lymphatic leukaemia the results were normal. In the acute leukaemias and in polycythaemia vera both normal and high results were found.

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PORPHYRIA

A SOUTH AFRICAN SCREENING EXPERIMENT

BY

GEOFFREY DEAN, M.D., M.R.C.P.
Senior Physician, Provincial Hospital, Port Elizabeth, South Africa

AND

H. D. BARNES, Ph.D.
South African Institute for Medical Research, Johannesburg

Garrod (1923) credits Gunther with the first recognition that porphyria was an "inborn error of metabolism." If the term "porphyria" is confined to inborn errors of porphyrin metabolism, it excludes purely acquired disturbances whether of a temporary or permanent nature. An increase of porphyrin production and excretion without a genetic basis could be termed "hyperporphyrinism."

It is essential to distinguish between the genetic and purely acquired disturbances of porphyrin metabolism, because an attack of "acute porphyria" with the possible death of the patient occurs only if the gene for porphyria has been inherited. Diagnosis of porphyria in the latent phase is most valuable because in this disorder acute attacks are usually precipitated by the inadvertent use of certain commonly prescribed drugs, particularly barbiturates.

The classification of the porphyrias has in the past presented many difficulties, and the classification was symptomatic. In 1937 Jan Waldenström made a brilliant clinical survey of 103 cases of acute porphyria in Sweden; among these cases light sensitivity did not occur. He also reviewed the published cases where light sensitivity had only occurred in adult life, and these cases he termed "porphyria cutanea tarda." A third very rare type of porphyria has been described. In this type light sensitivity is present from birth, the urine is dark red because of the high excretion of porphyrin, and anaemia and splenomegaly are present. This very uncommon type of porphyria has been called "congenital porphyria" (Gunther, 1922). Schmid, Schwartz, and Watson (1954) classified the porphyrias on the basis of the porphyrin content of the liver and bone marrow of 31 cases. In two cases of congenital porphyria they found porphyrins concentrated in the bone marrow, and this rare group they named "porphyria erythropoietica." In the remaining 29 cases porphyrins were mainly found in the liver and they were therefore classified as porphyria hepatica. This group included the type described by Waldenström, a mixed type in which photosensitivity and acute symptoms may occur in the same patient, and possibly a third group of purely cutaneous type unassociated with acute attacks.

Genetic Classification

A plea is now made that the term "porphyria" should be reserved for the genetic disease only, for inborn errors of porphyrin metabolism, and that the porphyrias should be classified according to their genetic basis, as this would appear to be a fundamental method of classification.

There are three clearly defined genetic types of porphyria; there may be more.

1. *The Congenital Type, or Porphyria Erythropoietica.*—This is exceedingly rare and should be easily diagnosed by the marked light sensitivity from birth, the dark-coloured urine, anaemia, splenomegaly, and pink staining of the teeth. It is thought to be inherited as a Mendelian recessive characteristic (Cockayne, 1933).

2. *The Swedish Type of Porphyria Hepatica.*—This is the type described by Jan Waldenström, who has now collected over 320 patients with this disorder (Waldenström, 1957). He has shown that it is inherited as a Mendelian dominant characteristic and many of his families trace back to an original forebear from Northern Sweden. This type corresponds to the acute intermittent group of the classification of Schmid *et al.* Most cases of acute porphyria in Europe and in the United States belong to this group.

It has the following characteristics: (1) It is inherited as a Mendelian dominant gene and is not sex-linked. (2) Acute attacks occur most commonly in females between the ages of 16 and 50. (3) Acute attacks are often precipitated by drugs, especially barbiturates. (4) Light sensitivity does not occur. (5) There is a high excretion of porphobilinogen G. and amino-laevulinic acid in the urine during the acute attack and for a long time afterwards. If the disorder is causing symptoms, the Erhlich reaction (Schmid *et al.*) is usually positive. (6) There is either no increase or very little increase in urinary and faecal porphyrin excretion except during an acute attack. (7) In a porphyric family it is difficult to detect all the latent cases. However, the majority of latent cases can be suspected if not proved by using quantitative methods of analysis for porphobilinogen and amino-laevulinic acid. If there has been an acute attack in the previous few years the increase in porphobilinogen is usually sufficient to give a positive Ehrlich reaction.

