PATHOLOGICAL ASPECTS OF THE DISEASE PATTERN

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You have heard the opinions of clinicians. They are good fellows really, but they so rarely know what is going on inside the patient. To establish the truth, you must call in the pathologist who gets a much wider view of the problem.

For example, one patient presents with pericardial effusion and congestive heart failure; the clinical diagnosis is tuberculous pericarditis. Another patient comes with fever, cough, foul sputum; clinically it is a lung abscess. Yet both those patients have cancer of the oesophagus; in the one infiltrating the pericardium, in the other causing a tracheo-oesophageal fistula. The oesophageal tumour was never suspected since the patients never complained of dysphagia. This, of course, highlights one major difficulty in Bantu medicine – the problem of eliciting an accurate history.

Bantu Pathology

All types of pathological findings in the White and Indian are found in the Bantu. We must get away from the old idea that certain diseases do not occur in the Bantu – it is too often a fatal concept. Of course the incidence of disease varies greatly – peptic ulcers are not common, but I have done many postmortem examinations where a perforation was missed because the patient was a Bantu. Gallstones are infrequent but I have seen patients die with undiagnosed lithiasis of the common bile duct. Another major difficulty is that the Bantu patient is likely to have not one but 2 or even 3 different pathological conditions.

A third major difficulty, this time concerning therapy, is that pathology in the Bantu is usually gross, such as that found in Western countries several generations ago, e.g. aneurysms of the aorta which erode vertebrae and expose the spinal cord and fulminating amoebic dysentery which destroys the whole of the colonic mucous membrane in patients dying soon after admission. I think the rows of skin incisions often explains the reason.

I should like to give you a bird's eye view of pathology as we see it at King Edward VIII Hospital in Durban – the biggest hospital in the southern hemisphere. Table I shows age groups of necropsies in 1962 when we had an 85% necropsy rate. We see that some 50% of hospital deaths were in children under the age of 3 years – and this excludes all neonatal deaths occurring in the first month.

TABLE I. TOTAL NECROPSIES 1962 (EXCLUDING NEONATAL DEATHS)

	No.	%
Under 3 years	1257	49
3 – 20 years	275	11
21 – 40 years	456	17
41 – 60 years	413	16
Over 60 years	171	7
Total	2572	

What is the reason for this high death rate among children? The answer is simply nutritional deficiency and infection. The deaths cannot really be so neatly compartmentalized as we see in Table II – so many infants have gastroenteritis, pneumonia *and* kwashiorkor. Tuberculosis and amoebiasis are also common at this age as in later life.

TABLE II. RESULTS OF NECROPSIES ON CHILDREN UNDER 3 YEARS

Malnutrition		
Kwashiorkor	275	80%
Gastroenteritis	304	
Bronchopneumonia	438	
Infection		
Tuberculosis	63	12%
Amoebiasis	33	
Meningitis	51	

