With regard to treatment, at Nauru and other leper stations in the Pacific intravenous medication was thoroughly carried out and all were agreed that it was the only satisfactory method. Both "Moogrol" and chaulmoogra oil were used with gratifying results. The vexed question of segregation was exercising the minds of all interested in the disease and many elaborate experiments on a large scale were being carried out at such places as Cullion in the Philippines, Molokai in Hawaii and Nakogai in Fiji. These combined segregation under ideal conditions (natural living conditions, few restrictions, normal activities of the patients, respect for social amenities) with rigorous intravenous medication giving the unfortunates hope of eventual cure. It must be remembered that such conditions were framed for very susceptible people in a tropical climate and that it would not be known for many years whether such methods would be efficient or whether they would be worth the enormous expense. In Nauru a similar method was being adopted with monthly inspections of the non-leprous part of the community in order to discover fresh cases.

The conditions in Australia were entirely different and Dr. Molesworth had, he thought, proved his case that in Australia segregation of such a lowly infective disease was unnecessary, ineffectual and, when the apathy with regard to such scourges as tuberculosis and venereal disease was considered, almost farcical.

Dr. Dew then moved:

> That the Commonwealth Government should take into consideration the desirability of dealing with leprosy as a national problem and that in doing so the necessity for humane methods be recognized.

The motion was seconded by Dr. Herman Lawrence who supported Dr. Molesworth in his advocacy of the discontinuance of the present system of internment, applied as it was to many lepers who could not be considered infective. It was carried.

DR. H. DOUGLAS STEPHENS, the President, said that he thought the occasion was such that the Branch might depart from its usual practice of not passing votes of thanks. Dr. Molesworth's paper had been extremely interesting and valuable and Dr. Tebbutt had demonstrated an excellent series of photomicrographs which constituted a most important contribution to the study of the pathology of leprosy. He had much pleasure in moving that the visitors be accorded a hearty vote of thanks and expressed hope that the interchange of ideas between the Branches in neighbouring States would become a regular practice.

The motion was seconded by Dr. J. Newman Morris, Vice-President, and carried with acclamation.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the Victorian Branch of the British Medical Association:

Mulcahy, James Edward, M.B., B.S., 1924 (Univ. Melbourne), Caulfield.

Best, John Cleveland, M.B., B.S., 1925 (Univ. Melbourne), St. Kilda Road, Melbourne.

Coffey, Frank Frederic, M.B., B.S., 1926 (Univ. Melbourne), 105, Stanhope Street, Malvern.

McCumisky, Philip Bernard, M.B., B.S., 1923 (Univ. Melbourne), Minyip.

Maling, Henry Coverley, M.B., B.S., 1925 (Univ. Melbourne), Ivanhoe.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants *locum tenentes* sought, etc., see "Advertiser," page xx.

ADELAIDE HOSPITAL: Medical Registrar.

QUEEN VICTORIA HOSPITAL, LAUNCESTON, TASMANIA: Resident Lady Superintendent.

ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, SYDNEY: Honorary Assistant Urologist.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association.
QUEENSLAND: Honorary Secretary B.M.A. Building, Adelaide Street, Brisbane.	Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Honorary Secretary, 12, North Terrace, Adelaide.	Contract Practice Appointments at Ceduna, Wudinna (Central Eyre's Peninsula), Murat Bay and other West Coast of South Australia Districts.
WESTERN AUSTRALIAN: Honorary Secretary, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Diary for the Month.

SEPT. 20.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPT. 21.—Tasmanian Branch, B.M.A.: Council.
SEPT. 21.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 22.—Victorian Branch, B.M.A.: Council.
SEPT. 22.—Western Medical Association, New South Wales (Annual).
SEPT. 24.—Queensland Branch, B.M.A.: Council.
SEPT. 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 28.—Illawarra Suburbs Medical Association, New South Wales.
SEPT. 30.—New South Wales Branch, B.M.A.: Branch: Election of two members of Federal Committee.
SEPT. 30.—South Australian Branch, B.M.A.: Branch.
OCT. 1.—Queensland Branch, B.M.A.: Branch.
OCT. 1.—New South Wales Branch, B.M.A.: Delegates of Local Associations meet Council (First Day).
OCT. 2.—New South Wales Branch, B.M.A.: Delegates of Local Associations meet Council (Second Day).
OCT. 5.—Tasmanian Branch, B.M.A.: Council.

Editorial Notices.

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THE MEDICAL JOURNAL OF AUSTRALIA

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BLOOD GROUPS OF NORTH QUEENSLAND ABORIGINES, WITH A STATISTICAL COLLECTION OF SOME PUBLISHED FIGURES FOR VARIOUS RACES.

By DOUGLAS H. K. LEE, B.Sc. (Queensland),

From the Australian Institute of Tropical Medicine, Townsville.

AT the suggestion of Dr. Heydon I undertook the examination of a number of full blooded aborigines at the Palm Island Settlement. Herein are published the results of this examination, a contribution to the pioneer work of Tebbutt and McConnel[8] [9] and also a collection of as many results obtained in various parts of the world as could be found in the literature available.

Technique.

The Palm Island Settlement lies about forty miles north-north-east from Townsville, which fact together with the irregularity of the steamer service increased somewhat the complexity of the technique. The open-slide method of Vincent[1] as modified by Heydon and Murphy[17] for work in the tropics was employed. The blood samples were collected by thumb puncture into half inch test tubes four-fifths full of 3·8% sodium citrate solution and allowed to settle for a period of about four hours. If the sample was a poor one, centrifuging by hand was employed. The supernatant fluid was then decanted, the corpuscles resuspended in 0·85% saline solution (test tube four-fifths full) by inversion and the mixture again allowed to settle for about three hours, when the supernatant fluid was decanted. A drop of the sediment was mixed with a drop of each of the test sera (Group II. and Group III.) at opposite ends of the slide, the slide placed on two tooth picks or matches on a moist filter paper and covered with half a Petri dish. The drop was well mixed by movements of the slide after five minutes and again after another five. The result was then read.

Sera for testing were taken from known Group II. and Group III. people. For a number of cases my own (Group II.) serum was employed, because

the regularity of its reaction was known, and the risk thereby obviated of selecting a donor whose serum did not possess the agglutinin β to the full or any degree.[15] [16] This precaution is advisable in all routine examinations. When different donors were used whose corpuscle reactions alone were known, the sera were often controlled by employing corpuscles of known reaction, though this was not always carried out. When such untried donors are used, it is advisable to collect blood from more than one donor of each group in case one should prove irregular.

Diagnosis.

Typical reactions are readily recognized with the unaided eye and in such cases the microscope was not used as part of the routine, though any doubtful appearances were always examined microscopically. The moist chamber method employed removes the difficulty experienced by Bais and Verhoef.[14] Many of these appearances were found to be due to rouleau formation, but this was easily detectable. More puzzling is an appearance noted quite well in one instance and possibly though doubtfully in more. This consisted of a number of small clumps of corpuscles, half a dozen or so in each, revealed by the microscope in a drop which appeared macroscopically to be free from agglutination. Of ninety specimens that yielded no reaction, and which were examined microscopically, only one gave a definite appearance of this nature.

