

CLINICAL OBSERVATIONS ON CANCER PATTERNS AT THE NON-WHITE HOSPITAL. BARAGWANATH, JOHANNESBURG, 1948 - 1964*

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The fundamental work undertaken by Higginson and Oettle¹ on the cancer incidence in the Bantu and Cape Coloured races covered the years 1953 - 1955. In addition the work carried out by the late Dr Oettle² in subsequent years has laid the foundations of our knowledge of the cancer incidence in the Bantu of Southern Africa. In 1965 it was decided to extend the scope of the Bantu survey and analyse the total admissions to the Baragwanath non-White Hospital from June 1948 until December 1964.

The object of this publication is to stress certain clinical observations on the distribution of cancer in the non-White patient and some changes in cancer incidence occurring at this hospital during the past 17 years.

Situated on the outskirts of Johannesburg, Baragwanath Hospital was transformed from a military to a civilian non-White hospital in June 1948. Approximately 800 beds were available when the hospital opened, and the number of beds has increased steadily to the stage where, in 1967, more than 2,100 beds were in constant use. In December 1967, after excluding medical, orthopaedic, paediatric and maternity beds, it was found that there were approximately 700 adult surgical beds, with an equal sex distribution.

The population served is the civilian population of Johannesburg excluding the Reef mines which have their own medical services. There are in addition 3 other small non-White hospitals in the Johannesburg area. The Baragwanath Hospital has several highly specialized departments and some patients are referred from hospitals of the Reef towns situated within 70 miles of Johannesburg, and, in addition, some patients are transferred from outlying hospitals of the Transvaal as rural admissions. In this series 88.7% of patients were from the Johannesburg area, 8.2% from local Reef towns, 2.3% were rural admissions and 0.8% were unspecified. The local population served is approximately 600,000, but cases are referred from all the southern Transvaal hospitals, including mission hospitals, thus increasing the service for some specialized treatments to possibly 3½ million.

The total number of admissions included maternity, paediatric, gynaecological, medical and surgical cases, as well as readmissions. These admissions have doubled in under 14 years. The 3 categories of patients consisted of 95.0% Bantu; 4.0% Coloured; 0.7% Asiatics and 0.3% 'other' (not specified).

METHOD OF SURVEY

A preliminary sample analysis was undertaken of total admissions to the wards of all cases for two separate months in 1964, as a guide to the variety of admissions.

Table I analyses the total admission of cases from Johannesburg, the Reef towns and the rural areas for 2 sample months in 1964, as a basis for comparison. Distribution in hospital by sex, age and category of complaint has been noted.

However, owing to the growth of population served,

enlargement of hospital accommodation and variables of accidents and other reasons, it was decided to relate the sites and types of cancer to the percentage of total annual cancer admissions in order to give comparable annual figures.

TABLE I. ADMISSIONS TO BARAGWANATH HOSPITAL: ANALYSIS BY MONTHLY SAMPLE

	March 1964			October 1964			% of admissions
	Gen.	Mat.	Total	Gen.	Mat.	Total	
<i>Domicile—including</i>							
<i>maternity</i>							
Johannesburg	2,682	1,131	3,813	2,838	1,265	4,103	88.7
Johannesburg area	321	45	366	325	44	369	8.2
Transvaal							
country towns	108	2	110	93	2	95	2.3
Other	27	4	31	37	—	37	0.8
Total admissions for month	3,138	1,182	4,320	3,293	1,311	4,604	
Male	1,801			1,980			
Female	1,337			1,313			
<i>Age—excluding</i>							
<i>maternity (yrs)</i>							
0-4	327			299			
5-9	107			112			
10-14	125			102			
15-19	196			258			
20-24	389			375			
25-29	400			391			
30-34	375			390			
35-39	286			293			
40-44	229			280			
45-49	222			222			
50-54	150			172			
55-59	66			99			
60-64	89			105			
65-69	49			56			
70+	85			108			
<i>Race—including</i>							
<i>maternity</i>							
Bantu	2,985	1,113	4,098	3,165	1,212	4,377	95.0
Asiatic	30	5	35	20	9	29	0.7
Coloured	113	62	175	98	86	184	4.0
Other + unspecified	10	2	12	10	4	14	0.3
<i>Ward</i>							
Medical	909			1,045			
Surgical	1,367			1,499			
Gynaecological	426			337			
Paediatric	436			412			
Maternity	1,182			1,311			

The survey itself included the examination of the filing system at the registry of Baragwanath Hospital and the extraction of all relevant bed letters since 1948. With the assistance of the special filing system of the registry, it was possible to eliminate maternity and other non-relevant material from the inspection. The cancer cases were extracted, and history details after classification were

*Date received: 19 November 1968.

correlated with other earlier surveys including one by Higginson and Oettle¹ in 1948-1953, and a further 1953-1955 survey by Oettle² used for analysing specific sites, types, rates and ratios of the various racial groups. This extraction was continued for all hospital records until the end of 1964. A précis was made of each case and then completed by the addition of the pathological reports, by perusal of the records at the South African Institute for Medical Research from 1948 onwards, and from the Baragwanath Branch established in 1954. These included the special departments of neuropathology and clinical pathology and postmortem examination reports. All pathology records were examined and included if relevant. Finally, all cases were correlated to exclude dual recording in different years and were subdivided under the code numbers of the International Classification of Diseases for each of the 17 years.³

TABLE II. TOTAL ADMISSIONS TO BARAGWANATH HOSPITAL 1948-1964

Year	Total admissions	Total No. of cancers		Cancer as % of			
		Total cancers	Male	Female	Total admissions	Male	Female
1948	Not available	83	44	39	—	53.0	47.0
1949	Not available	185	85	100	—	45.9	54.1
1950	Not available	233	119	114	—	51.1	48.9
1951*	26,110	240	129	111	0.923	53.7	46.3
1952	29,530	331	182	149	1.120	55.0	45.0
1953	29,620	392	195	197	1.323	49.7	50.3
1954	33,470	353	161	192	1.054	45.6	54.4
1955	37,616	348	168	180	0.925	48.3	51.7
1956	37,756	420	205	215	1.112	48.8	51.2
1957	39,671	481	254	227	1.212	52.8	47.2
1958	41,607	491	249	242	1.180	50.7	49.3
1959	47,678	561	283	278	1.176	50.4	49.6
1960	46,842	594	305	289	1.268	51.3	48.7
1961	48,599	678	364	314	1.395	53.7	46.3
1962	52,974	744	403	341	1.404	54.2	45.8
1963	51,936	793	439	354	1.526	55.4	44.6
1964	55,612	890	508	382	1.600	57.1	42.9
	579,021	7,817	4,093	3,724	1.263	52.4	47.6

* International Code started in 1951. Total admissions include maternity, paediatrics, ophthalmic, medical and surgical male and female.

Table II indicates the total number of admissions to the hospital, the total cancer cases and the proportions of males and females of these cancer cases expressed as percentages of total and of cancer admissions. This table also shows a steady increase of cancer admissions compared with admissions as a whole, with an average of 1.263/100. The over-all gradual increase in male and female admission over the past 8 years should be noted, but may possibly be accounted for by the increase in cases of oesophageal cancer, and the provision of specialist services. During the period 1957-1964 the percentage of male cases consistently exceeded that of females. This too may be accounted for by the steady increase in cases of oesophageal cancer in males.

There were 7,817 cases registered as first admissions, consisting of 4,093 males and 3,724 females, subsequent admissions being recorded with the first as one cancer case. Missing or inadequate bed letters, mainly in the earlier years, were mostly traced through pathology records, but full clinical details were in these cases occasionally incomplete. The diagnosis was proved histologically in 84% of cases and in the remaining 16% the diagnosis was made on clinical evidence. Many of the latter category were very advanced, moribund and clinically obvious, and not fit for any radical treatment. The records at the clinical laboratory were unavailable for leukaemias, and final correlation of these with bed letters was not possible, so that these cases were slightly underestimated.

Intra-epithelial carcinoma of the skin and conjunctiva, carcinoma *in situ* in the oesophagus and cervix, and other conditions that are known to be precancerous were excluded, on the grounds that they were not invasive. These transitional cases, between acceptance and rejection, would seem to warrant greater histochemical study as a link between cancer and normality. There were 13 cases which had dual cancers and one case with triple primary cancer pathology.

The sources of information, checks from other surveys, the pathology records and other corroborative data indicated that the survey was sufficiently complete for adequate analysis and that very few cases had been unaccounted for.