3. *The South African Type.*—A number of papers (Barnes, 1945, 1951; Murray, 1949; Barnes and Marshall, 1952; Dean, 1953, 1956, 1957; Dean and Barnes, 1955) have shown that porphyria is a common disorder among the European population of South Africa. It is genetically different from the Swedish, or Waldenström, type, and has the following characteristics: (1) It is inherited as a Mendelian dominant gene and is not sex-linked (Barnes, 1945). (2) Acute attacks occur most commonly in females between the ages of 16 and 50. (3) Acute attacks are in our experience always precipitated by drugs, especially barbiturates. The acute attack resembles the acute attack in the Swedish type and in the past was usually fatal. (4) Light sensitivity will usually be found in some of the affected male members of the family, but it may be very slight or absent in the females. (5) There is a high excretion of porpho-

bilinogen during the acute attack, but the excretion returns to normal as the patient recovers. (6) There is greatly increased excretion of urinary and faecal porphyrin during the acute attack. (7) Latent porphyria can be detected by the slight increase in urinary and definite increase in faecal porphyrin excretion. For routine testing the detection of increased faecal porphyrin is particularly valuable. The Ehrlich reaction, however, in the quiescent phase is practically always negative.

In this type acute attacks, cutaneous manifestations, and symptomless cases with only increased excretion of porphyrin occur in the same family group, and are different manifestations of the same inherited disorder, just as asthma, hayfever, and eczema are different manifestations of inherited atopy.

Incidence in European South Africans

Porphyria is common among European South Africans because a very small group of early settlers have multiplied in ideal surroundings, so that to-day 1,000,000 of the present 3,000,000 white population hold the names of 40 original settlers and are direct descendants of those settlers in the 9th, 10th, 11th, and 12th generations. One of these early settlers was a porphyric. A subsequent paper will demonstrate that practically all South African porphyrics of European stock are descendants of this original prolific forebear who married at the Cape in 1688. That porphyria is a common disorder in South Africa is attested by the fact that one of us (G. D.) has diagnosed 564 cases of porphyria in 54 family groups.

Screening for Porphyria

In the Swedish type of porphyria the Ehrlich aldehyde test will usually be positive if the disorder is causing symptoms. To screen a large group taken at random for this type of porphyria in order to estimate the incidence of the disease will present great difficulty until a simple method for detecting slight increase in porphobilinogen and amino-laevulinic acid has been discovered, but once a case of porphyria has been diagnosed, all other members of the family should be investigated.

In the South Africa type the Ehrlich reaction is positive only during the acute phase. The disease is very common in South Africa, and the problem has been how to diagnose the disorder while the patient is still well and before an acute attack is precipitated by the inadvertent use of barbiturate drugs or a thiopentone anaesthetic. Fortunately the faecal and often the urine porphyrin excretion is usually above normal in adult porphyrics. The increased urinary porphyrin can be detected spectroscopically, although this is sometimes difficult, and by extraction with a solvent which then shows purple fluorescence in ultra-violet light.

The most useful test in the consulting-room can be carried out with a small fragment of stool; a little obtained on the end of a gloved finger by a "rectal examination" is sufficient. The small specimen is dissolved in 2 ml. of a solvent consisting of equal parts of glacial acetic acid, amyl alcohol, and ether. The brown, supernatant fluid is then decanted and viewed by ultra-violet light, using Wood's filter. In this light the negative specimens show a green or grey fluorescence. If there is an excess of porphyrin the fluorescence is a brilliant pink. A few specimens fall into an intermediate "doubtful" group, and an excess of chlorophyll can also cause pink fluorescence. In the positive and doubtful cases a quantitative analysis of faecal coproporphyrin and protoporphyrin is then carried out, and the urine is also examined for porphyrin excess.