Lattes and Cavazzuti[15] give as the criteria for "pseudo-agglutination" as distinguished from true agglutination: lack of specificity, absence of fixation of the agglutinin, inhibition by lecithin and low titre. The macroscopic appearances are various: "Commonly they were mediocre and slow; in some were seen, in addition, among the irregular masses, distinct rouleau formations. But it must be admitted that in certain cases intense and rapid agglomerations were seen, perfectly analogous in their appearance to a strong and authentic iso-agglutination."[15] They offer no explanation of the phenomena, biological or otherwise (". . . *la vraie nature ne peut être mise en évidence que par des recherches soigneuses et complexes*"), so that the position of these reactions, defined only by negative characters is somewhat vague. Is this appearance I am describing caused by pseudo-agglutination or can it be explained by supposing that the corpuscles contained the agglutinogen *A* in very small amounts or were only slightly susceptible to the action of the agglutinin α?

The classification of the blood of one other subject was somewhat obscure. This was a girl of five whose corpuscles agglutinated strongly with Group II. serum, weakly with Group III. serum. Retested against two other samples of Group II. serum and one other of Group III. serum they agglutinated well with the former and not at all with the latter (the diagnosis was confirmed by the microscope). May this phenomenon be explained according to the findings of Lattes and Cavazzuti, by supposing that the corpuscles contained in very weak amount the agglutinogen *A*, so weak that it could be detected only by a serum with a high concentration of α agglutinin (the second Group III. serum proved to be typical when tested with other corpuscles whose grouping was known)?

It may be well worth while introducing the use of lecithin into routine examinations, if its differential action on pseudo- and true agglutination is what Lattes[15] claims for it.

I should like to record also, whilst speaking of the work of Lattes and Cavazzuti, that four out of the six samples of Group IV. corpuscles found in this series of examinations induced noticeably less agglutination with Group II. serum than with Group III. serum, the agglutination with Group II. serum being less also than that of Group III. corpuscles with the same Group II. serum. This is a further indication of a variation in titre of agglutinogens in the corpuscles.

Stability of Agglutinogens and Agglutinins.

In the examination of the aborigines the corpuscle samples were always examined the same day to avoid the effects of bacterial contamination. I have since found stated in the literature that corpuscles may be desiccated[8] or even heated to 100° C.[15] without diminution of the iso-agglutinogen content. An effect of bacterial contamination is hæmolysis of the corpuscles, but it seems very probable that sufficient asepsis or antisepsis could be obtained in the light of the stability of the agglutinogen to secure the preservation of corpuscle samples over a sufficient period to render very simple the routine examination at institutions situated similarly to Palm Island.

Again in this examination I endeavoured to use sera as fresh as possible. At the commencement of the series only were some samples of serum more than thirty-six hours old. The serum used at the commencement was prepared in Townsville on February 3, placed in sterile tubes and sealed, opened when required and used on the fourth and fifth. Compared on the seventh with serum then only thirty hours old, they showed in every case (nineteen samples were tested with both sets) exactly the same reaction. On another occasion serum thirty hours old was compared with serum prepared the same day in its reaction on twenty specimens shown to belong to Groups II., III. and IV. by the fresh serum. Again no qualitative discrepancy in the result was found. On a third occasion eight samples diagnosed as belonging to Group I. by serum thirty hours old were retested by fresh serum; no trace of agglutination was seen even under the microscope. In some cases, it must be admitted, it seemed that the older serum required longer to produce the same degree of agglutination, but the total result was about the same.

An examination of literature yields several references to the stability of the agglutinins. Sanford[2] says: "It is well known that the iso-agglutinins in human serum are thermo-stable" and records his

TABLE I.—FREQUENCY OF VARIOUS GROUPS (CLASSIFICATION OF JANSKY).

Author.	Group I. Agglutinogen *O*		Group II. Agglutinogen *A*		Group III. Agglutinogen *B*		Group IV. Agglutinogen *AB*		All Groups	Biochemical Index $\frac{AB+A}{AB+B}$
	No.	%	No.	%	No.	%	No.	%		
Tebbutt and McConnel ..	105	55·0	73	38·2	11	5·8	2	1·1	191	5·7
Lee	227	60·3	120	31·7	24	6·4	6	1·6	377	4·2
All Authors ..	332	58·5	193	33·9	35	6·2	8	1·4	568	4·67

"Biochemical" Index.—The first name assigned to this index was apparently that of the Hirschfeld[8]: "Biochemical Race Index." This has become shortened to "Biochemical Index" or simply to "Race Index" (Kossovitch,[18] *"Indice des Races"*). Tebbutt[9] suggests the much more appropriate name of "Isoagglutinin Index."

experiments to show that they resist also desiccation. Bais and Verhoef, working in the tropical climate of Sumatra and Java, state[14]: "The test sera . . . were . . . replaced by a fresh stock each week," though they do not say whether they were kept in an ice box or had some preservative, such as tricresol added. Vincent[1] used tricresol 0·25% "because it is desirable to keep the serum sterile," though he records the observation that there was "no apparent diminution in the activity of serum one year old which showed a moderate degree of contamination."

Results.

Three hundred and seventy-seven aborigines in all were examined, ten at the Townsville General Hospital, having come there from the Palms and the remainder at the Palms. Those examined at the Settlement were sent up by Superintendent as being full blooded aborigines and any of those who showed "frizzy" hair or absence of the flattened nose or in general appearance gave the impression of not being full blooded aborigines, were not examined. The names of those who on examination proved to belong to Groups II., III. or IV. were read over to one of the staff who stated them to be all full blooded aborigines to the best of his knowledge. Beyond the limits of these precautions I cannot vouch for the purity of the subjects, since records when available are always to be taken with reservation in cases like this, where parentage is often a doubtful question.

The results of examination are given in Table I., in which appear also the results previously obtained by Tebbutt and McConnel.[8] [9]

The results obtained in the two sets of investigations agree in placing the Australian aboriginal very high up in the series of racial "biochemical" indices, though they differ considerably in the actual magnitude of the index. When the indices for various races are considered, it will be seen that in the list there given only one race lies above the position assigned by Tebbutt and McConnel's results, the North American Indian, who has an index of 9·2. Between 5·7 and 4·2 lie three European races, the Angles (5·35), the Alpine race (4·95) and the English (4·55); all the other races so far investigated (as far as I could find in the available literature) lie below 4·2, reaching as low as 0·5.