Table III shows the incidence of multiple primary cancer

TABLE III. MULTIPLE PRIMARY CANCERS BY SEX, SITE, AGE AND TRIBE BY YEAR

Year	Sex	Site I	Site II	Age (yrs)	Tribe
1950	M	Oesophagus: mod. diff. sq. Ca.	Stomach: adenocarcinoma	38	Nyasa
1952	Albino				
	M	Ear: well-diff. sq. Ca.	Lip: mod. diff. sq. Ca.	30	Zulu
1953	M	Gum: spindle-cell sarcoma	Abdominal wall: angiosarcoma	70	Sotho
1953	M	Cervical lymph gland: meta. sq. Ca.	Bladder: proved transitional-cell Ca.	52	Sotho
1954	F	Primary oesophagus Dermato-fibrosarcoma	(2) Skin: metastatic adenocarcinoma. (3) Also neurofibrosarcoma of abdominal wall	58	Xhosa
1956	F	Cervix: grade II sq. Ca.	Vulva: condylomata with areas of carcinomatous degeneration	44	Sotho
1957	M	Bladder: poorly diff. sq. Ca.	Haemangiosarcoma of skin	65	Xhosa
1959	F	Hysterectomy: cervix sq. Ca.	Ovary: cystadenocarcinoma	50	Swazi
1960	F	Stomach (linitis plastica)	Breast	74	Sotho
1960	M	Stomach: colloidal Ca.	Liver: malig. hep.	44	Zulu
1960	F	Cervix: adenocarcinoma	Breast: some adenocarcinoma, some colloidal Ca.	56	Coloured
1961	F	Corpus: adenocarcinoma	Ovary: cystadenocarcinoma	41	Tswana
1964	M	Malignant lymphoma	Kaposi's sarcoma	80	Rolong

TABLE V. TOTAL CANCERS BY SEX, 1948-1964

Total	Code No.	Male	% of total	Female	% of total
Buccal cavity and pharynx	140-148	349	4.5	69	.9
Oesophagus	150	831	10.6	108	1.4
Stomach	151	171	2.2	79	1.0
Small and large intestine and rectum	152/3/4	101	1.3	84	1.1
Liver	155	468	6.0	85	1.1
Liver, pancreas and unspec. digestive organs	156/7, 8 & 9	142	1.8	77	1.0
Nasal sinuses	160	121	1.5	47	.6
Larynx	161	87	1.1	3	.04
Lung	162	320	4.1	42	.5
Lung, unspecified, mediastinum and thoracic secondaries	163/4 & 5	18	.2	7	.08
Kidney	180	51	.7	50	.6
Bladder	181	121	1.5	28	.4
Skin	190 & 191	159	2.0	170	2.2
Eye, brain and central nervous system	192 & 193	150	1.9	105	1.3
Thyroid and other endocrine glands	194 & 195	20	.3	71	.9
Bone	196	52	.7	41	.5
Connective tissue	197	122	1.6	57	.7
Secondary glands and unspec. sites	198 & 199	130	1.7	91	1.2
Lymphosarcoma and reticulosarcoma	200	65	.8	49	.6
Hodgkin's	201	69	.9	24	.3
Lymphoma	202	37	.5	22	.3
Multiple myeloma	203	49	.6	27	.3
Leukaemia	204	91	1.2	52	.7
Mycosis fungoides	205	1	.01	3	.04
		3,725		1,391	
<i>Genital organs—hormone-dependent</i>					
Breast	170	24	.3	170	5.1
Prostate	177	230	2.9	171	20.8
Testis	178	18	.2	172	.9
Malignant neoplasm and unspec. male genital organs	179	96	1.2	173 & 174	.6
				175	1.5
				176	.9
		368		2,333	
Total		4,093	52.4	3,724	47.6
Grand total		7,817			

TABLE VIA. TOTAL CANCERS FOR 1960 EXPRESSED AS INCIDENCE (PER 100,000) OF POPULATION AT RISK IN TRANSVAAL BETWEEN AGES 25 AND 69 YEARS

	Population at risk	
	Males	Females
Total population	1,067,573	801,216
Total cases	236	231
Incidence	22.1	28.8
Cases (excluding breast and genital)	220	65
Incidence	20.6	8.1
Cases breast and genital	16	166
Incidence	1.5	20.7

Table VII shows a series of random sites by age and sex—oesophagus, stomach, lung and bronchus, larynx (with an almost complete lack of involvement in females) and breast. All these showed the maximum number of cases in the 45-54-year age-group, with slight minor variations, in both males and females. This age frequency is supported by reference to hospital admissions (Table I), as there is no commensurate increase in admissions at the ages indicated. This age factor also applied to connective tissue and the haematopoietic and lymphatic cancers.

TABLE VIB. CANCER AS SEX INCIDENCE PER 100,000 AT DIFFERENT SITES AT RISK IN TRANSVAAL EXPRESSED AS AN ANNUAL AVERAGE OVER 17 YEARS

Site	ICD Code No.	Male	Female
Oesophagus	150	4.2	.68
Stomach	151	.90	.49
Liver	155	2.34	.57
Larynx	161	.45	.01
Lung	162	1.67	.27
Skin	191	.55	.54
Thyroid	194	.07	.31
Connective tissue	197	.54	.27
Lymphatic and haematopoietic	200-204	1.19	.80
Breast	170	—	3.8
Cervix	171	—	16.02

Thyroid cancer showed an unequal sex distribution, but in the reverse ratio, and also the increasing number of male cases in the older age-groups. This would suggest a possible hormonal association.

'Cervical' cancer showed a greater incidence over a wider age range as the maximum numbers were in the 35-54-year age-group.

Table VIII demonstrates that the maximum number of

TABLE VII. RANDOM CANCERS AT DIFFERENT SITES BY AGE, 1960-1964

ICD Code No.	Male									Female								
	15-19	20-24	25-34	35-44	45-54	55-64	65-74	75+	N/S	15-19	20-24	25-34	35-44	45-54	55-64	65-74	75+	N/S
Oesophagus 150	—	2	40	166	291	181	101	46	5	—	3	7	24	37	11	15	12	—
Stomach 151	—	—	19	33	58	33	22	6	2	—	—	8	15	18	14	13	8	—
Large intestine 153	2	—	2	13	14	7	4	3	1	1	—	2	7	7	7	5	1	—
Rectum 154	—	2	7	8	12	11	6	7	—	—	5	4	14	11	6	6	4	—
Liver 155	12	17	87	134	111	63	30	16	5	1	2	10	15	19	22	12	3	2
Pancreas 157	—	—	5	5	18	17	8	4	—	—	—	2	4	8	8	6	6	—
Larynx 161	—	—	3	17	35	21	8	3	—	—	—	2	1	—	—	—	—	—
Bronchus 162	—	2	9	53	120	78	44	13	3	2	—	9	9	7	8	5	2	—
Breast 170	—	1	—	3	5	5	4	1	—	—	7	41	102	113	65	43	24	—
Cervix 171	—	—	—	—	—	—	—	—	—	—	12	242	455	444	263	140	56	10
Melanoma 190	1	2	5	2	11	11	5	1	1	1	1	5	9	11	16	22	8	—
Skin 191	4	1	25	25	22	19	10	2	2	9	9	14	18	14	14	14	9	—
Brain 193	5	6	17	20	19	3	3	2	1	4	8	11	4	11	3	—	2	1
Thyroid 194	—	—	—	2	3	6	2	—	—	2	5	8	7	10	13	5	6	3
Bone 196	8	7	11	7	9	—	—	—	—	8	4	8	4	5	3	—	1	—
Connective tissue 197	3	4	19	25	29	14	12	3	2	2	5	16	8	6	5	2	2	—
Lymphatic + haematopoietic 200-204	14	21	48	77	39	35	18	11	4	13	11	23	25	29	20	12	6	3
	49	65	297	590	796	504	278	121	27	43	72	412	721	750	478	300	150	19

TABLE VIII. PRIMARY LIVER CANCER (155) BY SEX AND AGE

Year	Male								Female							
	15-19	20-24	25-34	35-44	45-54	55-64	65-74	75+	15-19	20-24	25-34	35-44	45-54	55-64	65-74	75+
1948	—	—	1	1	—	—	—	—	—	—	—	—	—	—	—	—
1949	1	1	3	3	—	1	1	—	—	—	—	—	1	—	—	—
1950	—	1	4	3	2	—	—	—	—	—	1	—	—	—	—	—
1951	2	—	1	4	2	1	1	—	—	—	—	—	1	—	—	—
1952	—	—	3	9	7	3	1	1	—	—	—	—	—	—	—	—
1953	—	1	7	9	2	1	1	—	1	1	—	1	—	—	—	—
1954	—	3	3	10	6	3	—	1	—	—	1	—	—	3	2	—
1955	2	—	3	6	7	2	2	—	—	—	1	1	—	—	1	—
1956	2	1	6	7	5	6	3	2	—	—	1	1	1	—	—	—
1957	—	3	5	11	8	7	4	1	—	—	1	3	3	1	2	—
1958	—	—	3	9	8	8	2	2	—	—	2	4	4	1	1	—
1959	2	3	8	16	4	4	2	2	—	—	—	—	—	6	—	1
1960	—	1	5	14	9	5	3	2	—	—	1	1	1	—	—	—
1961	—	—	10	5	7	7	—	—	1	—	2	1	2	1	1	—
1962	2	1	9	7	12	3	3	—	—	—	2	1	2	2	1	2
1963	—	1	6	15	14	5	3	4	—	—	1	4	—	2	2	—
1964	1	1	10	8	18	7	4	1	—	—	—	—	4	6	2	—
	12	17	87	134	111	63	30	16	1	2	10	15	19	22	12	3

Age of five males and two females unspecified.

