better index of the adrenocortical function than is the total 17-ketosteroid output.

Comparison of the effect of operation and of cortisone on D.H.A. output in cases conforming to type β has not proceeded far enough to admit of definite conclusions, but preliminary work indicates that as a result of unilateral adrenalectomy D.H.A. is reduced in much the same proportion as the total 17-ketosteroids. In other words, there is no deviation from type β . The effect of treatment with cortisone compared with that of unilateral adrenalectomy depends on the dosage. When only enough cortisone has been administered to halve the total 17-ketosteroid output—that is, corresponding approximately to the reduction that can be achieved by the removal of one adrenal—there is again no deviation from type β . If, however, enough cortisone is given to bring down the total 17-ketosteroids to normal values, the D.H.A. is almost completely eliminated, signifying a change from type β to type α .

Summary

A short clinical and biochemical review of 37 cases of adrenal pseudohermaphroditism is presented.

Further biochemical studies show, on the basis of dehydroisoandrosterone excretion, that these patients fall into two distinct groups.

The effects of unilateral and bilateral adrenalectomy on 17-ketosteroid excretion are shown.

Observations of the action of cortisone on 17-ketosteroid excretion are also discussed.

Treatment is more effective when undertaken before puberty.

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PORPHYRIA

BY

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Porphyria is a disorder involving the pyrrole pigments which take part in respiratory metabolism. It is a familial condition characterized by chronic mild neurological and cutaneous symptoms with occasional acute exacerbations. I have personally attended 12 cases of acute porphyria. The first three of these patients died, the last nine recovered. They were from eleven different family groups.

In each family the genealogical tree has been worked out in the fullest detail possible. Some of the groups were large—for instance, in the first family there are 460 descendants of the first member of the family who is known to have had the typical sensitive skin. As will be seen, many in each family group have inherited porphyria. The sex distribution was about equal, but the most severe cases of acute porphyria were in women between the ages of 19 and 50. The reason for this predominance of women is discussed below. All the families seen were Afrikaners perhaps related far back in South African history. It is a common con-

dition in South Africa, and Barnes (1945, 1951) has already published the history and laboratory findings of over 40 cases. The mild cutaneous type is common in the Bantu. The condition has also been described in cattle (Fourie, 1936).

The porphyrin structure is present in haemoglobin and in certain other respiratory enzymes. When porphyrins are produced in excess they are excreted in the urine as uroporphyrins, as coproporphyrins, or as a colourless chromogen, porphobilinogen. They are also excreted in the faeces. Porphyrins may colour the urine red or reddish brown. Garrod (1923) considered that porphyria belonged to the group of familial disorders which were inherited as "inborn errors of metabolism." The disturbance in metabolism is aggravated particularly by certain drugs and by pregnancy. Porphyrinuria, usually an excess of coproporphyrins, may also occur in liver disease, anaemia, or after poisoning by certain chemicals, in individuals who do not suffer from familial porphyria.

My experience is limited to porphyria in the adult, when it presents a familial syndrome which is characteristic. Unfortunately, however, the syndrome can best be seen in retrospect, and during a remission it is often difficult to diagnose. In the acute stage diagnosis may be made too late to save the patient's life. It is a syndrome that is not well described in the medical textbooks, and if it is to be diagnosed early a much clearer concept of the condition will have to become widespread knowledge among the profession. However, it is extremely important that the diagnosis be made, because frequently it is the doctor who is unwittingly responsible for the patient's death.

The Syndrome

Porphyrics often have an apparently neurotic make-up. Many have complained for years about various pains, particularly in the abdomen. Because of these pains they often become drug addicts, taking barbiturates, aspirin, or a variety of patent medicines. They have usually seen many doctors, and unfortunately they may have had a number of abdominal operations. The urine is generally normal in colour, but, on questioning, patients may recall that their urine has been red during an illness in the past. The abdominal pain sometimes occurs in acute attacks accompanied by vomiting, when a diagnosis of appendicitis, intestinal obstruction, or some other abdominal emergency may be made. Patients, or some of the relatives, frequently have a skin that breaks down easily, forming blisters and then ulcers. This sensitivity of the skin is not usually apparent until adult life. The exposed areas of skin are particularly affected—the back of the hands and, in women, the front of the legs. Sunlight and minor injury appear to be precipitating causes. This ulceration is more common during pregnancy. The ulcers are usually small and multiple, and take a long time to heal. They are deep enough to leave permanent scars. Rather surprisingly, the ulcers of the skin are often most troublesome when the condition is quiescent, and may be absent during an acute attack. Conjunctivitis is a not uncommon symptom, and small pigmented spots that fluoresce in ultra-violet light may be seen in the retina.