As far as Tebbutt and McConnel's results for aborigines are concerned there can be no reasonable doubt as to their authenticity, because all their findings were confirmed by cross-agglutination experiments.[16] My examination on the other hand, was made by the straight-out method of Moss which is open to two objections: (i.) That there is no check on the actual recording of the results, (ii.) that errors in technique interfering with the power of the serum to react are less easily noticed. The major errors affecting a whole batch are easily noted. With regard to the first, all due care was exercised and the slides checked over against the entries on the record sheets before their removal. With regard to the second also, the greatest care was exercised and a regular methodical routine used throughout. As some proof of the reliability of the results the retests may be cited. Thirty-seven out of forty-eight subjects whose blood was found to belong to Group I. on two days, were re-examined (the remaining eleven being unavailable) and all the samples yielded exactly the same results. Of the thirty-nine comparative tests (see above) made on the same samples with two sets of serum, not one showed any discrepancy. With careful working the number of such errors must be small and if they do occur, they will follow the laws of chance and should not affect the index, which is a ratio between two frequencies determined at the same examination.

It seems that the difference between the results may be referred to the geographical positions of the subjects examined on the two occasions. Quite

TABLE II.

Germans.		Japanese.		Koreans.		Chinese.		Russians.	
Heidelberg	3·12	South	1·82	North	1·05	Central	1·42	Central	1·5
Baltic	3·02	Middle	1·68	Middle	1·26	South	1·08	Siberia	1·26
Leipzig	2·04	North	1·58			Pekin	0·50	Perm	1·07
Berlin	2·0							Ukraine	1·05
Munich	2·0								

frequently within the same race (this will be referred to again later) I find that the index varies with the geographical position of the people examined (by "position" is meant their place of birth; migrants, of course, cannot be expected to show an association of grouping with place of residence), as Table II. will show.

It may be that such is the case with the Australian aboriginal. Tebbutt and McConnel give the origin of those of their subjects for whom records were available[9]: Cape York 39 (26%), north Queensland 21 (14%), central Queensland 36 (24%), southern Queensland 53 (36%). Thus well over half (including the nineteen from New South Wales) came from well south of Townsville. An analysis of the origins of those examined by me, on the other hand, yields the following figures:

Total number examined: 377.

Total number for whom records were available: 358.

TABLE III.—DISSECTION OF RECORDS.

Peninsula (south to Cairns and district inclusive)	163
Gulf	55
North-west Queensland	28
Coast (south of Cairns to Townsville inclusive)	71
Coast (south of Townsville to Mackay inclusive)	24
Northern (exact location unknown)	17
Total	358

These records were furnished by courtesy of Mr. Hoffmann, of the Settlement.

I may say, therefore, that the series of aborigines examined were wholly from north Queensland.

Hence I may justly conclude that I have here a further indication of variation of index within a race, with the geographical origin of the group examined. I shall see later that this is only a continuation of a general variation of index with geographical position. I may notice here the recent publication of results obtained by Cleland[20] on South Australian aborigines. Out of one hundred and one examined forty-six belonged to Group I. and fifty-five to Group II. He found none belonging to Group III. or IV. This is the extreme end of the gradient that I suggest exists.

Variation on Successive Days.

The figures given in Table IV. showing the variation in results from day to day during the

TABLE IV.

Date.	Percentage.				Biochemical Index.	
	I.	II.	III.	IV.	For Day.	For Total to Date.
Jan. 28	50	40	10	0	4·0	4·0
Feb. 4	66	33	0	0	—	9·0
Feb. 5	56	33	9	0	2·8	3·8
Feb. 6	70	22	6	4	2·6	3·3
Feb. 7	70	20	5	0	4·0	3·3
Feb. 8	52	38	6	2	5·0	3·75
Feb. 12	57	39	4	0	9·0	4·3
Feb. 13	68	24	4	2	4·3	4·3
Feb. 14	52	40	8	4	3·7	4·25
Feb. 15	58	36	6	2	4·75	4·3
Feb. 16	48	20	8	0	2·5	4·2

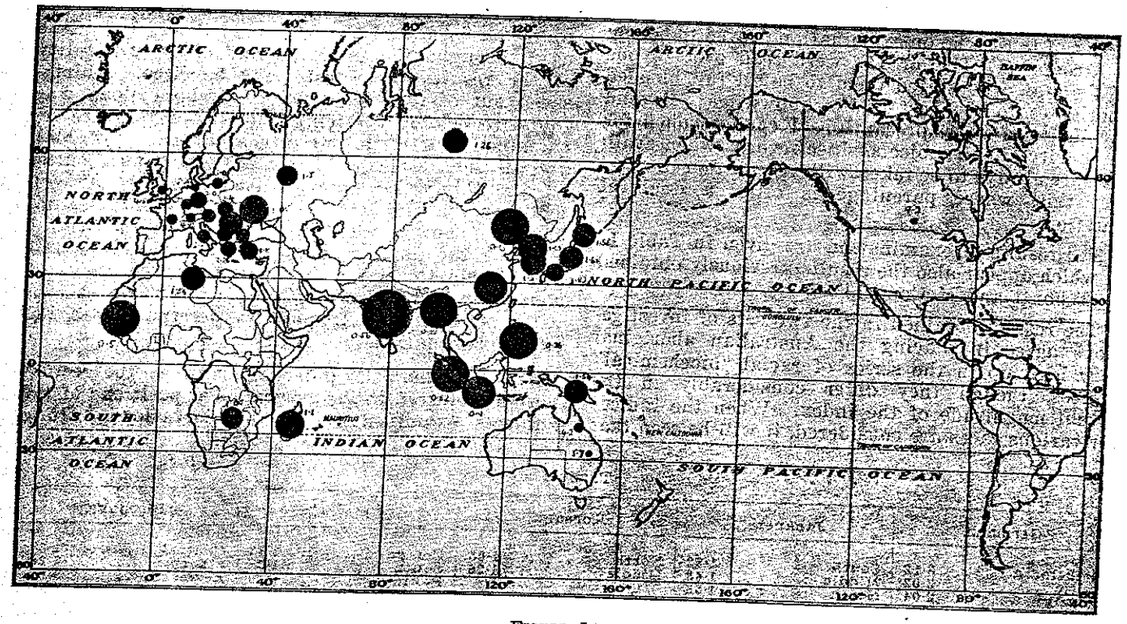

FIGURE I.

examination may serve a double purpose; firstly to show the unreliability of small numbers and secondly to give additional support to the authenticity of the index, in that it remains stable over the last five days of the examination, when the numbers become appreciable.

Comparison Between Calculated and Observed Frequency of Groups I. and IV.

In their article in *The Lancet* the two Doctors Hirschfeld give a method of calculating from the observed frequencies of Groups II., III. and IV. the probable frequencies of Groups I. and IV. (*O* and *AB* respectively). Unfortunately, the method they set out for calculating that of Group IV. is incorrect and this error has been perpetuated by those few workers who have endeavoured to calculate the probable frequencies.[1] The error is best illustrated by setting out the correct method and then pointing out where that used by Doctors Hirschfeld is incorrect.

[1] In his paper read to the Pan-Pacific Science Congress Tebbutt included a calculation of this nature which was made by the correct method. The publication[9] quoted in this article is a summary of the proceedings.