TABLE IX. CARCINOMA OF LIVER (155) ARRANGED BY HISTOLOGICALLY PROVED CASES ONLY

Year	Hepatoma	Carcinoma gallbladder	Cholangiocarcinoma
1960	26	4	—
1961	30	3	—
1962	30	3	—
1963	35	3	1
1964	35	9	1

TABLE X. OCCURRENCE OF CARCINOMA OF LIVER (155) BY MONTHLY INCIDENCE TO INDICATE ANY POSSIBLE SEASONAL INFLUENCE (DUE TO STORAGE OF FOOD, ETC.) AS BY MONTH OF FIRST SYMPTOM

Year	Male												Female														
	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sep.	Oct.	Nov.	Dec.	N/K	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sep.	Oct.	Nov.	Dec.	N/K	
1960	1	1	1	4	4	3	1	4	3	1	3	2	10	—	—	1	—	—	—	—	1	—	—	—	—	—	2
1961	1	2	1	1	3	2	—	3	2	3	—	4	7	1	—	—	—	2	2	1	—	—	2	—	—	1	
1962	2	5	3	5	—	—	4	2	3	4	1	2	6	1	1	—	—	—	—	1	2	—	—	—	2	2	
1963	2	2	8	3	1	1	3	2	2	7	2	5	11	3	1	1	1	—	—	—	—	2	—	—	—	1	
1964	4	1	7	5	5	1	4	3	2	2	2	4	12	1	1	2	3	—	—	1	—	2	1	—	—	1	
	10	11	20	18	13	7	12	14	12	17	8	17	46	6	1	5	4	1	2	3	3	6	1	2	4	5	

TABLE XI. LIVER CANCER SURVIVAL RATE FROM DATE OF FIRST SIGN TO DEATH, 1952-1964

Length of survival from first symptom	1952	1953	1954	1955	1956	1957	1958	1959	1960	1961	1962	1963	1964	Total
Males														
0-3 months	10	13	17	12	16	18	10	10	19	13	19	17	18	192
3-6 months	7	4	2	3	5	4	7	6	3	5	4	7	7	64
6-9 months	1		2		2	2	1	2		1	1	1	2	15
9-12 months	1							1		1				3
12-15 months			1	1	1		1		1	1	1	1	1	9
15 months-2 years								1		1				2
Death date unknown	4	4	5	4	7	13	10	15	16	10	11	16	22	137
Females														
0-3 months		1	3	2				3	1		2	4	6	22
3-6 months			1		1	1	2	1	1	2	1		3	13
6-9 months													1	1
9-12 months														
12-15 months														
15 months-2 years														
Death date unknown			3	1		4	7	1	1	2	3	4	1	27

TABLE XII. CASES OF CANCER OF LIVER AND OESOPHAGUS BY AGE AND SEX INDICATING SURVIVAL RATE FROM FIRST SYMPTOM TO DEATH FOR 1960-1964

Age in years	Duration of survival (months)													
	0-3		3-6		6-9		9-12		12-18		18-24		>24	
	M	F	M	F	M	F	M	F	M	F	M	F	M	F
Liver														
15-19	1													
20-24	3													
25-29	5		1	2		1								
30-34	9	2	4						1					
35-39	6	2	4			1	1		1					
40-44	8	3	5											
45-49	11	2	4		3	1								
50-54	10		1			1			1					
55-59	4	4	3				1		1					
60-64	6	3	1	2		1			1					
65-69		1	1	1										
70+	8	4	4						1					
Oesophagus														
20-24	1	2												
25-29	1		2			1								1
30-34	2	1	5		5	2			1				1	
35-39	12	3	12	2	3						1			
40-44	16	1	12	2	5	1	4		2					
45-49	18	4	15	2	6	1	3						3	
50-54	27	1	13		7		6			2			1	
55-59	8	1	13		5		1		1				1	
60-64	12	3	19		8	1	2	1			1			
65-69	15	1	7	1	3				2	1			1	1
70+	17	1	7	6	1		1		1	1			1	

TABLE XIII. RANDOM TABLE OF SURVIVAL TIMES AT VARIOUS SITES FROM FIRST SYMPTOM OR SIGN TO DATE OF DEATH (1952-1964)

Length of survival to death	Male					Female				
	Oesophagus	Pancreas	Ca. lung	Haemato-poietic and lymphatic	Oesophagus	Pancreas	Ca. lung	Cervix	Haemato-poietic and lymphatic	
0-3 months	165	20	45	52	23	10	10	88	44	
3-6 months	146	10	34	23	14	4	7	85	13	
6-9 months	74	1	20	17	9	2	1	63	4	
9-12 months	32	—	10	6	3	1	1	34	—	
12-15 months	14	—	11	4	3	—	—	44	3	
15-18 months	—	—	—	—	1	—	—	13	—	
1½-2 years	6	—	3	2	2	1	3	14	—	
>2 years	18	—	11	—	3	—	—	17	—	
>3 years	—	—	—	2	—	—	—	8	3	
>4 years	—	—	—	—	—	—	—	3	—	
>5 years	—	—	—	—	—	—	—	8	—	
Details not complete	342	19	153	175	43	16	15	1,073	86	

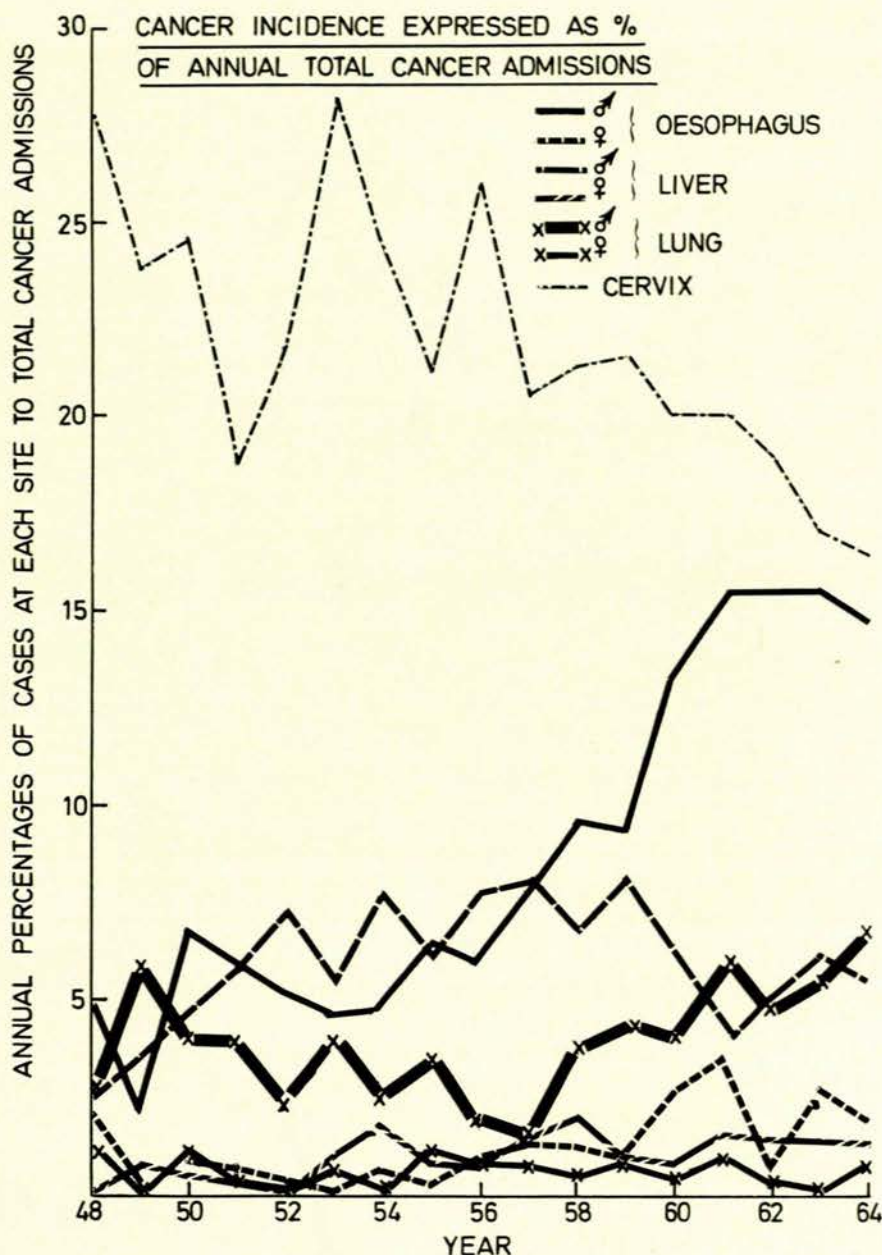


Fig. 1. Cancer incidence expressed as a percentage of the total annual cancer admissions.

cases of liver cancer occurred in a somewhat earlier age-group in males than in females. It indicates the increasing susceptibility in males with a possible association with liver dysfunction and/or cirrhosis. This host difference could be connected with hormone imbalance and alterations in liver metabolism.

Table IX indicates the histological type of liver cancer found in the years 1960-1964. Frequent pathological reports were 'postnecrotic cirrhosis of liver with hepatocellular carcinoma'. This would seem to indicate the recovery of a necrotic liver from an earlier stage of damage, either

by an external toxin or by a carcinogen, and suggests probable damage to a cell or actual mutant cell formation.

Table X shows that cancer of the liver was not related to a possible seasonal exhibition of carcinogens as has been suggested.¹

Table XI shows the survival rate in malignant hepatoma from the first sign or presenting symptom, and not the date of hospital admission. The majority of patients were dead within 6 months of the first symptoms.

