	11·4	0·96
English	5·6	1·70 ($P<0·001$)
Other White	4·4	1·89 ($0·05>P>0·02$)
Coloured	0·5	2·20 ($0·01>P>0·001$)

recent influx of young Afrikaans-speaking South Africans into the cities from the country areas, so that possibly the prevalence as shown for the Afrikaans-speaking South African is on the low side.

The remaining cases attending the anticoagulant clinic consist of cases of rheumatic heart disease, polycythaemia, cerebrovascular disease, peripheral vascular disease, thrombophlebitis, and others. With the exception of the patients with peripheral vascular disease who show an excess of A, the blood-group distribution is not different from that of the controls. The 74 patients with diabetes mellitus and ischaemic heart disease who were excluded from the series showed an A:O relative incidence of 1.5 and an A+B:O relative incidence of 1.4, neither of which was significant on chi-square analysis.

Discussion

These data suggest that a highly significant association between the ABO blood groups and ischaemic heart disease exists. The probability of this being a chance association is less than 1 in 1,000. Expressing the findings in the form of relative incidence of one or more groups to another would allow one to state that in certain racial groups the risk of developing myocardial infarction in group A and B individuals is more than one and a half times that in group O individuals. On the other hand, as the study was confined mainly to survivors of myocardial infarction, the findings could have the opposite interpretation in that group A and B individuals are more likely to survive than group O. As was the case in the association between serum cholesterol levels and ischaemic heart disease, this argument cannot be refuted until a large prospective study is undertaken.

What seemed at the outset to be a simple problem of assessing the ABO blood-group pattern in ischaemic heart disease turned out to be utterly complex. In part this has been due to the fact that we could not regard our White group as homogeneous. Secondly, there appeared to be differences dependent upon whether the presenting feature was myocardial infarction or angina pectoris. Thirdly, we have rigidly excluded all cases without unequivocal evidence of ischaemic heart disease and those with any other coexistent diseases. This has resulted in what must be regarded as a small series in relation to most studies on blood groups and disease, but we have attempted to circumvent analyses on small sample sizes where possible by pooling and following the technique of Woolf (1955).

Despite the small sample size, we have been prompted to report these findings at this stage for the following reasons. Having exhausted the main supply in this area, an increase in sample size by any appreciable amount would take many more years. The consistency of the results throughout has impressed us. For example, the initial pilot study on only 100 cases revealed an A:O relative incidence of 1.39 ($P<0.001$). Additions since our preliminary report 18 months ago on the first 600 cases have not altered the findings then reported (Bronte-Stewart *et al.*, 1960). Furthermore, as emphasized in the report on carcinoma of the oesophagus (Aird *et al.*, 1960), small samples do have value. In certain instances it has been through the combination of data from various areas that really significant associations have emerged (Fraser Roberts, 1957).

The strength of the association depends in large measure on the validity of our controls. We have carefully considered this and have been reassured by the close resemblance between our control figures and those reported from the respective countries of origin of the ethnic groups concerned. For example, the slight excess of O over A in our English-speaking controls is very similar to that seen in the control figures used by Aird *et al.* (1960), whereas the Jewish pattern resembles that reported on Ashkenazi Jews in Israel and Lithuania (Mourant *et al.*, 1958). In our Afrikaans-speaking group the greater excess of O over A is not unlike the Zeeland pattern in the Netherlands (Mourant *et al.*, 1958).

In the analysis two points emerged that initially caused concern. These were the differences dependent, firstly, upon race or religious faith, and, secondly, on the manner in which ischaemic heart disease presented—namely, myocardial infarction or angina pectoris. Even though this sample of 792 cases is small for subdivision in this manner, the chi-square analysis reveals that the probability of the racial differences being due to sampling variation is less than 5%. Obviously it is desirable to have the experience from other areas before these associations can be fully assessed.

The explanation for the difference in the ABO blood-group association with myocardial infarction on the one hand and angina pectoris on the other cannot be due to the differing proportion with which the races are represented in these categories because the controls are weighted to allow for this. The angina pectoris sample is only 160 cases, however, which is too small for extensive subdivision. Perhaps on this score too much weight should not be attached to the differences we have found between these cases and the cases of myocardial infarction. Mention is made of it in view of the distinct differences in the serum lipids between cases of angina pectoris and myocardial infarction reported by Lawry *et al.* (1957) and by Acheson (1961), while Brown *et al.* (1958) report different relationships to social class in patients with myocardial infarction compared with those with other manifestations of coronary disease. All these data could add to the view of Acheson (1961) that fundamental differences may underlie the pathogenesis of these two conditions.