During pregnancy the patient is usually much worse, perhaps partly because she is then given sedatives. There is often a history that previous pregnancies were terminated because of pains, vomiting, and hysteria.

In many cases a family group is first found to be porphyric when one of its members has an acute attack. More likely than not this acute attack follows an abdominal operation, particularly if thiopentone has been given as an anaesthetic, or it occurs during pregnancy. Women are more prone to be operated upon for lower abdominal pain than men; this fact and pregnancy probably account for

the frequency of acute attacks in women. In my experience the chronic stage is found just as commonly in men. After operation the patients' behaviour is usually hysterical. They complain of severe pains all over the body, but particularly in the abdomen. Their operation wound is slow to heal; they are uncooperative, and it is difficult to maintain their nutrition. After a few days they complain of weakness, which at first may be regarded as hysterical. Then it is realized that they have developed a lower motor neurone type of paralysis. Their reflexes disappear, their pupils are dilated, and their heart rate is rapid. The blood pressure may be raised. During the acute illness they pass reddish-brown urine which darkens on standing. As the paralysis progresses their mental symptoms and abdominal pains lessen, and they often have an awareness of approaching death. At this stage the prognosis should be guarded, but it is not as grave as was formerly thought. If the patient does not die, the peripheral neuritis may persist for many months.

Evidence of impaired liver function can be found during an acute attack. For instance, the icteric index is raised and the albumin/globulin ratio is altered. The blood chloride may be lowered if there has been much vomiting.

There is usually a leucocytosis. The cerebrospinal fluid is normal. In my experience the electrocardiogram shows a tachycardia, but no very characteristic change. Chemical and spectroscopic examination of the urine confirms the presence of porphyrins.

Post-mortem examination of the liver shows hepatitis and a deposit of pigment in the liver cells. There is strong evidence that in the adult the production of porphyrins takes place in the liver. Examination of the spinal cord shows degenerative changes in the anterior and posterior horn cells; the sympathetic ganglia show similar changes. There is a demyelination of peripheral nerves (Goldman and Kaplan, 1951).

Differential Diagnosis

The chronic or mild case seen in the consulting-room, with complaints of abdominal and other pains for which no organic cause can be found, presents a difficult diagnostic problem. Diagnosis depends on a very full personal and family history, with special reference to "nervous breakdowns," abdominal pains, operations, health during pregnancy, drug-taking, and any personal or familial tendency for the skin to blister. Inquiry should be made into the cause of death of any blood relation, death from generalized paralysis being of particular significance. Often an older member of the family, perhaps an aunt, can cast great light on the problem by describing the skin lesions or death from paralysis of members of a previous generation. Other organic disease must of course be excluded. A history in any member of the family of passing red urine may be a most useful clue. The urine should be tested chemically and spectroscopically, as a positive result will confirm the diagnosis. Sooner or later porphyrins will usually be found in the urine on careful spectroscopic examination.

When the patient is first seen during an acute attack the differential diagnosis must be made from hysteria, appendicitis, or intestinal obstruction. The attack generally follows drug-taking or an anaesthetic, but it may arise apparently spontaneously, particularly during pregnancy. If paralysis has already developed, it must be differentiated from poliomyelitis, the Guillain-Barré syndrome, or hysteria. The history and examination of the cerebrospinal fluid and of the urine will settle the diagnosis. At this stage porphyrins are present in excess in the urine. An increased excretion of porphyrins may also occur in hepatitis in an individual who is not suffering from familial porphyria, but the amount is less and porphobilinogen is not usually present.

Tests for Porphyrins

In the acute stage the urine may immediately suggest porphyria because of its red colour, or it may darken on standing in sunlight, particularly after the addition of

hydrochloric acid. If the urine is normal in colour Ehrlich's aldehyde saturated with sodium acetate should be added, the urine shaken, and chloroform added. When porphobilinogen is present a red colour will appear in the aqueous solution (the Watson-Schwartz test). Porphyrin urine will fluoresce on exposure to ultra-violet light. The presence of porphyrins can be confirmed by spectroscopic examination using a thick layer of urine and a good spectroscope; the porphyrin absorption bands will then be seen. The methyl esters of porphyrins have a characteristic melting-point after crystallization from urine. It should be remembered that blood, beetroot, or drugs may also cause red urine.