It would appear that there is a sudden diminution in host resistance and that once unrestrained carcinogenesis

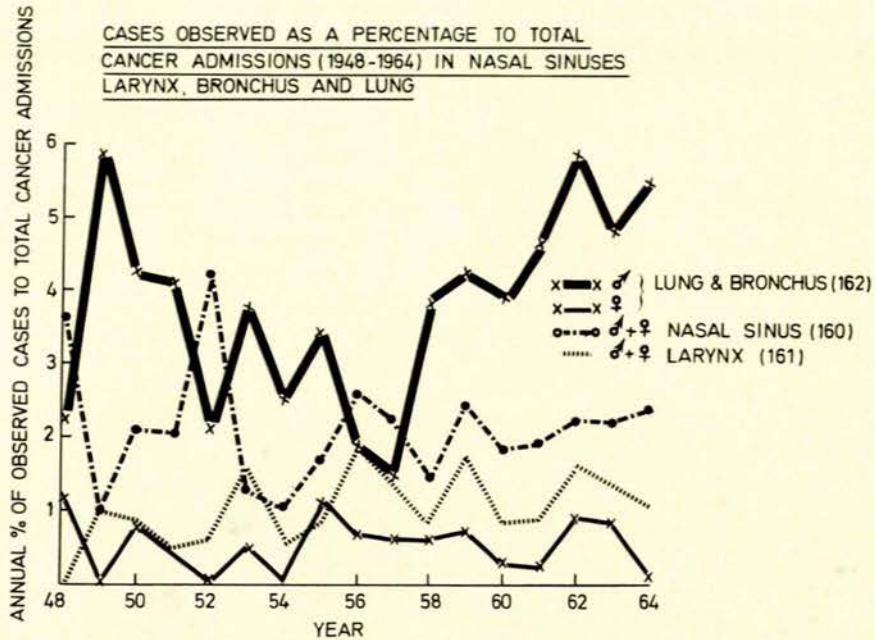


Fig. 2 (a). Incidence of cancer of the nasal sinuses, larynx, bronchus and lung expressed as a percentage of the total cancer admissions.

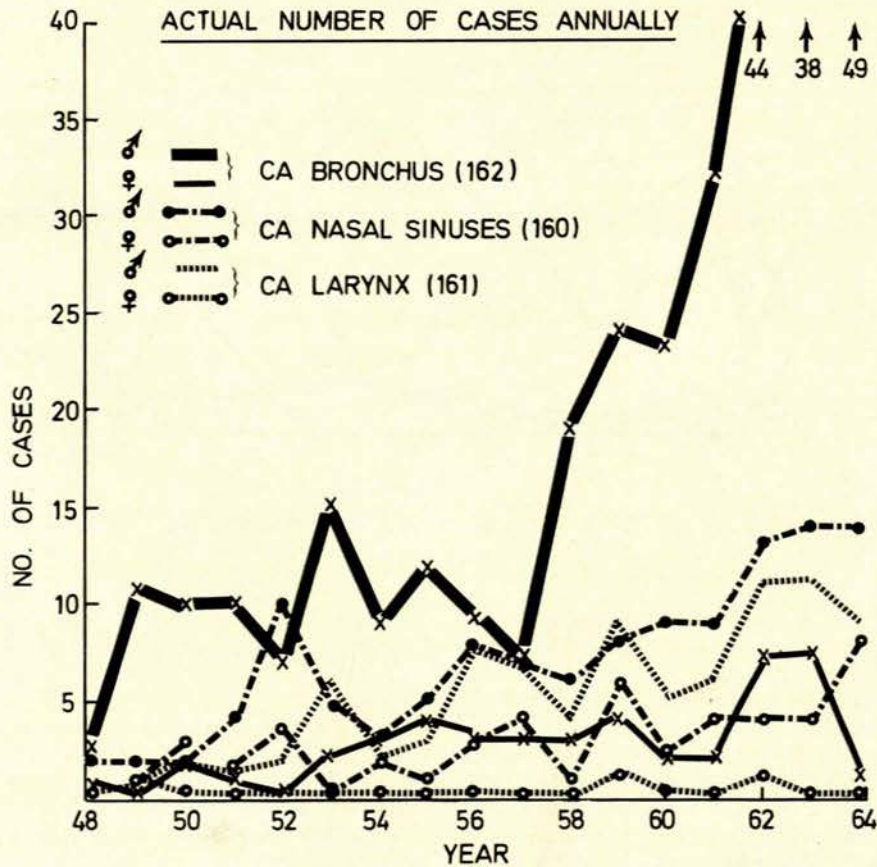


Fig. 2(b). Actual number of cases observed annually.

% OF OBSERVED CASES TO TOTAL CANCER ADMISSIONS
(1948-1964) FOR OESOPHAGUS, BUCCAL CAVITY, PHARYNX
AND STOMACH

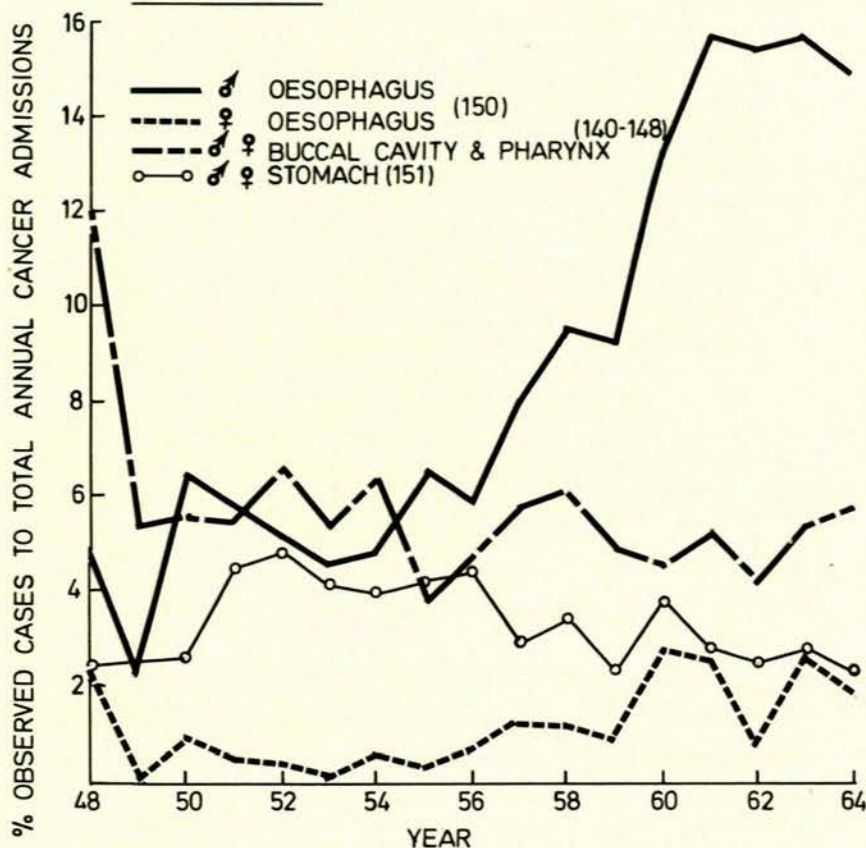


Fig. 3(a). Incidence of cancer of the oesophagus, buccal cavity, pharynx and stomach expressed as a percentage of the total cancer admissions.

sets in, the disease runs a rapid course.

Table XII gives the survival rates for liver and oesophageal cancers for the years 1960-1964. The rate of growth of the tumours did not appear to be related to the age or sex of the patient.

Table XIII gives the survival rates for a series of random sites and suggests that once the host resistance is overcome, very rapid growth of the cancer takes place. The high number of cases with 'details or date of death unknown' are mainly due to the preference of the majority of patients (if fit enough) to return to relatives in remote areas of the country, where follow-up study is obviously impracticable. In fact, this practice is encouraged after palliative treatment has been completed. These findings appear to indicate the trend to rapid development once host control is released.

A recent article giving details of a 99% 'follow-up' of nearly 400 cases of carcinoma of the oesophagus showed that only 2 patients had survived 2 years—one of whom died in the 25th month—and only 10 more than 1 year. In fact, 90% were dead within 6 months of admission.⁴