The detection of increased porphyrin excretion is not a diagnosis of porphyria; the increased excretion may simply be due to a temporary hyperporphyrinism, often because of liver dysfunction, but sometimes of unknown aetiology. The possibility of porphyria must then be considered.

The diagnosis of porphyria of South African type in the non-acute state is sometimes difficult and time-consuming. The following factors should be taken into consideration: (1) The personal history; particularly any history suggestive of a previous attack of acute porphyria. (2) Whether the skin, particularly on the back of the hands, abrades and blisters easily, or has done so in the past. The skin on the back of the hand should be scraped with the finger-nail to see if it abrades easily. (3) Increased porphyrin excretion in the urine and faeces, particularly if this persists for some months. Normal total faecal porphyrin does not usually exceed 70 μg./g. dry weight. (4) The finding of other affected members of the family is important evidence in favour of porphyria. A careful family history should be taken and a family tree of all the relations should be drawn. These relations should then be interviewed and specimens obtained from them for porphyrin analysis. Often a number of them will be more clearly porphyrics than the patient who is first seen.

Results of Screening in South Africa

Two groups of the community were screened for porphyrin in order to help establish the incidence of the disorder in South Africans of European stock. The first group consisted of 608 patients from a mental hospital. The second group consisted of 645 nurses from four hospitals who voluntarily took part in the experiment.

Among the 608 mental hospital patients the sexes were about equal; the specimens were examined in small batches because it is not wise to screen too many specimens in a day as the ultra-violet light has an irritating effect on the eyes. Seven patients had greatly increased porphyrin excretion, in four of whom (Cases 1–4) this was considered due to porphyria (see Table I and case histories below).

Case 1.—No history of acute attack but skin abraded easily. High faecal and urinary porphyrin on four occasions. Member of family group (already investigated by G. D.) with 36 known porphyrics, of whom at least three have died from acute porphyria in the last few years.

Case 2.—No history of acute attack but skin abraded easily since age of 18. Persistently raised faecal and urinary porphyrin. Another member of family affected.

Case 3.—Recurrent blisters and sores on hands for past 15 years. Skin abraded with the slightest scratch. General health

TABLE I.—*Porphyria Among Patients at Mental Hospital*

Case No.	Sex and Age	Faecal Porphyrin (μg. g.)			Porphyrin in Urine	Mental Disorder	Skin Sensitivity *	Porphyrics in Family Study	Acute Attacks
		Copro-	Proto-	Total					
1	F 37	46	115	161	++	Epilepsy. Congenital blindness	+	36	Minor attacks
2	F 43	31	113	144	+	Catatonic schizophrenia	+	1	None known
3	M 33	327	259	586	+	,, ,,	++	—	,, ,,
4	F 36	210	740	950	+	Depressive psychosis after acute porphyria	++	27	Acute porphyria six months before admission
						Hyperporphyrinism Not Due to Porphyria			
5	F 83	596	445	1,041	+	Dementia	+	—	None known
Six months later					—	Mentally improved	—	—	
6	M 58	31	90	121	—	Congenital imbecile	—	—	None known
7	F 66	12	66	78	—	Congenital syphilitic imbecile	—	—	,, ,,

* Skin sensitivity. + = No scars or sores on skin, but skin on back of hand can be abraded with finger-nail more easily than usual. Skin sensitivity slight. ++ = Skin can be easily abraded and sores or scars are present. +++ = Skin sensitivity is marked with blisters and sores.

and nutrition good; not on drugs. Faecal and urinary porphyrin high on four occasions. No member of family traced.

Case 4.—Attended by G. D. in 1956 during severe acute attack. Developed a depressive psychosis a few months after recovery, but responded excellently to E.C.T. At least one relation died from acute porphyria.