In the chronic or latent stage the usual tests for porphyrins may be negative. Both the urine and the faeces should be examined by a biochemist who has had considerable experience in testing for porphyrins. If the urine has to be sent away, a few drops of chloroform may be added as a preservative. A 5-in. (12.5-cm.) layer of urine should be examined spectroscopically after the urine has first been filtered carefully. This is usually a more sensitive test than looking for fluorescence in ultra-violet light. However, excess porphyrin may not be present during a remission. As an experiment 15 specimens of urine from proved porphyrics, in the chronic or latent stage, were sent to a biochemist especially interested in porphyria detection. Porphyrins were found in excess in four specimens. Another specimen of urine was sent from most of the same patients, and three of the previous four were still positive but one was now negative. Three more specimens, however, were now positive. A positive result was finally obtained in 12 out of the 15 cases. These specimens of urine were sent in identical numbered bottles with 18 controls. All the controls were negative.

Case Histories

Only a brief summary will be given of seven cases of acute porphyria. They all come from different family groups, and in each case a genealogical tree has been worked out in the greatest detail possible. All known members of the family groups were contacted. Members of the family have been regarded as porphyrics only if the spectroscopic examination of the urine was positive, or if they showed the typical skin lesions and their immediate relatives were proved porphyrics. In the families studied porphyria was inherited as a Mendelian dominant. Detailed family trees and the inheritance of porphyria will be discussed in a subsequent article.

Case 1.—February, 1951. A nurse aged 19 suffered severe abdominal pains after the use of barbiturates. As she was thought to have an intestinal obstruction a laparotomy was carried out and thiopentone was used to induce anaesthesia. After the operation she was delirious, the abdominal pains increased, and she vomited repeatedly. Within three days she was completely paralysed, her pulse was rapid, and her pupils were dilated and fixed. Her urine was red and was strongly positive for porphyrins. She was fed intravenously, but died eight days after the operation.

Over the course of a year a very detailed genealogical tree was obtained. All living members of the family were contacted. The original head of the family, who is still remembered by one remaining son-in-law, died in 1884. He himself had a sensitive skin that had blistered repeatedly and left numerous scars on his hands. As descendants he had 10 children, 61 grandchildren, 173 in the third generation, 197 in the fourth generation, and already 19 in the fifth generation. Fifty-one inherited a sensitive skin that blisters and ulcerates easily. A further 6 women have porphyrinuria, but no history of a sensitive skin. At least 57 of the family appear to have inherited porphyria (22 male, 35 female). Thirty-nine of these are still alive, and excess porphyrins have been found in the urine of 37 of them. Of the remainder eight women and one man have died with typical symptoms of acute porphyria, including two of the sisters of Case 1. Altogether 402 members of

this family have now had their urine tested for porphyrins ; all the others were negative.

Case 2.—February, 1951. A married woman aged 42 had been regarded as neurotic all her life. In 1932, following thiopentone anaesthesia, she became paralysed and was unable to walk for six months. She was seen by me after a hysterectomy, during which she was given thiopentone ; she complained of severe abdominal pains and vomited repeatedly, and became paralysed over the course of three weeks. Porphyrins were found in her urine. She died six weeks after the anaesthetic. Of the 162 adult relations traced on the affected side of the family, 39 (17 male, 22 female) had inherited porphyria ; most of these had a sensitive skin that blistered easily. Her brother and two other members of the group died with symptoms of acute porphyria.

Case 3.—July, 1952. A married woman aged 41. The skin of her hands and the front of her legs had blistered easily all her life. Following thiopentone anaesthesia for operation on varicose veins she developed abdominal pains and vomiting, and became paralysed. Porphyrins were found in the urine, which was reddish black. She died ten days after the operation in spite of A.C.T.H. and intravenous drips. Of 44 adult relatives on the affected side of the family 17 (7 male, 10 female) were porphyrics. A male cousin of this patient also had an attack of acute porphyria after taking barbiturates. He did not become paralysed and made a good recovery.

Case 4.—August, 1952. A married woman aged 27 had recurrent attacks of vomiting and abdominal pain during her pregnancies. She became partially paralysed after thiopentone anaesthesia when the third pregnancy was terminated, but she recovered. Six months later the abdominal pains recurred after the use of barbiturate sedatives. She was given thiopentone for a laparotomy. A few days afterwards she became delirious and partially paralysed. Porphyrins were present in her urine. She slowly recovered, but is now troubled with recurrent blisters and ulcers on her hands and arms. Of 114 adult members of her family on the affected side 24 (17 male and 7 female) have inherited porphyria. Five (3 male, 2 female) other members of the family have died from generalized paralysis after drugs and anaesthetics.