Relationship of Cancers to Population

According to the 1960 population census 42.95% of

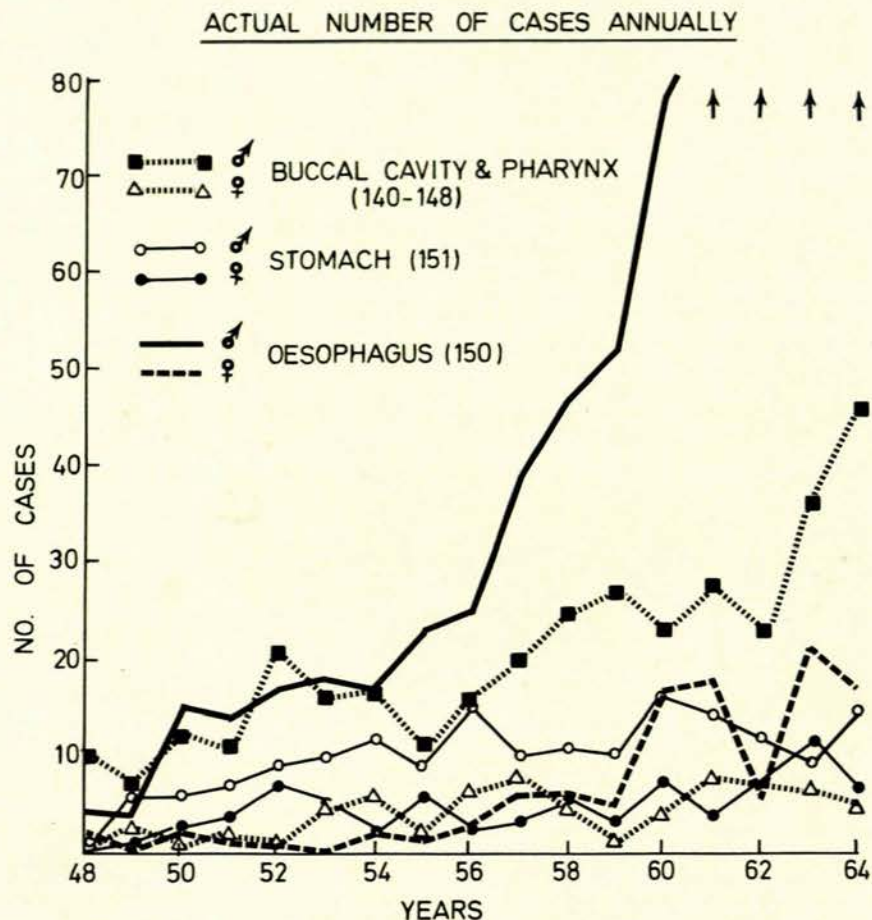
males and 37.41% of females were between the ages of 25 and 69 years, and yet 79.18% of cancers occurred in this age-group. The populations at risk under the age of 25 years and over the age of 70 years were 57.05% for males and 62.59% for females, and the cancer incidence in these groups was 20.82%.

This shows clearly that the predominating number of cancer cases occur between 25 and 69 years, and this is evident in the cancer admissions (most dying before 70 years). The increasing numbers are also emphasized by the smaller population at risk (between 25 and 69 years) although cancer continues to play a major role in the diminishing population over 70 years of age.

Observations on Figures

The figures shown below emphasize the trends indicated in the tables. Figs. 1-5 compare the rates and actual numbers of cases annually for various sites—showing sex differences.

Fig. 1 represents cancer admissions expressed as a percentage of total annual cancer admissions. This is really a facet of Table IV which is condensed to show the variations of cancers of the oesophagus, cervix, lung, bronchus and liver in males and females. It will be noted



that the relative percentage of cancer of the cervix declined and that there was a steep rise in male cancer of the oesophagus, whereas the female oesophagus cancer rate remained relatively stable. The percentages of lung and bronchial cancers showed a slight increase in males, but none in females. The percentage of primary liver cancer seems to have passed the peak, but has been unaccompanied by a commensurate rise in the female percentages.

Fig. 2 is a representation of the annual cancer rates occurring in cancer of the nasal sinuses, cancer of the larynx and cancer of the lung and bronchus in males and females. It suggests that simple inhalation of carcinogens will not explain the differences, unless the susceptible tissues in the nasal passages, larynx, bronchus and lung react differently to the external carcinogens to which they are exposed; for the differences are too marked to be coincidental. In addition the sex differences in each site, particularly lung and bronchus, would suggest differing host susceptibility or resistance rather than differences in exposure to carcinogens.

Fig. 3 shows a similar pattern, suggesting that ingestion of carcinogens must involve differences in tissue susceptibility, for the buccal cavity, pharynx, oesophagus and stomach are not similarly affected. It would also appear

that, apart from differences at the site exposed, there is a difference between male and female host involvement in cancer of the oesophagus.

Fig. 4 indicates great differences in incidence between malignancies of the cervix uteri, corpus uteri and ovary, suggesting an environmental agent in the case of the cervix differing from that of the corpus uteri and ovary.

Fig. 5 shows the sex differences in cancer of the liver and biliary passages. This suggests differences in host and tissue susceptibility, especially as in Table VIII it appears to occur in different age-groups.

GENERAL OBSERVATIONS

It must be made clear that these statistics and figures apply to a non-White hospital in Johannesburg and have no direct relevance to the Johannesburg White population. One obvious feature seems to be the marked difference between the sexes, and the preponderant number of cases at most sites which occur in the 45-54-year age-group together with the rapidly fatal termination from onset of symptoms, even though the exhibition of carcinogens is unlikely to be at uniform ages or doses. This could involve the removal of host restraint, enabling the latent

or dormant damaged or mutant cancer cell or cells to develop.

These observations on a Bantu hospital population confirm that host resistance varies with sex. Carcinogens appear to have some degree of specificity for certain tissues. Malignancies appear in greater numbers in certain age-groups and a large proportion of clinical cancers show rapid unrestricted growth followed by the patient's death, not directly related in set time or dose to carcinogenic exhibition, suggesting a release of control of latent mutant cells.

The adaptation of incidence rates of cases from a single hospital is, of course, not possible because all cancers are not admitted to one hospital, but population numbers can still be compared at different ages as they apply to the ages of hospital admissions and the Transvaal population, making them comparable as confirmed in Table XIV. They are on different standards to incidence although in proportion. They are expressed as one in 10,000 to draw attention to the fact that they are not 'incidence'.

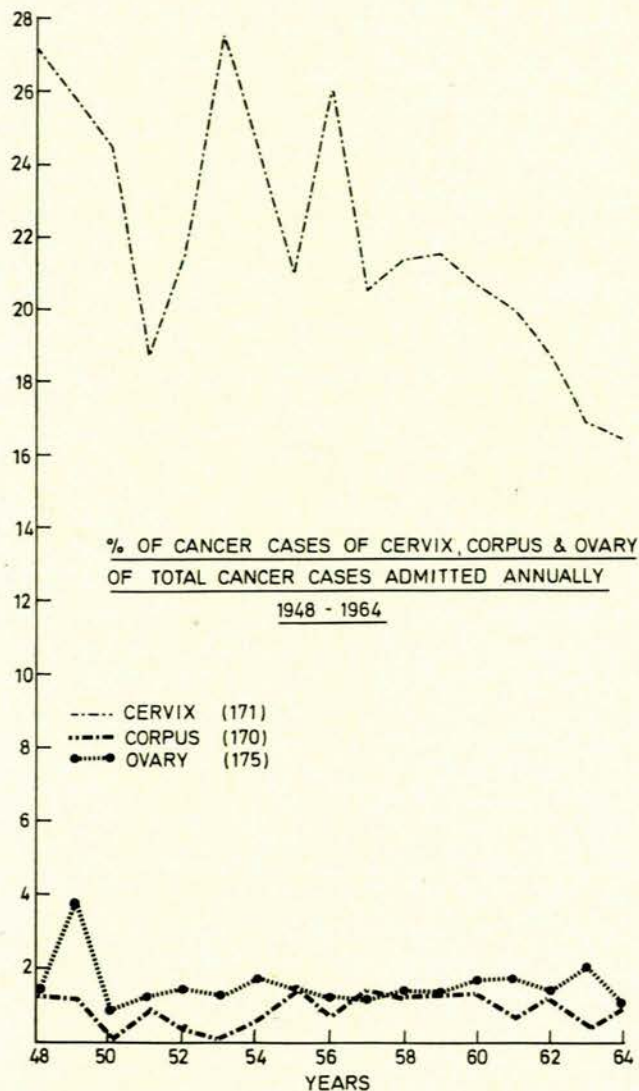


Fig. 4(a). Incidence of cancer of the cervix, corpus uteri and ovary expressed as a percentage of the total cancer admissions.

Relationship to Transvaal Population

Further observations relating to the Transvaal population at risk are shown in the following tables and figures, using years 1960 - 1964 as examples. Some interesting facts are apparent.

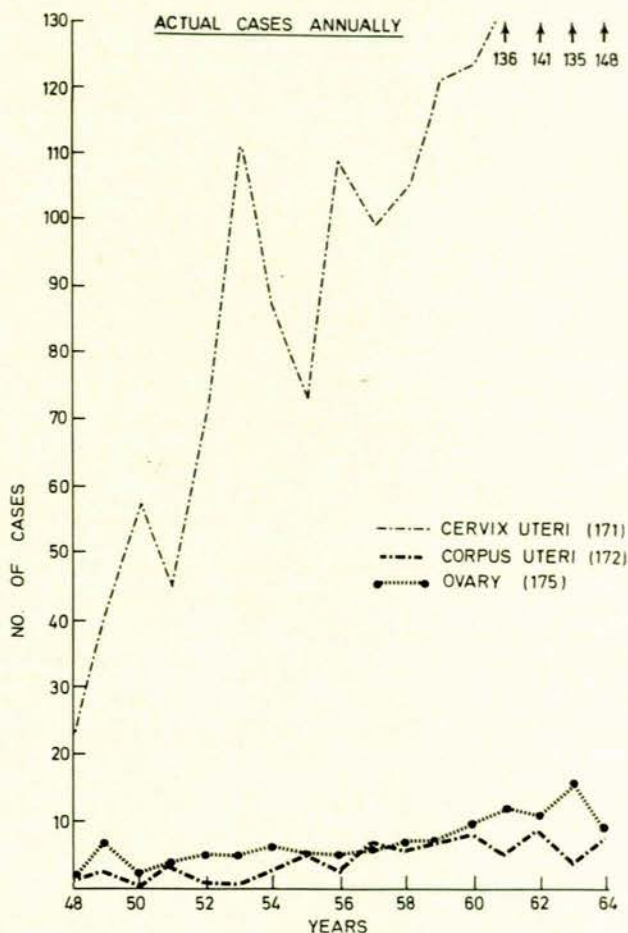


Fig. 4(b). Actual number of cases observed annually.