I shall take for convenience the same figures (English) that Doctors Hirschfeld employed in their considerations. It is necessary to note that the classification used by Doctors Hirschfeld is Moss's, while that used here, in common with American serologists, is Jansky's. According to the Jansky classification the figures are:

TABLE V.

I.	II.	III.	IV.
(*O*)	(*A*)	(*B*)	(*AB*)
46·4	43·4	7·2	3·0

The *A* factor occurs in 43·4 + 3·0 = 46·4% of the population. That is out of any group selected at random (as far as the possession of *A* is concerned) out of this population, 46·4% may be expected to contain the *A* factor in their corpuscles.

Similarly, the *B* factor occurs in 7·2 + 3·0 = 10·2% of the population.

Now, if the possession of *A* factor and the possession of *B* factor are absolutely independent of each other, it may be expected that 10·2% of those who

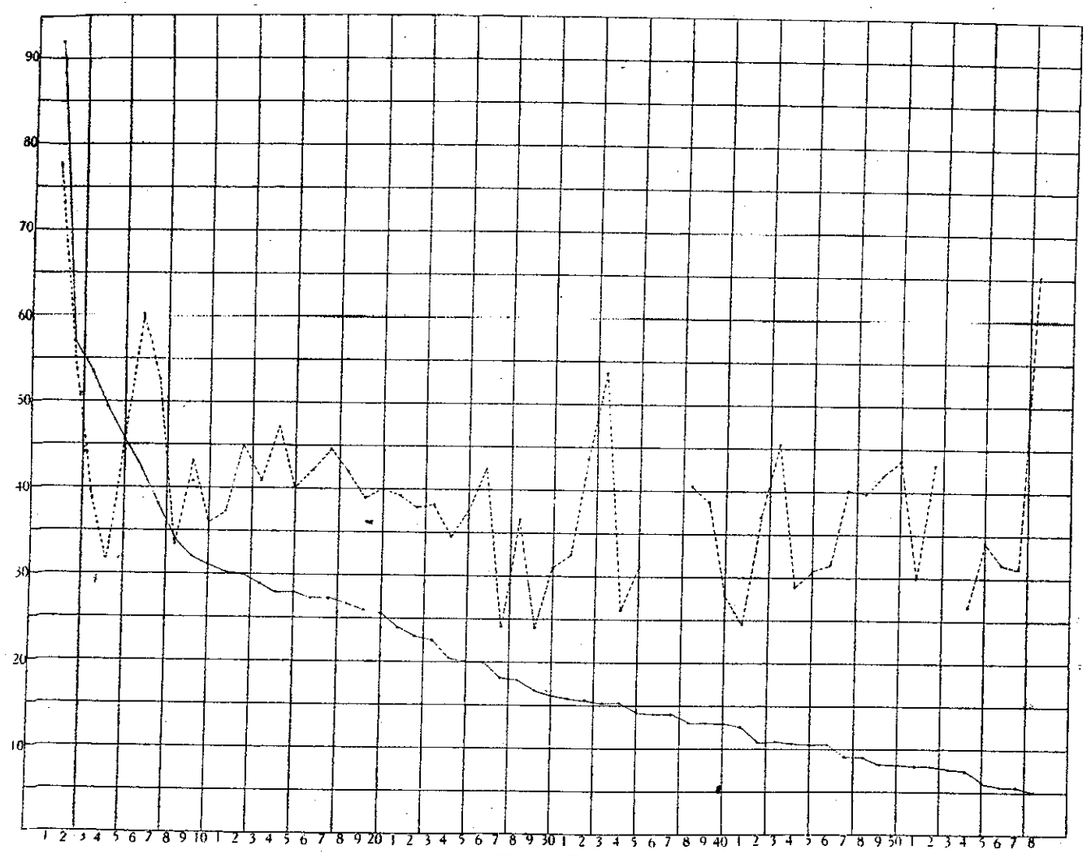

FIGURE II.

contain *A* factor, contain also the *B* factor, because this group of people, although selected to contain *A*, have been selected quite at random as far as the possession of *B* is concerned. That is to say, that of the 46·4% of people containing *A* factor, I should expect 10·2% to harbour *A* and *B* together, that is, to belong to Group IV.

Therefore, the probable frequency of Group IV. in this population is $\frac{46{\cdot}4 \times 10{\cdot}2}{100} = 4{\cdot}7\%$.

By a similar process the probable frequency of Group I. may be calculated in the following manner:

The *A* factor occurs in 46·4%; therefore the non-*A* factor occurs in 100 – 46·4 = 53·6%.

Similarly the non-*B* factor occurs in 100 – 10·2 = 89·8%; therefore the probable frequency of non-*A* and non-*B* together (that is Group I.) is $\frac{53{\cdot}6 \times 89{\cdot}8}{100}$ = 48·1%.

I may now point out the error in the Hirschfeld method for the first part of this calculation. Instead of multiplying the *A* factor frequency by the *B* factor frequency and dividing by 100, they multiplied Group *A* (Group II.) frequency by Group *B* (Group III.) frequency and divided by 100. Now clearly this cannot be allowed for two reasons. (1) Group *A* does not represent the total frequency of the *A* factor, it is less than that frequency; the same obtains in regard to the *B* factor. (2) Group *A* is specially selected as regards the *B* factor. It is selected as not possessing the *B* factor, a negative relation, still a relation. What the method attempts to do, in other words, is to calculate how many Group II. people will also belong to Group III., when all the time the two groups are mutually exclusive. The error is nothing more than one in fundamental deductive logic, though that logic happens to be applied to mathematical considerations. I do not doubt that the Hirschfelds would have realized this mistake if only they had used the term *A* factor to distinguish the total frequency of the agglutinogen *A* from the frequency of people who possessed the agglutinogen *A* in isolation (Group II.); and similarly for *B*.

Strangely enough, they rectified their mistake when calculating for Group I., their method being that outlined above. It is a great pity that this error has been allowed to pass for so long.

Geographical Distribution of the Biochemical Index.

In going through the available literature on the question of the distribution of biochemical index