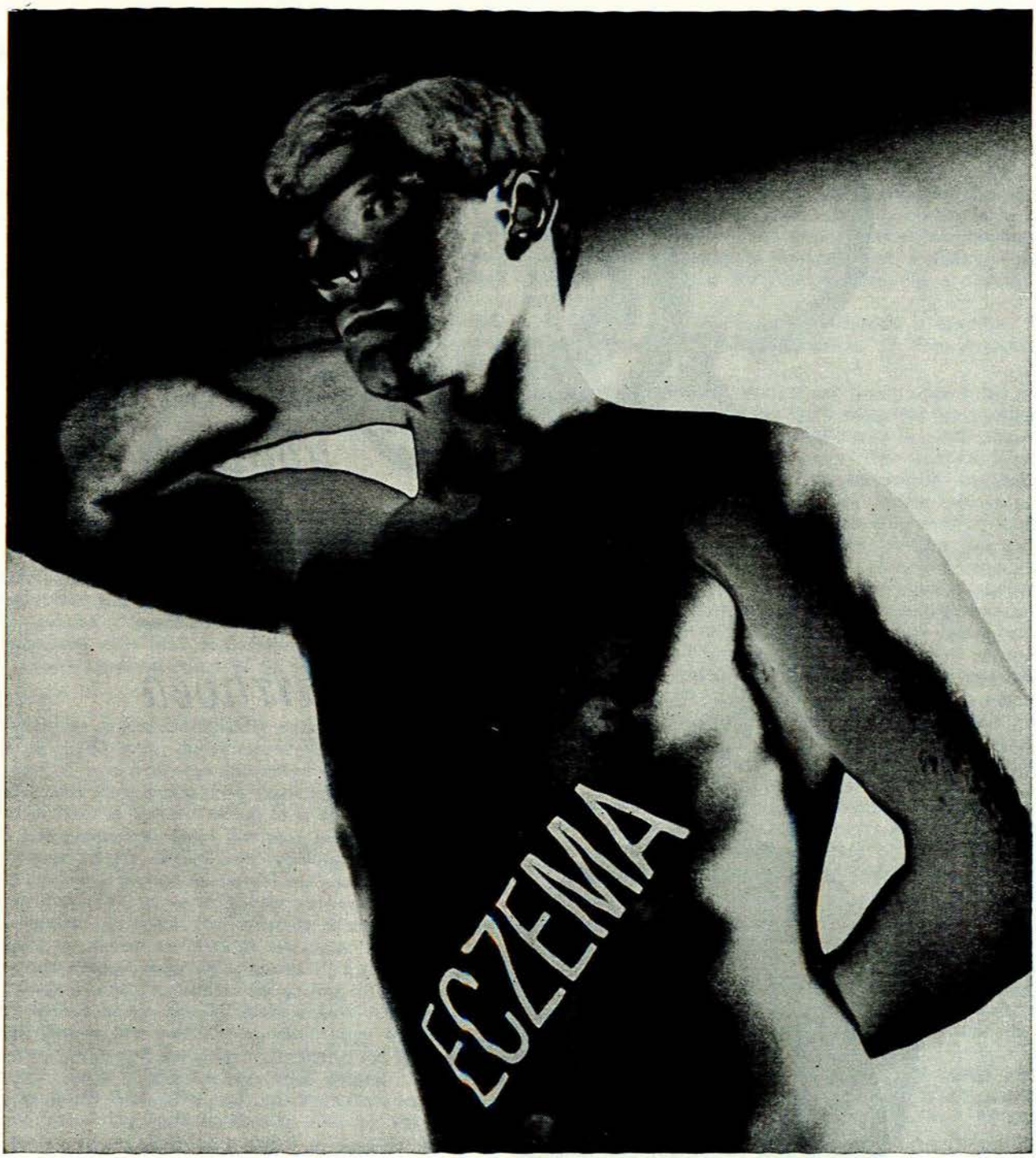
Kwashiorkor

I should like to say a few words about kwashiorkor. That name has reached the headlines the world over, and everyone knows that it is basically a protein-deficiency disease. Striking pathological features are:

Dermatosis. Here we find the normal melanin pigment migrating to a surface parakeratotic layer which is then exfoliated, leaving depigmented, erythematous and often ulcerated areas.

Fatty liver. This is the most striking feature at necropsy and the liver is of course a vital organ in metabolism. Hypoglycaemia and oedema owing to low plasma protein may be related to the liver disease. Perhaps the most surprising fact is that this enormous fat infiltration – the liver will actually float – can disappear on treatment without apparently leaving permanent scars. I say this because liver cirrhosis is uncommon in children.

Pancreatic atrophy. This explains the lack of digestive



■ *immediate symptomatic improvement* ■ *clinical manifestations cleared*
■ *less chance of complicating infections*

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enzymes and hence the failure of digestion and absorption of food.

Infections. Perhaps it is the low β-globulins – all plasma protein fractions are depressed – which leads to lowered resistance to infection.

Virus Infections

Measles often leads to a fatal staphylococcal pneumonia, but many deaths are due to the virus itself – giant cell pneumonia of infancy.

The virus of herpes simplex may become disseminated, causing a rapidly fatal disease, the characteristic feature being a focal necrosis of the liver, the cells showing intranuclear inclusions.

Fungal infections are rife. Monilia is well known but we see many cases of mucormycosis, a disease previously recorded only in diabetics.

NUTRITIONAL FACTORS

Nutritional factors dominate pathology of the adult Bantu. Many cases of pellagra are encountered – but I would warn you that malnutrition often masks other pathology, particularly glandular tuberculosis. Many cases of tuberculous adenitis involving the deep-seated nodes – para-aortic and iliac – cannot be palpated by the clinician, and biopsy of a superficial node may be negative. Siderosis is, however, the most widespread lesion of the adult Bantu.