Table XIV and Fig. 6 give the population of the Transvaal by 5-year age-groups for the ages 25 - 69 years, with sex differences, based on the figures of the 1960 census. This represents the steep fall of a population with a shorter life-span than the White population.

Table XV and Fig. 7 indicate that the cancer rate in the Transvaal per 10,000 has a gradual increase up to 50 years of age, diminishes during the next few years and thereafter again increases rapidly in the following ageing population up to 60 years.

Table XVI and Fig. 8 show a dip in the curve after ± 50 years of age when cases are expressed as a percentage of the total annual cancers and also the following

LIVER CANCER 1948-64 IN MALES & FEMALES EXPRESSED AS % LIVER CANCERS TO TOTAL CANCER ADMISSIONS ANNUALLY

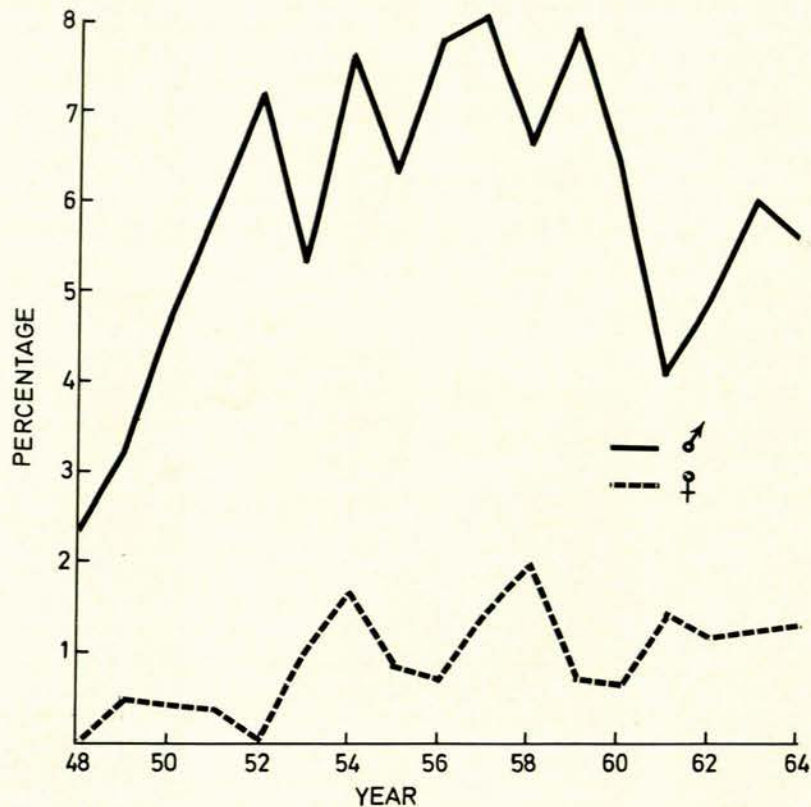


Fig. 5(a). Incidence of cancer of the liver expressed as a percentage of the total cancer admissions.

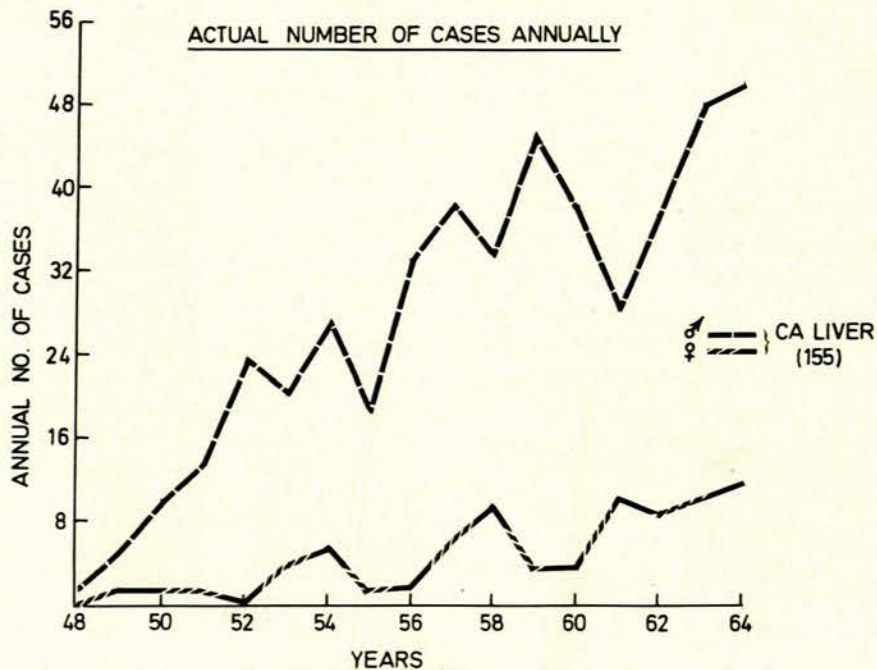


Fig. 5(b). Actual number of cases observed annually.

TABLE XIV. POPULATION CENSUS 1960 OF TRANSVAAL SHOWING PERCENTAGE MALE, FEMALE AND TOTAL IN AGE-GROUPS

	Age-groups (years)								
	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69
Males	9.51	7.80	7.02	5.96	4.89	3.14	2.20	1.44	0.92
Females	8.13	6.92	5.89	4.97	3.74	2.84	1.94	1.76	1.18
Males + females	8.87	7.40	6.50	5.50	4.36	3.01	2.08	1.59	1.04
Total 1960 hospital admissions as percentage by age	12.4	12.0	9.1	8.0	7.0	5.1	2.6	3.0	1.6

Males under 25 and over 70 years: 1,418,055 (57.05%). Males between 25 and 69 years: 1,067,573 (42.89%).
 Females under 25 and over 70 years: 1,340,591 (62.53%). Females between 25 and 69 years: 801,216 (37.37%).

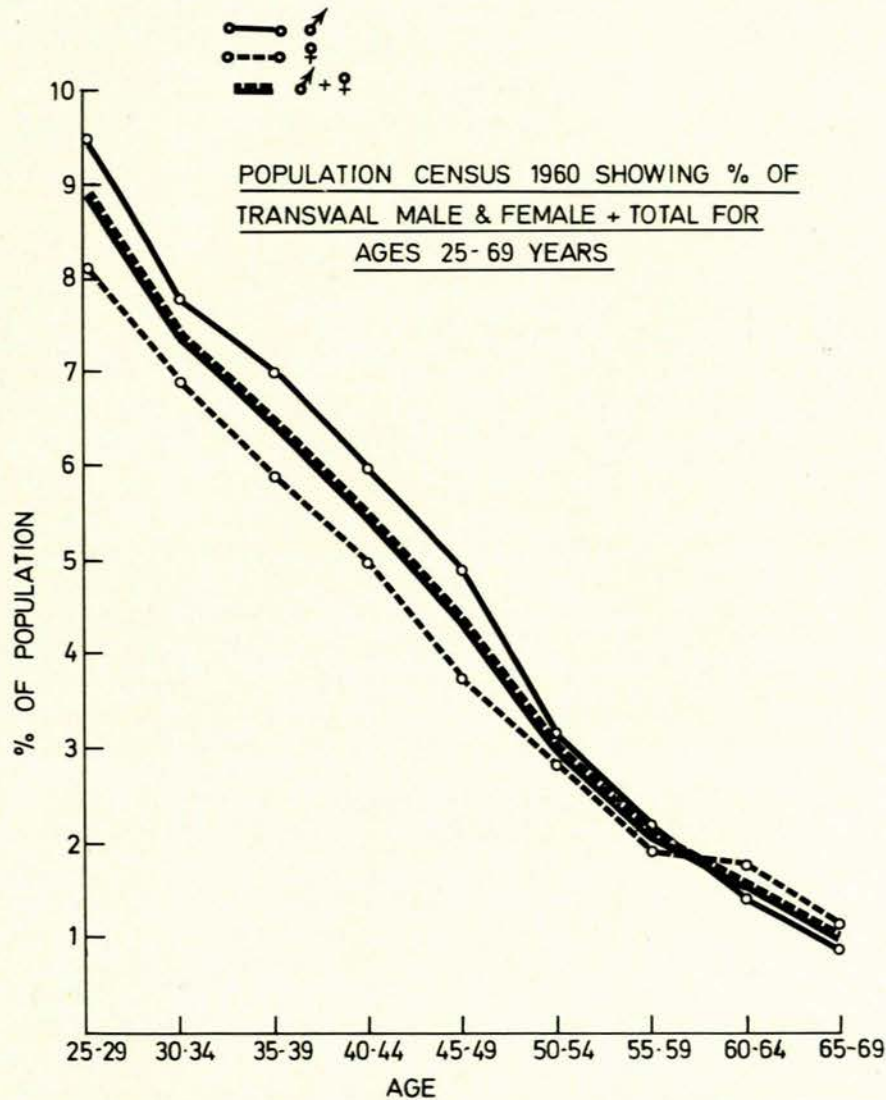


Fig. 6. Population of the Transvaal by 5-year age-groups for the ages 25 - 69 years, based on the figures of the 1960 census.

temporary rise in the diminishing admissions in the older age-groups.