In the remaining three patients the raised porphyrin excretion is thought to have been caused by temporary hyperporphyrinism.

Case 5.—Very high faecal and urinary porphyrin and skin abraded easily shortly after admission, when extremely malnourished and mildly demented. With good feeding porphyrin levels slowly fell, general condition improved, and skin no longer abraded. No personal or family history of increased skin sensitivity or increased porphyrin excretion; members of family examined normal. The hyperporphyrinism was probably due to a disturbance of liver function.

Case 6.—Faecal porphyrin raised only when first tested. Skin not sensitive, urine normal, no family history.

Case 7.—Faecal porphyrin at first just above normal, later normal. Skin not sensitive.

Specimens for examination were received from 645 nurses. Of these, 28 stools gave a pink fluorescence when examined by the method described and were sent for quantitative porphyrin analysis. In nine of these cases the fluorescence had been due to chlorophyll, but 19 nurses had a porphyrin excretion above 40 μg./g., four of these had an excretion level less than 70 μg./g., no history of a sensitive skin, and none of their relations were affected. The 15 remaining nurses were interviewed and their families were investigated, and 11 of them (Cases 8–18) were thought to have inherited the gene for porphyria (see Table II and case histories below).

TABLE II.—*Porphyria Diagnosed in Nurses*

Case No.	Sex and Age	Faecal Porphyrin (μg./g.)			Porphyrin in Urine	Skin Sensitivity *	Porphyrics in Family Study	Acute Attacks
		Copro	Proto	Total				
8	F 26	25	115	140	+	+	1	Minor attacks
9	F 46	105	470	575	+	−	4	Yes
10	F 28	38	134	172	++	+++	1	Cutaneous porphyria
11	F 19	69	127	191	+	+	1	No
12	F 18	71	132	203	−	−	27	,,
13	F 21	98	107	205	+	++	37	,,
14	F 47	229	585	814	−	++	2	Yes
15	M 27	88	135	223	++	+	4	No
16	F 51	21	69	90	+	+	2	,,
17	M 52	12	51	63	+	+	2	,,
	Mother	33	105	138	+	++		,,
18	F 19	163	250	413	−	−	2	,,
	Father	51	52	103	−	−		,,
	Possible Further Cases Among Nurses							
19	F 18	10	52	62	−	−	3	
	Sister	46	94	140	+	+	−	
	Mother	19	57	76	+	+	−	
	Aunt	23	79	102	+	+	−	
	Hyperporphyrinism Not Due to Porphyria							
20	F 29	54	194	248	−	−	−	
21	F 31	172	142	314	+	−	−	
22	F 33	12	63	75	−	−	−	

* See footnote to Table I.

Case 8.—Long history of recurrent abdominal pain; apparently neurotic temperament. Skin abraded easily; excess porphyrin in faeces and urine on three occasions. Another member of family had sensitive skin and high porphyrin excretion.

Case 9.—Long thought neurotic because of unexplained complaints of abdominal pain and vomiting. Skin did not abrade easily, but high faecal and urinary porphyrin excretion. During acute attack had porphobilinogen in urine. Other members of family affected. At time of investigation laparotomy under consideration.

Case 10.—Received treatment for recurrent blisters on hands diagnosed as epidermolysis bullosa. Increased faecal and urinary porphyrin excretion. Another member of her family similarly affected.

Case 11.—Mildly sensitive skin that abraded easily; faecal and urinary porphyrin was raised on four occasions. Had been adopted as baby. Another member of family affected.

Case 12.—No skin sensitivity, but faecal and urinary porphyrin was repeatedly raised. Member of large family group of porphyrics.

Case 13.—Scars on hands from previous blisters; skin abraded very easily; raised faecal and urinary porphyrin. Member of family group of 37 known porphyrics.