Siderosis

Subsisting on a predominantly maize diet, but well fortified by kaffir-beer, the Bantu absorbs a phenomenal amount of iron from the alimentary tract. Some 80% of adult males show excess iron deposits in the tissues. The onset is later in females owing to iron loss in menstruation and pregnancy. There has been great controversy on the source of this dietary iron – cooking in iron pots has been reputed to fortify the natural iron content, but present evidence also points to kaffir-beer as having a very high iron content. I gather that there is a surprisingly high faecal iron content on Monday mornings.

Storage disease. Siderosis is essentially a storage disease. The body has no mechanism for getting rid of unwanted iron, so that, in the form of haemosiderin, it accumulates in the tissues. We see it at an early stage in the bone marrow, spleen and the liver parenchyma, the main storehouse for iron. When the liver cells become overloaded they probably disintegrate and the iron spills over into the portal tracts. This cell breakdown may be the natural senescence of the liver cells, but it is possible that iron accelerates cell necrosis. Should the liver become cirrhotic then it is incapable of coping with the enormous iron overload and haemosiderin begins to appear in other tissues – the pancreas, thyroid, muscle, etc.

How important is this iron overload? Bantu surviving to a ripe old age are often loaded with iron, so it does not invariably shorten the life span. I do not think we can attribute the abnormal protein pattern of the Bantu to siderosis, since a similar pattern is found in parts of Africa where siderosis does not occur.

On the other hand, after seeing some thousands of siderotic livers, I find it difficult to avoid the conclusion that the iron overload does cause hepatic fibrosis and that this may progress to the stage of diffuse pigmentary portal cirrhosis with its inevitable sequelae.

Furthermore, we are finding more and more Bantu with siderotic and fibrotic livers who develop porphyria. This is an acquired type of porphyria with light sensitivity; the patients showing skin vesication especially on the backs of the hands. These patients also show decreased carbohydrate tolerance – both conditions indicative of a failing liver. Another suggestion is that siderosis may be a factor in osteoporosis.

I think we can conclude that siderosis is harmful, its only blessing being the prevention of iron-deficiency anaemia.

Other types of liver disease are also common in the Bantu and the suggestion has been made that malnutrition may make the liver more susceptible to hepatotoxins. It certainly appears sensitive to alcohol! Post-alcoholic hypoglycaemic coma is another common occurrence – especially after the weekend – and may be fatal unless intravenous dextrose is given. These patients show a definite focal hepatitis with transient abnormality of liver-function tests.

Severe liver necrosis occurs more commonly than in other racial groups. There are two main types – 'massive', where large areas of liver tissue undergo cytolytic necrosis, and 'zonal', a diffuse lesion involving every lobule uniformly. A common assumption is that 'massive' necrosis is viral in origin, but until we are able to isolate the virus of infective hepatitis the aetiology must remain in doubt.

However, we see far more cases of 'zonal' necrosis; we have recognized over 100 deaths from zonal necrosis in the past 5 years as compared to half that number from massive necrosis. These patients usually give a history of abdominal pain, vomiting and often develop hypoglycaemic coma with convulsions. This picture occurs at all ages and the symptoms are often superimposed on diverse pathological conditions, making a difficult clinical problem.

Herbal remedies. We are convinced that this zonal necrosis is due to herbal remedies supplied by inyangas, taken either orally or as enemas. Death may ensue within 24 - 48 hours from hypoglycaemic coma – the liver lesion being only just recognizable. Others survive several days, develop jaundice and die from liver failure. A third group survive 7 - 10 days to die of renal failure owing to acute tubular necrosis.

I am certain this is common throughout Natal and many necropsied cases are certified as dying from cardiac failure because the liver shows a nutmeg pattern. At one time we thought the plant *Adenia gumniferia* – known to the Zulu as imfulwa and used as an emetic – was one of the causes of zonal necrosis, but our efforts at extraction failed.

We hear that aflatoxin, produced by the fungus *Aspergillus flavus*, which may contaminate peanuts and maize in humid climates, causes zonal necrosis in animals. Is it possible that most of our liver necroses are toxic rather than viral in origin? There is much investigation needed on this point.