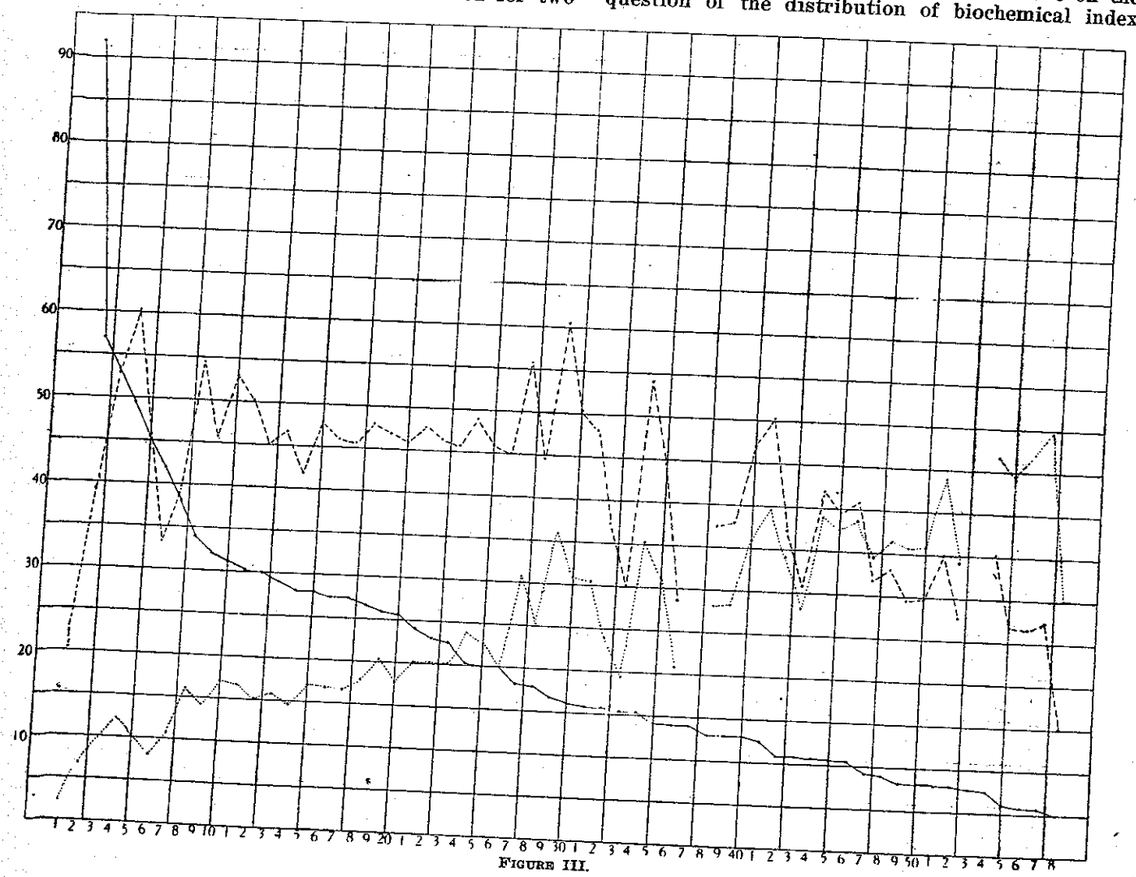

FIGURE III.

through the races of the world I found numerous references to the outstanding feature that from Western Europe eastwards to India there is a steady falling off in the index, followed by a tendency to rise again in Indo-China. But I found only one attempt at a statistical collection of the published figures, namely that given by Steffan.[18] This list has some important omissions, Australian aborigines, Melanesians, Bantus and certain other figures have since been published. This collection, moreover, contains some inaccuracies. I have, therefore, thought it wise to make as complete a collection as possible before trying to follow the geographical distribution of the index. The figures given in Table VI. have been culled from the available literature and in submitting them I wish to state that I do not claim for it completeness to date, since both time and the literature available have been necessarily limited. I invite all interested to criticize, correct and rectify omissions as soon as possible. The figures which are especially wanting, are those for South American natives, Maoris, Fijians, South African bushmen, Norwegians, Swedes and Eskimos. In several places I have found inaccuracies both in quotations and in calculation and these I have endeavoured to rectify. The calculations for probable frequencies of Group IV. have been made according to the method given above and disagree, therefore, from previously published figures. Discussion is especially invited on the question, as to whether the differences between observed and calculated frequencies of Group I. and Group IV. are compatible with the idea that the *A* and *B* factors are independently heritable.

Figure I. shows the distribution of the biochemical index. It will be seen that India forms the centre of a cone-like depression with an index of 0·56 and that as we pass out from it the figures for the most part show a steady gradient, but more especially to the east and west, until the extremes of 5·35 in north-west Europe and 5·7 in Australia and 9·2 in North America are reached. If instead of symbols the figures were plotted, it would be found that this appearance is very well marked. With symbols the smaller variations cannot be well shown. This coincidence of variation in the index with geographical distribution may be tentatively referred to two sets of causes, the one to be found in geographical factors acting as such upon the hereditary transmission of the *A* and *B* factors from parent to offspring and thus modifying the index of the race; the other to be found in considerations of the place of origin of man and his subsequent migrations. The first suggestion has apparently been dismissed up to the present as being unlikely, but it seems that this decision is somewhat too summary. The idea, it would seem, it at least worthy of a little more consideration.

As regards the second suggestion it would seem from this that at one time in the racial history of man there was a "biochemical" race *A*, containing only the *A* agglutinogen in the corpuscles, of practically universal distribution; later a second "biochemical" race *B* arose in India or thereabouts and gradually infused into the race *A*. In that way would be produced the high percentage of *B* individuals, leading to a low index in the Indian region, while a gradual decrease in percentage of *B* individuals with a corresponding increase in *A* individuals and in the index, would be met with the further we get away from that region. The point of origin of the first race *A*, must be left an open question, as far as the evidence given by blood groups is concerned; all that I can say is that at the time of the supposed origin of the *B* race the *A* race was apparently universally distributed.

To return to the two series of investigations on the Australian aborigines, it will be noticed that the direction of the gradient is exactly that which would be expected, an increase from north to south. Cleland's results confirm this very strikingly.[20] I should very much like to have measurements made on the aborigines of north-west Australia and of the Northern Territory.

Figure II. gives the frequency of Group I. as compared with descending biochemical index, while Figure III. gives the frequency of *A* and *B* factors when the races are arranged in order of descending biochemical index. It is hoped that these figures will give rise to some discussion amongst those interested and then this will lead to the disclosure of some noteworthy facts deducible therefrom. It cannot be too strongly insisted that truth must be sought in this line by cooperation between anthropological and serological methods.

In conclusion, I should like to urge the undertaking of numerous measurements on well-defined races in all parts of the world and a collection of all these figures for anthropological and statistical investigation. More especially does the duty devolve upon us, as Australians, of making as many investigations of this nature on the Australian aborigine as possible, while the opportunity exists. Let us not be forced to admit that we neglect those features of scientific interest for which our country is unique. The aborigine is undoubtedly a dying race and in very few generations the opportunity will be gone.

Acknowledgments.

This work was undertaken at the Institute of Tropical Medicine, Townsville and my thanks are due to the Division of Tropical Hygiene, Commonwealth Department of Health, for bearing the expenses incurred in the investigation and for the loan of all materials employed. Amongst the staff, Dr. Heydon especially has earned my gratitude; it was he who led me to make this research; I thank him for his indispensable help and suggestion. throughout. Thanks are also due to Dr. Broben for assistance in German translation. The members of the staff of the Aboriginal Settlement were of great assistance and especially Mr. Hoffmann by his interest in the work. My thanks are also due to Dr. A. H. Tebbutt for his kindly suggestion and for further references.

TABLE VI.