Case 14.—In 1949 desperately ill after course of sulphonamides (severe abdominal pain, repeated vomiting, and red urine; mentally disturbed and partially paralysed). Skin abraded easily; faecal and urinary porphyrin raised on four occasions. Another member of family with high porphyrin excretion.

Case 15.—Patient and his father and uncle had very sensitive skins and high faecal and urinary porphyrin excretion.

Case 16.—Skin abraded easily; two members of family similarly affected; faecal and urinary porphyrin repeatedly raised.

Case 17.—Slight tendency for skin on hands to abrade; faecal porphyrin at upper level of normal, urinary porphyrin increased. Mother and brother more obviously affected.

Case 18.—Faecal porphyrin persistently very high; father's and paternal grandmother's faecal and urinary porphyrin greatly increased. Probably inherited genetic porphyria with absence of symptoms.

Case 19.—Diagnosis still in doubt. Porphyrin excretion at upper limit of normal, but sister, mother, and aunt had increased skin sensitivity and raised porphyrin excretion.

A further three nurses (Cases 20–22) had increased faecal porphyrin but later specimens were normal; they did not have a sensitive skin and no other affected members of their family could be found. They are thought to have had a temporary hyperporphyrinism.

Case 20.—Faecal porphyrin raised on two occasions only. Three members of family investigated with negative results.

Case 21.—Two months after first investigation porphyrin excretion was normal.

Case 22.—Porphyrin excretion persistently at the high side of normal.

Conclusions

Fifteen porphyrics were found among the total number (1,253) who were screened in this survey, an incidence of about 1%; this is a high incidence. Screening for porphyria should be routine practice in South Africa, and is, in our opinion, as important a routine test there as testing the urine for sugar.

It must be remembered that porphyria is much less common in other parts of the world, although not so rare as was formerly believed. Several hundred cases have been reported by Waldenström (1957) in Sweden and the disorder is not very uncommon in the United States.

When porphyria is diagnosed the danger of taking certain drugs should be explained. A letter should be given to the patient stating the evidence for the diagnosis and warning about the uses of drugs in this condition; this letter should be shown to any doctor subsequently consulted.

In the present survey 15 were thought to have inherited the gene for porphyria among 1,253 who were screened for the disorder. This suggests that the gene among the white population in the Eastern Cape Province of South Africa is about 1%.

Summary

Porphyria has been defined as an "inborn error of metabolism." This excellent definition distinguishes porphyria from hyperporphyrinism due to disturbances of porphyrin metabolism which are not of genetic origin.

Increased porphyrin excretion may be due to porphyria or hyperporphyrinism. It is important to distinguish between the two because in porphyria certain commonly used drugs may precipitate an acute disturbance of porphyrin metabolism with possible paralysis and death. Acute porphyria occurs only if porphyria has been inherited. Although the Swedish and South African types are genetically different disorders the acute attacks are very similar in both types.

The classification and diagnosis of porphyria are discussed. The methods used for detecting the South

African type of porphyria during the non-acute or quiescent stage are described.

The way to distinguish between porphyria and hyperporphyrinism is described; particular emphasis is laid on the importance of studying the relations of the patient. The differential diagnosis is sometimes difficult.

A simple method for screening the stool for excess porphyrin is described. Sufficient stool for the test can usually be obtained on the gloved finger while making a rectal examination. The urine should also be examined for porphobilinogen and excess porphyrin.

Two groups of South Africans were screened for porphyria using the methods described. Among the 608 patients at a mental hospital four were found to have porphyria, and among 645 nurses 11 were thought to have inherited this gene. The incidence among the nurses was greater than at the mental hospital, but the numbers are not sufficient to be statistically significant.

Screening for porphyria should be routine practice in South Africa, where the disorder is often found.

We would like to thank Professor Jan Waldenström, of Lund University, for enabling one of us (G. D.) to see a number of Swedish porphyric family groups. Professor Waldenström is in agreement with us that the Swedish type of porphyria is a different genetic disorder from the South African type. A detailed comparative study of these two types of porphyria is in progress.