Table XVII and Fig. 9 show the annual rate per 10,000 of the Transvaal population, with breast and genital organs expressed separately because of their very different behaviour.

These tables and figures show that there is an increasing susceptibility or diminishing host resistance to cancer and that maximum numbers occur at about 50 years, after which diminution of susceptibility (or increased resistance) in the next 5 years takes place, followed again by increasing susceptibility (or diminished host resistance) in the 60 - 65-

TABLE XV. INCIDENCE CANCER CASES TO POPULATION AT RISK IN TRANSVAAL BY AGE AND SEX GROUPS (1 : 10,000) (1960 CENSUS)

	Age-groups in years								
	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69
<i>Males</i>									
Yearly average Ca. cases (1960-1964)	9.6	21.0	28.2	46.8	54.0	57.6	32.6	44.4	26.8
Transvaal population by age-grouping (1960)	236,621	194,092	174,709	148,258	121,665	78,273	54,907	36,001	23,047
Per 10,000	0.405	1.08	1.61	3.15	4.43	7.35	5.93	12.34	11.62
<i>Females</i>									
Yearly average Ca. cases (1960-1964)	13.0	30.4	34.6	39.2	40.2	38.6	22.4	29.2	17.2
Transvaal population by age-grouping (1960)	174,283	148,383	126,174	106,405	80,105	61,017	41,650	37,734	25,465
Per 10,000	0.745	2.04	2.74	3.68	5.01	6.32	5.37	7.73	6.75

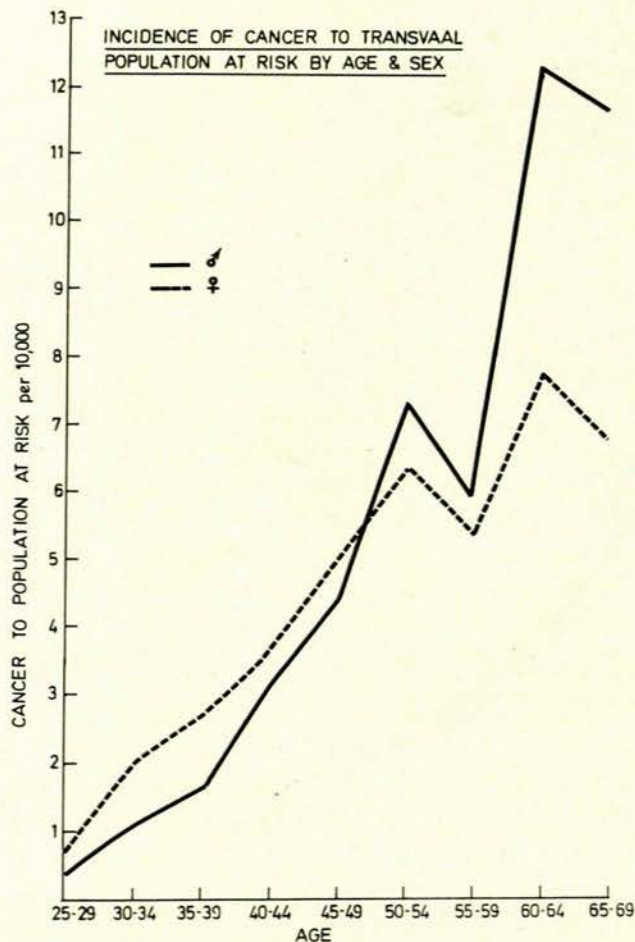


Fig. 7. Incidence of cancer to Transvaal population at risk based on the figures of the 1960 census.

year group and then a lower number of cases in the ageing population. It would seem to be that some effective check is applied temporarily on the cancer risk, possibly by hormonal imbalance. This temporary decrease in susceptibility would seem to be difficult to reconcile with the exposure and delayed onset of cancer involving a purely carcinogenic dose-time exposure theory.

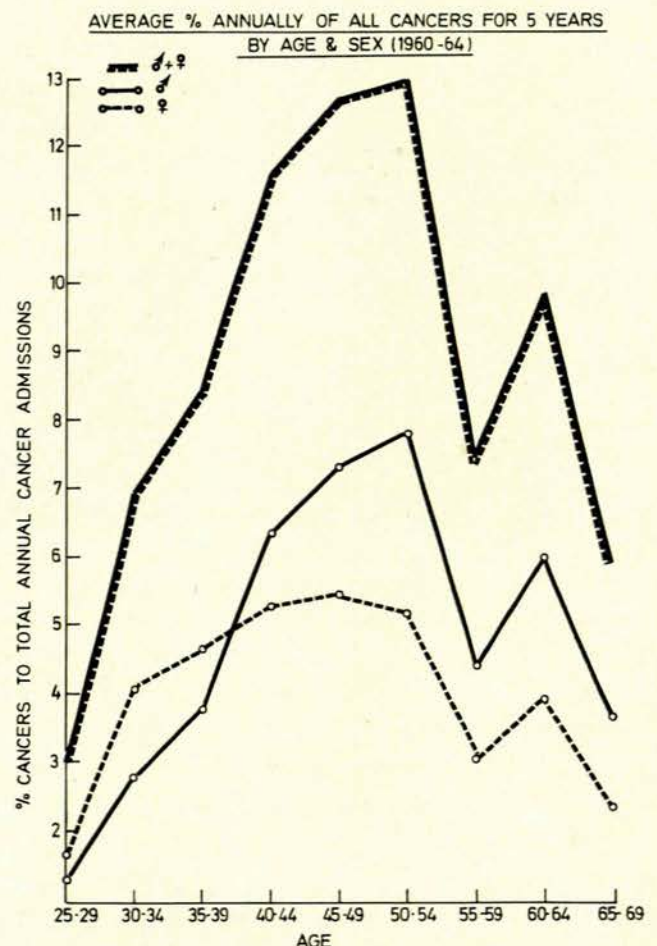


Fig. 8. Age incidence of all cancer cases observed at Baragwanath Hospital during the 5 years 1960-1964.

INTERPRETATION AND SUGGESTED HYPOTHESIS

Clinically the host-tumour relationship of cancer has continually attracted attention and has recently received increasing recognition.

The specificity of carcinogens to certain tissues is known and also their association with certain sites, but the delay in onset of cancer and apparent latency have been unexplained.

TABLE XVI. CASES EXPRESSED AS % OF TOTAL ANNUAL CANCER CASES AT BARAGWANATH HOSPITAL BY AGE-GROUPING (1960-1964)

	Age-group in years									Total for year
	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	
1960 number	20	43	59	59	67	64	55	62	38	594
% of total	3.3	7.2	9.9	9.9	11.2	10.7	9.2	10.4	6.3	
1961 number	18	36	56	82	84	90	61	65	39	678
% of total	2.6	5.3	8.2	12.0	12.3	13.2	8.9	9.5	5.7	
1962 number	25	60	47	81	105	112	49	81	37	744
% of total	3.3	8.0	6.3	10.8	14.1	15.0	6.5	10.8	4.9	
1963 number	23	61	69	98	103	110	45	73	47	793
% of total	2.9	7.6	8.7	12.3	12.9	13.8	5.6	9.2	5.9	
1964 number	27	57	83	110	112	105	65	87	59	890
% of total	3.0	6.4	9.3	12.3	12.5	11.7	7.3	9.7	6.6	
Total	113	257	314	430	471	481	275	368	220	3,699
	3.0	6.9	8.4	11.6	12.7	13.0	7.4	9.9	5.9	

20.8% of all cases (770) were under 25 years and over 70 years: 1960—127 cases (21.3%); 1961—147 cases (21.6%); 1962—147 cases (19.7%); 1963—164 cases (20.6%) and 1964—185 cases (20.7%).

TABLE XVII. AVERAGE ANNUAL CANCER RATE (PER 10,000) EXCLUDING BREAST AND GENITAL TO TRANSVAAL POPULATION AT RISK (1960-1964)

	Age-group in years								
	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69
<i>Male</i>									
Total 1960-1964	47	102	136	212	261	268	151	195	108
Average annual	9.4	20.4	27.2	42.4	52.2	53.6	30.2	39.0	21.6
Population at risk	236,621	194,092	174,709	148,258	121,665	78,273	54,907	36,001	23,047
Rate	0.397	1.05	1.55	2.85	4.29	6.84	5.50	10.83	9.37
<i>Female</i>									
Total 1960-1964	30	44	54	67	59	59	37	49	28
Average annual	6.0	8.8	10.8	13.4	11.8	11.8	7.4	9.8	5.6
Population at risk	174,283	148,383	126,174	106,405	80,105	61,017	41,650	37,734	25,465
Rate	0.344	0.593	0.855	1.25	1.47	1.93	1.77	2.59	2.19
<i>Male + female</i>									
Total 1960-1964	77	146	190	279	320	327	188	244	136
Average annual	15.4	29.2	38.0	55.8	64.0	65.4	37.6	48.8	27.2
Population at risk	410,904	342,475	300,883	254,663	201,770	139,290	96,557	73,735	48,512
Rate	0.374	0.852	1.26	2.19	3.17	4.69	3.89	6.62	5.60