Graph No.	People.	Workers.	Reference No.	No. Exam.	Percentage. Group. I. % obs. (O_1.)	Group. I. % calc. (C_1.)	Group IV. % obs. (O_4.)	Group IV. % calc. (C_4.)	Percentage. A. II. + IV.	Percentage. B. III. + IV.	Bloch. Index.
1	North American Indians	C., Di.	12	862	77·7	78·1	0·1	0·4	20·3	2·2	9·21
2	Australian aborigines	Kt., Mc.	9	191	55·0	56·5	1·1	2·7	39·3	6·9	5·27
3	Anglo-Australians (Peterstal)	St., Sh.	18	252	39·5	42·3	2·4	5·25	58·0	9·9	3·35
4	Australian aborigines	Kt., St.	18	502	31·8	34·8	4·4	7·4	60·4	12·2	4·95
5	Australian whites	H.	3	500	46·4	47·9	3·1	4·7	46·4	10·2	4·55
6	German settlers in Transylvania	Lee	..	377	49·3	61·3	1·6	2·7	38·3	8·0	4·2
7		T., Mc.	9	1,176	52·6	53·7	3·1	4·2	39·9	10·6	3·8
8	French	Mu.	..	..	..	..	..	..	..	..	..
9	Germans (Heidelberg)[2]	H.	18	301	33·5	38·2	4·0	8·7	54·5	16·0	3·4
10	Germans on the Baltic	v. D., H.	3	500	43·2	46·6	3·0	6·4	45·6	14·2	3·2
11	United States	St.	18	[3]500[10]	36·0	39·0	5·7	9·0	47·9	17·0	3·12[3]
12	German settlers in Hungary	After Vz.	18	1,363	37·4	41·5	4·5	8·3	50·2	16·6	3·02
13	Italians	Vz.	18	?	45·0	46·3	5·0	6·75	45·0	15·0	3·0
14	Germans[5]	H.	3	476	40·8	46·0	3·1	7·3	46·6	15·7	2·9
15	Berlin Jews	v. D., H.	3	500	47·2	49·5	3·8	6·2	41·8	14·8	2·8
16	United States[11]	St., Ze.	18	[3]500	40·0	44·1	5·0	8·2	48·0	17·0	2·83
17	Austrians	Hk., Mo., Ot.	3	230	42·1	44·9	4·9	7·7	46·0	16·8	2·74
18	Bulgarians	L.	4	456	44·7	45·2	7·0	8·6	45·7	16·7	2·74
19	Banat Germans	H.	3	?	42·0	42·6	8·0	9·5	48·0	18·0	2·67
20	Czechos	Mu.	18	500	39·0	42·3	6·2	8·2	46·8	20·4	2·6
21	Serbs	Kv.	18	414	40·0	44·3	3·9	6·7	46·0	17·9	2·57
22	Greeks	H.	18	218	39·2	41·7	7·8	9·2	47·8	20·2	2·4
23	Leipzig	H.	3	500	38·0	42·7	4·6	9·7	46·4	20·2	2·3[4]
24	Berlin	Sk., Ze.	3	500	38·2	46·4	4·0	9·2	45·6	20·2	2·26[5]
25	Munich	St., Ze.	18	1,000	34·5	38·4	7·5	11·8	49·0	24·0	2·04
26	South Japanese	D., S.	4	750	37·8	41·8	6·4	10·4	45·8	22·8	2·0
27	Turks	F.	3	155	42·6	48·0	2·6	8·0	40·0	20·8	2·0
28	Japanese (middle)	F.	3	170	24·1	30·6	10·6	17·2	55·9	30·8	1·82
29	Hungarians	Vz.	3	500	36·8	41·5	6·6	11·2	44·6	25·2	1·8
30	North Japanese	F.	18	353	24·0	25·3	20·0	21·8	60·5	35·2	1·68
31	Arabs	H.	18	1,500	31·0	34·4	12·2	15·6	50·2	31·0	1·62
32	Melanesians	Hy., M.	18	151	32·5	35·4	11·3	14·7	48·3	30·5	1·53
33	Roumanian Jews	Mu.	17	500	43·6	47·5	5·0	9·0	37·4	24·0	1·56
34	Central Chinese	F.	16	753	53·7	56·3	3·2	6·8	30·0	19·5	1·54
35	Bantus	P.	17	211	26·1	29·8	15·3	19·0	54·1	35·1	1·54
36	American Negroes	Hd.	18	45	31·1	38·2	6·7	13·8	44·5	31·1	1·42
37	Russians	H.	18	260	..	..	..	..	28·8	20·8	1·4
38	Jews (Monastir)	K., Li.	3	1,000	49·0	51·4	5·5	7·8	32·4	24·0	1·4
39	Chinese	K., Li.	3	500	40·7	44·9	6·3	10·5	37·5	28·1	1·3[6]
40	Koreans (middle)	F.	12	100	38·8	44·5	5·0	10·7	38·0	28·2	1·3
41	South Chinese	F.	10	179	28·0	33·9	11·0	16·9	47·0	36·0	1·3
42	Malagasies	H.	3	35	24·6	30·0	14·5	19·9	50·3	39·6	1·3
43	Chinese (mixed)	C., Di.	12	400	37·1	41·4	8·6	12·8	44·5	35·1	1·26[7]
44	Chinese (all)	F. aft. C., Di.	12	111	45·5	49·5	4·5	8·6	44·5	31·1	1·08[7]
45	North Koreans	F.	10	184	29·0	35·4	4·5	8·6	46·5	30·7	1·09
46	Chinese (mixed)	B., V.	14	592	30·9	37·6	8·4	16·4	47·2	42·0	1·07
47	Javanese	B., V.	14	1,346	31·5	36·4	9·4	14·9	49·6	39·6	1·07
48	Annamese	H.	8	500	40·2	44·2	9·9	15·7	40·8	37·7	1·05
49	Sumatrans	B., V.	14	500	39·9	44·2	7·4	11·0	40·8	38·6	1·05
50	Chinese[13]	Li., W.	8	500	42·0	45·3	7·2	12·0	31·9	34·5	0·92
51	Senegalese	H.	14	5,546	43·7	48·8	6·7	10·5	33·1	36·4	0·9
52	Filipinos	Cb., Wd.	8	1,000	30·7	36·4	10·0	15·4	29·6	35·7	0·83
53	Manchus	..	7	500	43·2	47·6	5·0	9·4	29·7	35·7	0·82[8]
54	Gipsies in Hungary	F.	10	132	64·7	66·9	2·5	3·2	35·0	44·0	0·8
55	Chinese	Vz.	18	199	26·6	34·6	5·0	9·4	27·6	34·2	0·8
56	Cingalese	B., V.	18	385	34·2	40·4	6·8	12·0	15·7	20·3	0·8
57	Indians	H.	3	75	31·6	38·8	6·3	12·5	35·1	26·9	0·76
58	Pekin Chinese	H.-S.-L., St.	18	1,000	31·3	38·5	6·3	18·7	46·7	44·7	0·75
				4227	66·4	59·5	10·3	4·5	15·0	30·0	0·50[9]

For footnotes see the following page.

FOOTNOTES TO TABLE VI.