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Nursing and Midwifery, the latest booklet in the Central Youth Employment Executive's "Choice of Careers" series, is the first of two companion booklets on nursing; the other, *Nursing for Men*, will be published later this year. After describing the rise of nursing as a profession, the booklet proceeds to deal with the different branches of nursing, such as hospital work, home nursing, work in clinics, health visiting, and industrial health nursing. The surroundings in which the work is done are illustrated by photographs. Training for nursing cannot begin until the age of 18, so advice is given for those who leave school earlier on "bridging the gap"—for instance, by attending pre-nursing courses and "cadet" schemes. There is a section on the personal qualities of the nurse: "She must have kindness, patience, and sympathy, yet she must not be sentimental or emotional, and must know when to be firm . . . there is no place in the nursing profession for the girl who scamps her work and trusts to luck." Other sections contain information about the training of nurses and midwives and about the many additional qualifications they may obtain. Finally the openings and prospects for nurses in Britain and overseas are touched upon. (Her Majesty's Stationery Office, price 1s. 9d. net.)

THE HUMAN SPERMATOZOON

BY

LORD ROTHSCHILD, F.R.S.

From the Department of Zoology, University of Cambridge

[WITH SPECIAL PLATE]

Until the advent of the electron microscope comparatively little was known about the fine structure of human spermatozoa. Even when this instrument and the thin-section technique became available, progress was slow because of the unusual fragility of human spermatozoa as compared, for example, with those of the bull or the ram. An important review, including a comprehensive bibliography, of the ultrastructure of the human spermatozoon was published in Sweden by Ånberg in 1957. As this is not readily available in the United Kingdom, the electronmicrographs reproduced on the Special Plate may be of interest.

Structure of Spermatozoon

A longitudinal section of the head and middle-piece of a spermatozoon is reproduced in Fig. 1, the probable plane of section being shown in the inset diagram. The head is bounded by the cell membrane, the acrosome terminating near the equatorial segment, and the nuclear membrane. The fragility of the human spermatozoon may be put to good purpose in identifying these structures, as they often become detached while the material is being prepared for electron microscopy, as in Fig. 2. Two vacuoles containing partially opaque material are present in the head. According to Ånberg there is a basal plate at the posterior end of the sperm head, but this is not visible in the specimen illustrated. The neck contains a remarkable structure which seems to consist of a series of electron-dense rings, somewhat reminiscent of vertebrae. They may be connected with the tail and middle-piece fibrils which are shown in the transverse section through the middle-piece in Fig. 3. The middle-piece consists of 29 fibrils embedded in a protoplasmic matrix and arranged as follows: an outer ring containing nine fibrils; an inner ring, concentric with the outer one, containing nine pairs of fibrils; and two fibrils located at the centre of the system. At first sight the inner ring may seem to consist only of nine fibrils; but close examination shows that each of the nine "fibrils" is, at the least, double.

One or, possibly, two mitochondrial sheaths or ribbons, bounded by the cell membrane, appear to be wound in a rather irregular spiral round the fibril system. According to Ånberg the mitochondria, whose characteristic structure is clearly visible in Fig. 3, do not form a spiral ribbon round the middle-piece; but Fig. 1 suggests that they do, even though the spiral is somewhat irregular. The plane of the section in Fig. 1 is such that the spherical head centriole described by Ånberg (1957) and by Burgos and Fawcett (1955) is not visible; nor is the tail, which would be over 100 cm. long at the magnification of Fig. 1. The tail contains 20 fibrils, the outer ring of nine, which can be seen in middle-piece sections, being absent along most of its length. There is a fibrillar sheath, which may or may not be spiral, round the matrix in which the tail fibrils are embedded.

The electronmicrographs were taken with the Siemens Elmiskop I at the Cavendish Laboratory, University of Cambridge. I am indebted to Mr. R. W. Horne for assistance in taking them. This work is supported by the Medical Research Council.

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