Case 5.—March, 1953. A married woman aged 35 had had repeated operations for abdominal pains. She had severe attacks of vomiting and pain during her pregnancies. After thiopentone anaesthesia for a hysterectomy she became delirious. Her urine gave positive porphyrin reactions. Her liver function was impaired. Although all drugs were stopped she became completely paralysed within 48 hours. She was given A.C.T.H. and fed by intravenous drip. She is slowly recovering, and five months later was starting to walk. Of 25 adult members of the family on the affected side 17 have inherited porphyria.

Case 6.—March, 1953. A married woman aged 36 had suffered from recurrent blisters on the back of her hands and on the front of her legs since she was 18. Her first pregnancy was terminated because of vomiting and the passing of red urine. Following thiopentone anaesthesia for appendicectomy she was hysterical, complained of severe abdominal pain, and vomited repeatedly. Her urine became dark and porphyrins were present. Although all drugs were stopped and she was fed intravenously, she became almost totally paralysed. During the next six months she made a good recovery, but her jerk responses are still absent and there is impaired liver function. Nine of the 43 adult members of the family on the affected side have inherited porphyria.

Case 7.—December, 1948. A married woman aged 29 vomited repeatedly during her second pregnancy and complained of severe abdominal pain. She became jaundiced and her urine was reddish brown. She was given thiopentone for a curettage. After the operation she was delirious, and within three days she was totally paralysed. She recovered, but was in hospital seven months. She lost

a great deal of weight during this illness. Two years later she was given thiopentone for dental extractions ; she again became delirious, her urine was reddish brown, and all the tests for porphyrins were positive. Again she became paralysed, but recovered in about five months. Her skin now blisters on the back of the hands and at the ankles. Seven of the 45 adult members of the family on the affected side have inherited porphyria.

Other Cases.—Five men have also been seen by me during attacks of acute porphyria ; one has already been mentioned. Of the other four, one became partly paralysed after thiopentone, and one had an acute cutaneous reaction with hepatitis after painting wood with a preservative, pentachlorophenol. The remaining two became acutely ill with severe abdominal pains and vomiting, with a strongly positive red urine, after taking phenobarbitone. They recovered without becoming paralysed. Two other cases of porphyria (one man and one woman), belonging to other groups, were diagnosed in the consulting-rooms during the latent phase. They were complaining of abdominal pains and had been regarded as neurotic. Porphyrins were found in their urine. Several of their relatives were also affected. From these 13 family groups 216 porphyrics have been traced and warned.

Treatment

When porphyria is diagnosed in the chronic stage treatment consists primarily in stopping all drugs that may be aggravating the condition. This includes a wide range of substances, particularly sedatives such as barbiturates. The doctor must advise the patient that he may suffer some pain, but that all will be well so long as certain drugs, including patent medicines and alcohol, are scrupulously avoided. Penicillin may safely be given, but sulphonamides are harmful. Aureomycin should not be given intravenously because of its possible deleterious effect on the liver. The extreme danger of thiopentone anaesthesia must be pointed out to the patient and to the relatives, and to convince them they should be given a few anonymous case histories to read. If an operation is absolutely unavoidable because of serious organic disease ether is probably the least harmful anaesthetic. Many porphyric patients seen by me have had anaesthetics other than barbiturates and made good recoveries. If the skin is photosensitive the patient should keep out of the sun as much as possible, and wear a hat and gloves out of doors. Protective creams, such as 5% tannic acid in vanishing cream, are of some value.

In the acute stage good nursing, preferably in a private room in a hospital, is probably the most important single factor in aiding recovery. As there is as yet no specific antidote for porphyria, treatment depends on maintaining the patient's general condition and avoiding any harmful drug. These patients must not be given sedatives, except perhaps a little paraldehyde. In my experience it is possible to keep them fairly comfortable with the aid of placebos such as vitamin tablets. If there is vomiting the nutrition and electrolyte balance must be carefully watched and maintained by intravenous feeding. Repeated examination of the blood and urine chlorides and the blood potassium may be required. As there is often evidence of liver damage I give these patients a high-protein high-carbohydrate diet and a high fluid intake. I also give them methionine, vitamin B₁₂, vitamin B complex, and vitamin C. Dimercaprol protects the nerves against certain nerve poisons. It was given to one patient who was threatened with paralysis of respiration, but nevertheless she died : there is no evidence that it helps in porphyria. A.C.T.H. was given to seven patients ; it certainly made them feel better and appeared to be of value. As a last resort an artificial lung can be used, but if respiration is paralysed the patient usually dies. In the recovery stage physiotherapy is of the greatest importance. Recovery from paralysis takes many months if the paralysis is severe.