TABLE XVIII. AVERAGE ANNUAL RATE (PER 10,000) BREAST AND GENITAL CANCERS BY AGE TO TRANSVAAL POPULATION AT RISK (1960-1964)

	Age-group in years								
	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69
<i>Male</i>									
Total 1960-1964	1	3	5	14	7	18	10	27	26
Average annual	0.20	0.60	1.0	2.8	1.4	3.6	2.0	5.4	5.2
Population at risk	236,621	194,092	174,709	148,258	121,665	78,273	54,907	36,001	23,047
Rate	0.0084	0.0309	0.0572	0.188	0.115	0.459	0.364	1.49	2.25
<i>Female</i>									
Total 1960-1964	35	108	119	140	144	126	77	96	58
Average annual	7.0	21.6	23.8	28.0	28.8	25.3	15.4	19.2	11.6
Population at risk	174,283	148,383	126,174	106,405	80,105	61,017	41,650	37,734	25,465
Rate	0.40	1.45	1.88	2.63	3.59	4.14	3.69	5.08	4.55
<i>Male + female</i>									
Total 1960-1964	36	111	124	154	151	144	87	123	84
Average annual	7.2	22.2	24.8	30.8	30.2	28.8	17.4	24.6	16.8
Population at risk	410,904	342,475	300,883	254,663	201,770	139,290	96,557	73,735	48,512
Rate	0.17	0.648	0.824	1.21	1.49	2.06	1.80	3.33	3.46

It has not yet been established whether the cell (or cells) exists in a damaged, vulnerable but still viable form, or whether it is already a mutant cell in a dormant state before malignant changes are established.

The suggestion, based on clinical observations, is tentatively put forward that this state might be linked, as indicated by sex differences and age incidence, to possible hormone imbalance, whereby a mutant or malignant

cell is released from restraint and, still capable of mitotic development and survival, develops (partially controlled, or uncontrolled) at a rapid rate. The occasional delay in the onset of secondaries is probably related to a relative increase of resistance following removal of the primary, and later, hormone imbalance releasing the latent cancer cell. This is indicative of a 'dormant' cancer cell, the revival process being superficially the opposite of the

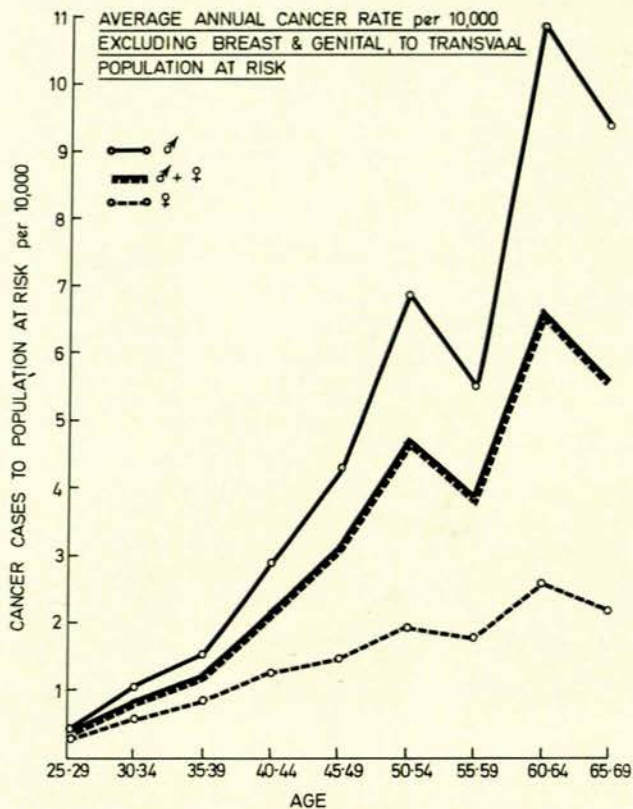


Fig. 9(a). Average annual incidence of cancer per 10,000 of the Transvaal population, excluding cancer of the breast and genital organs.

tissue rejection factor of transplantation.

It would appear that a genetic, or constitutional, component is involved with a deficiency or excess of the resistance factor so influencing the time of onset. This could account for the early or late cases, the non-development after exposure to carcinogens, the enzymal detoxification of carcinogens, perhaps even a carcinogen combining with, and so neutralizing, the resistance factor, and also rejection of cancer *in situ*, or many of the established racial differences—and other apparent anomalies. Genetic influence is apparent in retinoblastoma.

Another possible clinical example of this, observed in the cases, is that of renal tumours (Table XVIII) suggesting one genetic in origin, with early susceptibility, and the other later in onset, by loss of resistance.

Could this be of adrenal origin—with imbalance of hypo-, hyper- or abnormal secretion? The known influences on growth of cortisone, particularly in stimulating cancers and also in retarding the rejection of tissue transplants and impairing resistance to bacterial or viral invasion, as well as the temporary beneficial effects of adrenalectomy on metastases, are recognized effects.

Other hormone imbalances where cancer is involved with androgens and oestrogens in relationship to certain cancers (breast and prostate) are probably all interrelated with corticosteroid action.

SUMMARY

A survey of the clinical distribution of cancer cases at Baragwanath Hospital over 17 years was undertaken, showing cancer rates by site, sex and age. The difference in susceptibility of males and females at the similar sites has been noted as evidence of host resistance. The tissue susceptibility and the tissue specificity of external carcinogens for differing sites are confirmed.

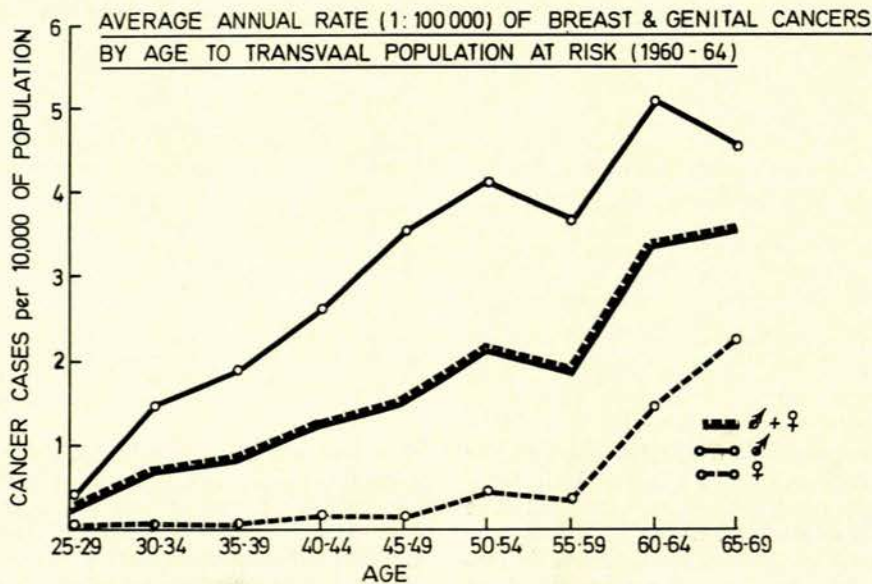


Fig. 9(b). Average annual incidence of cancer of the breast and genital organs per 10,000 of the Transvaal population.

TABLE XVIII. KIDNEY TUMOURS (180): WILM'S AND HYPERNEPHROMA BY AGE AND SEX

Year	Total No. of cases	Male		Female		Wilm's average age per annum	Hypernephroma average age per annum
		Wilm's	Hypernephroma adenocarcinoma + sarcoma	Wilm's	Hypernephroma adenocarcinoma + sarcoma		
1948	3	1	1	—	1	2 years	59 years
1949	3	—	1	1	1	4 months	59 years
1950	3	1	—	1	1	4 months	50 years
1951	1	—	1	—	—	—	35 years
1952	2	—	2	—	—	—	36 years
1953	5	—	2	1	2	2½ years	37 years
1954	3	—	1	2	—	8½ months	43 years
1955	5	2	2	1	—	3 years	44 years
1956	5	2	1	1	1	5½ years	34 years
1957	3	1	1	1	—	4 years	60 years
1958	6	2	1	1	2	7 years	65 years
1959	6	—	2	1	3	2 years	41 years
1960	8	—	4	2	2	9 months	66 years
1961	11	1	4	3	3	4 years	48 years
1962	6	—	2	2	2	1½ years	57 years
1963	9	—	4	2	3	4 years	41 years
1964	9	3	3	1	2	5 years	45 years
Total	88	13	32	20	23		

Other clinical observations are made on the increasing number of cases at certain ages, suggesting altering host resistance. This is followed on release of control by a relatively uniform rapid growth, suggesting that host-tumour relationship has been modified allowing a presumed dormant cell to develop uncontrollably.

The inference is that the host-tumour relationship is partially controlled by hormone imbalance.

I wish to thank Dr W. H. F. Kenny, Medical Superintendent of Baragwanath Hospital, for permission to undertake this survey, and Dr A. Schmanan for access to the pathology records. I am also indebted to the late Dr A. G. Oetlé who suggested this survey but unfortunately died in the early stages before having an opportunity to assemble the data—his analysis would indeed have been very valuable.

I should also like to thank Mr Haywood, of the registry at Baragwanath Hospital, for his help and adequate record keeping; Mr C. Mabaso for his assistance with extraction of the bed letters; and Mesdames D. Vickery, A. Woolford and J. Doody for their untiring assistance in transcribing and recording details from numerous sources.

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