[1] Steffan gives 8·8 which does not agree with the percentages he quotes, which are those of Coca and Diebert's second group of 862 students.

[2, 3] Hirschfeld quotes the figures of von Dungern and Hirschfeld given in No. 15 from memory. It is not clear whether the figures given by Steffan in No. 10 as those obtained by von Dungern and Hirschfeld are for the same series, rectified by consulting the original figures, or whether they are for a separate series.

[4, 5] Hirschfeld gives 2·5 for each, which does not agree with their published percentages.

[6] Hirschfeld analyses this series as regards the occurrence of *A* and *B* factors as follows:

Region.	Percentage. A.	Percentage. B.	Total.
Central Russia ..	37·6	25·2	400
Siberia	36·5	29·0	321
Ukraine	35·1	33·3	111
Perm, Vologda, etc.	36·8	34·5	84

This gives indices of 1·5, 1·26, 1·05 and 1·07 respectively.

[7] Fukamachi's published figure is 1·18 which is incorrect for his frequencies.

[8] The percentages add up to 102·2. This latitude is too wide. A similar discrepancy exists in his results for the Javanese (No. 47).

[9] These results are rather unexpected. Confirmation would be welcomed.

[10] Ottenberg[4] gives 348 as the number examined.

[11] The figures given by Ottenberg[4] as the results of his own previously unpublished work, together with the figures of Moss and Hektoen, have been averaged in this series. The authors do not expressly state whether the subjects were whites.

[12] These were not all full-bloods.

EXPLANATORY NOTES TO TABLE.

Workers' names are indicated by the following abbreviations:

B., Bais.
C., Coca.
Cb., Cabrera.
D., Descatelle.
v. D., von Dungern.
Di., Diebert.
F., Fukamachi.
H., Hirschfeld.
Hd., Henderson.
Hk., Hektoen.
H.S.L., Hung See Liu.
Hy., Heydon.
K., Kilgoe.
Kt., Ketterer.
Kv., Kossovitch.
L., Landsteiner.
Li., Liu.
M., Murphy.
Mo., Moss.
Mc., McConnel.
Mu., Manuila.
Ot., Ottenberg.
P., Pirie.
S., Sturli.
Sf., Shiff.
Sh., Schutze.
Sk., Sucker.
St., Steffan.
T., Tebbutt.
V., Verhoef.
Vz., Verzar.
W., Wang.
Wd., Wade.
Z., Zeigler.

APPENDIX TO TABLE.

Since the table given above was compiled and the graphs drawn, the author has seen a reprint of an article by Hirschfeld in *"L'Anthropologie,"* Volume XXIX., 1918-19, page 505. This contains a dissection of the usually quoted figures according to subraces. Since it seems to me that the averaging of subraces is seldom justifiable in a consideration of the biochemical index in its distribution and since time is not available for a revision of the table, graphs and map, I thought it best to reproduce these figures in the form of an appendix to this table. It must be noticed, that the dissected figures appearing hereunder are not shown in any of the graphs or in the map contained in the body of the article.

ENGLISH.

TABLE V.

Sub-Race.	Groups. I. No.	I. %	II. No.	II. %	III. No.	III. %	IV. No.	IV. %	Total.	B.I.
English	180	44·6	180	44·6	31	7·7	12	3·0	403	4·4
Welsh	12	—	11	—	3	—	3	—	29	—
Scotch	29	—	21	—	2	—	0	—	52	—
Irish	11	—	5	—	0	—	0	—	16	—

FRENCH.

The numbers in the separate provinces too small to be given.

ITALIANS.

TABLE VI.

Origin.	Groups. I. No.	I. %	II. No.	II. %	III. No.	III. %	IV. No.	IV. %	Total.	B.I.
North (Piedmont Lombardy, Venice)	90	44·8	84	41·8	20	9·9	7	3·5	201	3·4
Central	43	54·4	27	34·3	4	5·0	5	6·3	79	—
South	103	46·8	79	36·0	31	14·0	7	3·2	220	2·3

SERBS.

Differentiation into provinces revealed no great differences.

GREEKS.

TABLE VII.

Origin.	Groups. I. No.	I. %	II. No.	II. %	III. No.	III. %	IV. No.	IV. %	Total.	B.I.
Asia-Minor	48	31·8	71	47·0	26	17·0	6	4·0	151	2·4
Greece	50	38·5	55	47·2	21	16·6	4	3·0	130	2·6
Thrace	35	—	24	—	12	—	6	—	77	—
Archipelago	26	—	25	—	8	—	1	—	60	—
Crete	15	—	20	—	6	—	1	—	42	—
Macedonia	14	—	10	—	6	—	1	—	31	—
Epire	3	—	3	—	2	—	1	—	9	—

BULGARIANS.

There is perhaps a greater frequency of *B* around Varna and the shore of the Black Sea.

RUSSIANS.

The data are given as a note to Table VI. and in the map.

ARABS.

TABLE VIII.

Origin.	Groups. I. No.	I. %	II. No.	II. %	III. No.	III. %	IV. No.	IV. %	Total.	B.I.
Tunis	100	50·0	50	25·0	39	19·5	11	5·5	200	1·2
Algeria	118	39·0	112	37·6	56	18·6	14	4·6	300	1·8

MALAGASIES.

Of 400 examined 266 were Hovas. The rest were scattered among several races. The figures for the Hovas are given in Table IX.

TABLE IX.

Groups. I. No.	I. %	II. No.	II. %	III. No.	III. %	IV. No.	IV. %	Total.	B.I.
120	45·5	73	27·5	61	22·5	12	4·5	266	1·1

NEGROES.

Of the 500 examined 238 were from Senegal, 81 Bambaras and the rest very much assorted in race. The figures for these two main groups are given in Table X.

TABLE X.

Race.	Groups. I. No.	I. %	II. No.	II. %	III. No.	III. %	IV. No.	IV. %	Total.	B.I.
Senegal	110	46·2	50	21·0	71	29·7	7	2·9	238	0·7
Bambaras	29	35·8	20	24·7	32	6·0	6	7·4	81	—

INDO-CHINESE.

Of the 500 examined 397 were from Tonkin. The index for these is 0·84.

INDIANS.

Two series of 500 were examined, but Hirschfeld suspects that the second series is not as reliable as the first, because the blood samples were not tested until the day following their collection. Only the provinces from which more than one hundred were examined are given below.

TABLE XI.

Origin.	Groups. I. No.	I. %	II. No.	II. %	III. No.	III. %	IV. No.	IV. %	Total.	B.I.	Series.
United Provinces	23	22·3	18	17·4	52	50·5	10	9·7	103	0·45	1st
Central ,,	67	37·4	43	24·0	62	34·6	7	3·9	179	0·73	2nd
Punjab	63	24·0	41	15·6	121	46·1	37	14·1	262	0·50	1st

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POINTS FROM PRACTICE: DIAGNOSTIC AND PROGNOSTIC.[1]

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As an example of how we would all like to diagnose conditions I often think of a patient who suffered from debility and diarrhœa of three weeks' duration.