Once a case of porphyria has been diagnosed it is the doctor's responsibility to investigate the family history fully. The best method is to work out a complete family tree,

and this may take several months. Inquiry must then be made in order to discover which side of the family is affected and which members on the affected side have inherited porphyria. The urine of the relatives should be examined spectroscopically for porphyrins. It must not be forgotten that the syndrome will present in different ways in different members of the group. Many will have complained of symptoms the cause of which will not have been known. The doctor will be greatly rewarded when he makes the correct diagnosis in a patient who has long been regarded as neurotic, and who has had perhaps a number of operations on account of abdominal pains. All affected members of the group must be interviewed. Porphyrics should be given a letter stating the evidence on which the diagnosis has been made, and mentioning the danger of barbiturates and other drugs in this condition. They should show this letter to any doctor they may consult in the future.

Summary

Porphyria, a serious familial disorder, is common in South Africa.

In the chronic or latent stage the diagnosis will be made only if the doctor is alive to the possibility that the disorder may cause symptoms which are apparently neurotic, particularly abdominal pains.

In the latent stage no reliance can be placed on a negative urine test, as at this stage excess porphyrins may not be excreted. Diagnosis depends on a full personal and family history. During an acute attack examination of the urine will confirm the presence of porphyrins.

In acute attacks of obscure abdominal pain the abdomen should be opened only if there are definite indications that surgery is necessary, and after the urine has been carefully examined for porphyrins.

The increased use of barbiturates in the routine treatment of nervous disorders may well be responsible for bringing this syndrome to the fore. Intravenous barbiturate anaesthetics are particularly likely to precipitate a severe attack, often with resultant paralysis.

If one case of porphyria is diagnosed it is the doctor's responsibility to go very fully into the family history and interview as many relations as possible. This will lead to the discovery of other undiagnosed cases and result in preventing much suffering and unnecessary operations.

In the relatives of the cases described here, different manifestations of the syndrome were seen in different members of the family group. This suggests the unitary nature of porphyria in the adult, and that division into subtypes is artificial.

I would like to thank the doctors who called me in consultation to see these patients for their permission to publish the case histories, and for their unfailing help and encouragement in the preparation of this article. Also Mr. Barnes, of the South African Institute for Medical Research, Johannesburg, for investigating many specimens of urine for me. My thanks are also due to Dr. Harington and the staff of the Research Institute, Port Elizabeth.

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INTRA-ARTERIAL TRANSFUSION

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Intra-arterial transfusion is considered to be of great value in the treatment of haemorrhage. We agree that this is the main indication for its use, but the object of this paper is to report our observations on the value of the method in combating circulatory collapse due mainly to causes other than bleeding. An excellent survey, historical, experimental, and clinical, of the literature concerning the use of the intra-arterial route for transfusion has been published by Seeley and Nelson (1952). Valuable experimental and clinical evidence is given by Porter *et al.* (1948), Robertson *et al.* (1948), Jones *et al.* (1950), and Bingham (1952), among many others.

In general, where the effect of peripheral anoxia has been sufficient to cause relaxation of peripheral vessels and an increase in the volume of the vascular bed, with stagnation of the blood flow, fluids given intravenously will tend to accumulate in the relaxed venous channels. Vasopressors are often ineffective in restoring tone to the anoxic vessels, prolonged hypotension may have led to myocardial damage, and venous congestion may be present. Intra-arterial transfusion may even then restore the blood flow enough to reverse stagnation and permit adequate tissue oxygenation once more. When bleeding is not the cause of the collapse, such an improvement is only a respite during which definitive treatment may be applied, but it is an essential respite.

Apparatus

In brief, the method of intra-arterial infusion consists in the introduction of whole blood or blood substitutes under pressure into a suitable artery.

The apparatus* may be a simple transfusion set with a double bellows as the source of pressure, or take a more complicated form incorporating a manometer for measuring the infusion pressure. An oxygen tank with control valve is used as the motive force in some apparatus. If vasospasm is pronounced, a syringe and three-way stopcock are used.

*Since this paper was written we have used a form of rotary roller pump for rapid transfusions, and have found it superior to the apparatus described, and less apt to provoke vasospasm.

FIG. 1.—Apparatus for intra-arterial transfusion. 1=Air reservoir. 2=Blood. 3=Safety screw caps. 4=Aneroid manometer. 5=Sphygmomanometer bulb. 6=Drip chamber with float (Macintosh and Pask, 1941).