The concept of transient and balanced polymorphism (Ford, 1956) could be the explanation for the racial differences. Instead we believe that the inverse relationship to the prevalence of the disease might illustrate the interplay of genetic and environmental factors in the development of ischaemic heart disease. In our area, for example, there are marked differences between coloured and white groups with respect to the dietary fat intake (Bronte-Stewart *et al.*, 1955), and a recent dietary survey amongst the Whites revealed that the customary dietary fat intake of the Afrikaans-speaking groups was as much as one and a half times as great as that of a sample from the English-speaking community (Perrin and Bronte-Stewart, unpublished observations). Could these data mean that in areas or groups where a high degree of prevalence exists environmental factors may be so great as to overshadow genetic influences? On this hypothesis, data from other areas on blood-group associations in ischaemic heart disease, should these be forthcoming, would have to be assessed in relation to the degree of prevalence.

The only other studies on the ABO blood-group distribution in ischaemic heart disease of which we are aware are on small series. Speedby (1959) mentions that in a series of 140 cases he found almost twice as many group A patients among cases with anterior infarction as among cases with posterior infarction—a finding that we could not confirm. Further details are not given. Gertler and White (1954) studied 81 young patients with myocardial infarction: 37 were group A, 14 group B, 4 were group AB, and only 26 group O. This would give the relative incidence of A+B:O as 1.88 ($0.05>P>0.02$) when compared with their 145 controls. Pell and D'Alonzo (1961) likewise show a deviation in the same direction as our sample with an excess of A and B over O among 226 cases of myocardial infarction on comparison with 245 controls, but they regarded the differences as being well within the limits of sampling error.

Summary

In an attempt to obtain data on the interplay of genetic and environmental factors in the development of ischaemic heart disease, blood grouping was carried out on 792 patients attending prothrombin clinics throughout the Cape Peninsula.

There was an excess proportion of groups A and B and a deficiency of O in the patients when compared with controls.

The extent of these deviations was dependent upon the presence or absence of previous myocardial infarction and on the racial origin of the patient.

In those races where the prevalence of ischaemic heart disease was high there was no significant deviation, and this was felt to exemplify the complex interplay of genetic and environmental factors in the development of ischaemic heart disease.

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REFERENCES

Acheson, R. M. (1961). *Lancet*, **1**, 615.
Aird, I., Lee, D. R., and Roberts J. A. F. (1960). *Brit. med. J.*, **1**, 1163.
Bronte-Stewart, B., Botha, M. C., Fewster, M., Krut, L., Bouchier, I., Wells, V., and Wilkens, J. (1960). *S. Afr. med. J.*, **34**, 675.
—— Keys, A., and Brock, J. F. (1955). *Lancet*, **2**, 1103.
Brown, R. G., McKeown, T., and Whitfield, A. G. W. (1958). *Brit. med. J.*, **1**, 555.
Ford, E. B. (1956). *Genetics for Medical Students*, 4th ed., p. 102. Methuen, London.
Gertler, M. M., and White, P. D. (1954). *Coronary Heart Disease in Young Adults: A Multi-Disciplinary Study*, pp. 26 and 19. Harvard Univ. Press, Cambridge, Mass.
Keys, A., Kimura, N., Kusukawa, A., Bronte-Stewart, B., Larsen, N., and Keys, M. H. (1958). *Ann. intern. Med.*, **48**, 83.
Lawry, E. Y., Mann, G. V., Peterson, A., Wysocki, A. P., O'Connell, R., and Stare, F. J. (1957). *Amer. J. Med.*, **22**, 605.
Mourant, A. E., Kopec, A. C., and Domaniewska-Sobczak, K. (1958). *The ABO Blood Groups*, pp. 13 and 64. Blackwell, Oxford.
Paton, B. C. (1957). *Amer. J. Med.*, **23**, 761.
Pell, S., and D'Alonzo, C. A. (1961). *J. Amer. med. Ass.*, **175**, 463.
Registrar-General (1954). *Decennial Supplement, England and Wales, 1951. Occupational Mortality*, pt. 1, p. 13. H.M.S.O., London.
Roberts, J. A. F. (1957). *Brit. J. prev. soc. Med.*, **11**, 107.
Schrire, V. (1959). *Postgrad. med. J.*, **35**, 218.
Speedby, H. J. (1959). *Brit. med. J.*, **1**, 1042.
Toor, M., Katchalsky, A., Agmon, J., and Allalouf, D. (1957). *Lancet*, **1**, 1270.
Woolf, B. (1955). *Ann. hum. Genet.*, **19**, 251.

Work has started in Port Moresby on a training college for student nurses as the first step towards the founding of a medical school in the Territory of Papua and New Guinea. The college will house about 300 students and cost about £A.350,000. Most of the trainee nurses will come straight from native villages at the age of 15 or 16.