A doctor, admitting the patient to hospital, ordered catechu and opium and starch and opium enemata, but the treatment failed. A house surgeon then examined digitally the interior of the rectum and found it to be full of hard masses of fæces which were causing the diarrhœa. So the condition was really constipation. The bowel condition was soon cleared up. The patient's persistent weakness was now vaguely supposed to be due to the heart and he was having slight attacks of dyspnœa. After about a fortnight the diagnosis was made the very moment a new honorary stepped inside the ward. He said: "There's a gander cough! Which is the patient with the aneurysm?" He had noted the very resonant, brassy cough which is produced when the lumen of the trachea is encroached upon by a mediastinal tumour or by an aneurysm, particularly an aneurysm of the transverse arch of the aorta.

An aneurysm in that situation is quite devoid of physical signs, but causes attacks of dyspnœa with cough and stridor, all due to pressure on the trachea.

How convenient it is to have once seen an ulceration on the chest of a diabetic or to be acquainted with the fact that *herpes zoster* may be caused by arsenic in the system or to know that dizziness may be the only symptom of an otherwise untraceable abscess of the scalp, the only other signs being feverishness soon passing off and perhaps a big white tongue.

Pyelitis is a diagnosis that does not always give final satisfaction, even when the diagnosis is correct. It is not nice to treat a patient for some years for attacks of what you call pyelitis with copious pus in the urine and to be so sure of the diagnosis that you forget to have the kidney examined by X rays for stone. I believe this mistake is fairly common.

Perhaps another pyelitis patient after several attacks will mention that he or she suffers from sneezing and the nose specialist will discover what is at the bottom of the pyelitis, namely, pus in the maxillary antrum. Or in another case of pyelitis the testing of the urine for sugar may give a positive result and you will then have this interesting problem: Is the glycosuria secondary to the pyelitis, the pyelitis having lowered the renal threshold for sugar just the same as carbuncle or furunculosis may do or conversely is the pyelitis a complication of diabetes, just as other inflammatory conditions such as *otitis media* may complicate diabetes?

As to diabetes itself it is remarkable that I have discovered it in two patients who complained of feeling run down and of losing weight and who had just recently consulted other practitioners for this without the ailment having been discovered. The clue to the diagnosis of several diabetes cases has been: "Run down, legs very tired, losing weight," especially "legs very tired."

[1] Read at a meeting of the Queensland Branch of the British Medical Association on May 7, 1926.

Acute appendicitis is not necessarily accompanied by retching or vomiting, even if the patient eats, but particularly is it true in regard to some patients who cease eating when the attack comes on. Such a patient, if not operated on, runs just as much risk as one who vomits.

In regard to pneumonia, an initial chill is said to be more common in pneumonia than in any other acute disease; the temperature taken during this initial feeling of coldness often shows that the fever has already begun. Not always, though. The temperature at the onset may be found by you to be 35·6° C. (96° F.), even though the patient is in a warm bed. He looks miserably cold. There may be even then a slight suggestion of stitch in the side. You exclude very carefully any other causes of collapse temperatures, but make sure by coming back in an hour's time, when you find him looking more like a patient with pneumonia should. His temperature is now 39·4° C. (103° F.) say, possibly there is a suspicion of slight friction and after a day or two the usual complete signs and symptoms of pneumonia appear.

A patient is under observation for something else and the temperature shoots up practically at once to 40° C. (104° F.). Nothing is complained of, except perhaps a little headache and malaise. (That same description so far might apply to a hospital patient who is contracting smallpox.) With one particular pneumonia patient the temperature came down within twenty-four hours—a one-day pneumonia. Plain signs of consolidation appeared about twenty-four hours later without any recurrence of feverishness. The two correct diagnoses in this case and likewise in the smallpox case respectively were made straight away at first sight of the temperature chart by excellent guesswork.

Notice that headache may be the only complaint of a patient with pneumonia and also the respiratory rate may be almost normal. Hence for a day or two or even more, while waiting for the chest signs to come out, one often wrongly suspects that the condition is typhoid or in these days influenza. Delirium may occur for two days or so after the crisis and though it is worrying, it is not of bad omen, if your patient seems reasonably strong. Death may occur forty-eight hours or more after the crisis, the patient, a man, say, of sixty years, getting a far away look and dying of heart failure just when your hopes were high.

When attending any child with pneumonia, it is useful to ask frequently whether sweats and pallor are present. Sweats of course will often be present in a simple pneumonia, but they are often more prominent in empyema and pallor is a rather important sign of empyema. In older children and in adults pallor is perhaps not so pronounced. Still, you have a patient (an adult) who you think is suffering from pneumonia, but the face is not flushed; it makes you feel there is something uncanny about this case of pneumonia. There is in fact some paleness. This should make you think of empyema before unnecessarily blaming the heart. The apex beat often has a tendency to get out slightly beyond the nipple in a patient with empyema. A pneumonia that is not resolving satisfactorily, a case that lags, a patient that looks pale either before an expected crisis or after a crisis or lysis, should make you think of empyema.

The temperature in empyema is irregular. It may be quite normal on the one or two occasions when you happen to "take it" or even more persistently. Empyema may also be a very chronic disease, besides absence of feverishness there may be absence of pain and of dyspnœa and very little cough. But often there is very considerable dyspnœa, particularly in acute cases and one does see empyema without sweats. And some of the patients that later show pallor, are highly coloured earlier in the disease and even livid.

Tenderness evinced on pressure at one spot on a bone may be the only sign of a fracture or fissure. A Colles fracture with the fragments in good position is one of the easiest to overlook and perhaps especially if it is complicating a dislocated wrist which you have just reduced. Most of us find it easy to decide in such a case, but apparently not all of us always. I do not think any of us on finding a tender spot on a clavicle would neglect to do the right thing. Curiously a tibia which was fractured in the lower third puzzled me more than any. The great thing is to err on the proper side.

As illustrating how slight the signs of fracture may be, I mention a little girl who met with an injury that no doctor was consulted about until two years later, despite the fact that the family lived on the lodge doctor's doorstep. There is now a large ugly false joint in the middle of the clavicle, apparently causing no harm except disfigurement.

Vomiting is sometimes seen among the initial symptoms of final heart failure, even in persons who previously have given little or no indication of heart failure and so can be counted as denoting a serious prognosis.

With respect to phimosis in the new-born child, it is readily observable that a foreskin which at birth seems scarcely to require operation, will be relatively longer at six months and worse still at one or two years and this occurs even though the foreskin is dilatable. Practically the only cases that do not take the course that I have mentioned, are those in which without retracting or touching the foreskin you can see the meatus of the urethra and a definite small circle of the glans surrounding it. This amounts practically to a recommendation to circumcise all other male infants; dilatation is unsatisfactory.

A furred tongue in a patient who has had influenza and whose temperature has recently declined, is an indication that the patient is extremely likely to have further trouble if allowed to get up, either complication, relapse or protracted convalescence. But if the patient mentions that