Doubtless, severe liver necrosis – particularly massive necrosis – leads to a form of cirrhosis (post-necrotic scarring). This I believe is the other major type of cirrhosis in the Bantu. The close relationship of cirrhosis to liver

cancer – hepatoma – is well known, and this tumour is one of the main types of cancer found in the African male.

So far we have seen the black side of the traditional Bantu diet. It may have one saving grace: the low incidence of atheroma and its complications, particularly myocardial infarction (Table III).

TABLE III. INCIDENCE OF MYOCARDIAL INFARCTION

Age group	*Bantu*		*Indian*	
	Male	*Female*	*Male*	*Female*
40–60	0·3%	0·2%	14·3%	1·0%
60–80	0·0%	0·0%	16·0%	15·4%

In contrast to the rarity of myocardial infarction, cerebrovascular accidents are common (Table IV), but these appear to be hypertensive in origin. Whether the hypertension is related to the very common focal pyelonephritis we see at necropsy is an open question.

TABLE IV. INCIDENCE OF CEREBROVASCULAR ACCIDENTS

Age group	*Bantu*		*Indian*	
	Male	*Female*	*Male*	*Female*
40–60	8·7%	11·5%	18·0%	13·0%
60–80	14·1%	24·0%	25·0%	23·1%

CARDIOVASCULAR SYSTEM

The Bantu cardiovascular system may not be entirely immune to diet. We have the intriguing and often disputed condition of cardiomyopathy. Recurrent episodes of congestive heart failure in the absence of gross hypertension and valvular disease mark the course of this condition. At necropsy the heart is grossly enlarged and may show subendocardial fibrosis and mural thrombus formation. Embolic lesions are common. Some would attribute the primary lesion to the endocardium, others to the muscle fibres: could it be the result of infection? Or is it possibly dietary – a high intake of serotin has been suggested, or a deficiency of tryptophane, both of which can result in experimental cardiac lesions. Unfortunately the pathologist must make a humble apology and acknowledge that there seem to be no specific histological features by which he can unquestionably recognize this disease. One of my staff is attempting enzyme studies in the hope of finding some distinguishable feature.

The scope of this conference adequately covers infective processes and all I would emphasize here is the regional variation in diseases – in Natal amoebiasis is a major problem (Table V).

TABLE V. NECROPSIES OVER 10 YEARS

Tuberculosis	7%
Amoebiasis	6%
Pneumonia	5%
Pyelonephritis	3%

CANCER

Before concluding I should like to say a few words about cancer in the Bantu (Table VI). Its incidence will doubtless increase as malnutrition and infection are eliminated. We see some unusual tumours – Kaposi's sarcoma, Burkitt lymphoma, the malignant hepatoma, to mention a few – but by far the commonest cancer is carcinoma of the cervix and, as with so much pathology in the Bantu, the disease is usually at an advanced stage when first diagnosed. Frequent pregnancies and abortions, genital tract infection, carcinogenic action of smegma from the uncircumcised male, have all been advanced as possible explanations. With our present lack of knowledge of the aetiology, exfoliative cytology and treatment of the carcinoma-*in-situ* stage of the disease are the only methods of control. This is clearly impossible at this stage in the Bantu.

TABLE VI. NECROPSIES OVER 10 YEARS

Infection	26%
Trauma	20%
Cardiovascular	28%
Neoplasia	*14%*

In the male we have seen the startling increase in cancer of the oesophagus in many areas of the country. Once again we still have to find the cause, and palliative therapy is all that can be offered because of the advanced stage of the disease. A few years ago I thought bronchial carcinoma was uncommon in the Bantu, but recent figures show this as being one of the commoner tumours (Table VII).

TABLE VII. PREDOMINANT TYPES OF CANCER

Male		*Female*	
Oesophagus	26%	Cervix	42%
Liver	15%	Stomach	9%
Lymphoma and leukaemia	13%	Breast	6%
Bronchus	12%		

CONCLUSION

In conclusion, pathological studies indicate that preventive measures in the Bantu must be directed at:

1. *Nutrition,* to eliminate kwashiorkor and siderosis.
2. *Public health measures,* to control infection.
3. *Education,* to persuade the sick to seek early medical treatment but also to avoid the iatrogenic diseases of the inyanga.