

RELATIONSHIP BETWEEN PSYCHIATRIC DIAGNOSIS AND URINARY D.M.T.

Diagnosis	No of subjects	Age (yr, mean + s.d.)	No. with urinary D.M.T.
Schizophrenia	42	30.0 + 11.5	20 (48%)
Affective psychosis	30	45.3 + 14.7	4 (13%)
Other psychoses	18	28.0 + 13.4	7 (39%)
Neurosis and personality disorder	32	36.5 + 13.0	6 (19%)
Controls	20	32.5 + 11.6	1 (5%)

tients in whom D.M.T. was detected almost as frequently as in schizophrenics were the 18 with other non-affective psychoses.

We are aware of the pitfalls of this type of research.^{9 10} Since our patients had only lately been admitted when studied the differences found are unlikely to have been the result of long-term hospitalisation. All but 15 of our patients were taking psychotropic drugs, but the presence of D.M.T. was not related to medication. Although all were eating similar food, we did not study or control the diet. Tanimukai et al.¹¹ found D.M.T. in the urine of 4 schizophrenic patients whose diet excluded preformed indoleamines.

D.M.T. detectable by our method was seen more frequently in the urine of psychiatric patients than normal subjects and most often in the urine of psychotic patients. Nevertheless, the presence of detectable D.M.T. is not exclusively related to psychosis, since it was also found in the urine of people who had never been psychotic. Furthermore, patients with affective psychosis were no more likely to excrete measurable quantities of D.M.T. than those with neurotic reactions and personality disorders.

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URINARY-TRACT INFECTION IN CHILDREN

SIR,—Dr Saxena and his colleagues (Aug. 30, p. 403) attempt to justify early radiological investigation of children with urinary-tract infections (U.T.I.) by showing that a large proportion of children with apparent first infections have radiological abnormalities.

However, the benefits of routine investigation lie not in the number of abnormalities discovered, but in the usefulness of the information gained in guiding the future management of the patients. It has long been known, for example, that a large proportion of children with U.T.I. have reflux which disappears after the infection has cleared. Nothing is gained by discovering such an "abnormality," and much mischief can be done if discovery leads to unnecessary surgery.

None of the patients described by the authors were found to have conditions demanding immediate surgical correction. All of their patients should have been managed with appropriate antibiotic treatment until free of bacteriuria, and then followed with frequent urine cultures, with or without antibiotic prophylaxis. Any recurrence should have been treated similarly. There is nothing in the data presented to indicate that if such a treatment regimen had been followed and no radiological investigations made, any of the children would have been worse off. And if radiological investigation had been omitted entirely or restricted to those whose U.T.I. failed to clear or recurred, a great deal of radiation, cost, and inconvenience could have been saved, without any apparent detriment to patient welfare.

It may well be that some few children with first U.T.I. do have abnormalities which can only be discovered through

radiological investigation, the presence of which should modify initial management. If such is true, early radiological studies may be justified. But it is not justified by the data presented.

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ASTHMA AND CANCER MORTALITY

SIR,—Case-control studies, reviewed elsewhere,¹ have produced conflicting results regarding the relationship between cancer and previous allergic disorders. Alderson¹ reported that deaths from all cancers (excluding lung) were significantly reduced in asthma patients followed for 20–30 years, relative to expected numbers derived from a general population. Asthma patients were identified at age 25–60, so that inclusion of late-onset asthma not directly related to allergy was likely. The present study involves men reporting asthma as young adults, and should include "extrinsic" asthma with an allergic or immunological basis.

Between 1880 and about 1920 some 17 000 men at Harvard University were measured anthropometrically by the late D. A. Sargent and two assistants. All men were applicants for rental of a gymnasium locker. About 85% were "Old American" (all four grandparents born in the U.S.), largely of British ancestry, and another 10% were of immediate British or other Northern European ancestry. To ensure greater homogeneity, only those men were included who were born between 1850 and 1899, and who had spent two or more years at Harvard College. Also excluded were men who were lost to follow-up or known dead but lacking death certificates. This yielded a study

TABLE 1.—MORTALITY-RATES FOR SELECTED CAUSES.*

Causes of death (ICD codes, 7th revision) ²	Asthma (n=169)		Non-asthma (n=11 929)	
	No.	Rate per 100	No.	Rate per 100
Infections, parasites (001–138)	6	3.6	426	3.6
Neoplasms (140–239)	14	8.3	1060	8.9
Nervous system (330–398)	17	10.1	1139	9.5
Circulatory system (400–468)	50	29.6	3703	31.0
Respiratory system (470–527)	20	11.8	1049	8.8
Digestive system (530–587)	6	3.6	345	2.9
Genitourinary system (590–617)	0	0.0	230	1.9
Accidents, homicide, war (800–999)	10	5.9	745	6.2
Other causes	3	1.8	265	2.2
Total	126	74.6	8962	75.1

* Underlying cause of death from death certificates.

group of 12 098 men. As of June 30 1967, 9088 out of 12 098 men (75.1%) had died. Causes of death were obtained from death certificates coded according to the 7th Revision of the *International Classification of Diseases* (ICD)². "Malignant neoplasms" (ICD 140–205), "benign neoplasms" (ICD 210–229), and "neoplasms of unspecified nature" (ICD 230–239) were included in analyses of "neoplasms".

At the time of measurement each man was asked about his previous illnesses. The checklist included "asthma". Since the proportion of men reporting asthma was roughly similar across five birth-decade cohorts (1850–99), the cohorts were combined; 169 men reported asthma, or 1.4% of the total. Mortality-rates for the asthma and non-asthma cohorts for underlying cause of death are compared in table 1. The death-rate from neoplasms is slightly lower in the asthma cohort, but the difference is small (8.3 v. 8.9 per 100). The largest difference is for diseases of the respiratory system (11.8 v. 8.8 per 100); $z = 1.20$, $P > .20$). The overall death-rate is quite similar in

9 Wyatt, R. J., Termini, B. A., Davis, J. *Schizophrenia Bull.* 1971, **4**, 10
10 Rodnight, R. *Acta Neurol.* 1975, **30**, 84.
11 Tanimukai, H., Ginther, R., Spaide, J., Bueno, J. R., Himwich, H. E. *Br. J. Psychiat.* 1970, **117**, 421.

1. Alderson, M. *Lancet*, 1974, ii, 1475.
2. World Health Organisation *International Classification of Diseases*, 7th Revision. Geneva, 1957.

TABLE II.—MORTALITY FROM NEOPLASMS* BY SITE

ICD code (7th revision) ²	Asthma (n 169)		Non-asthma (n=11 929)	
	No.	Rate per 100	No.	Rate per 100
140–149 Buccal cavity, pharynx	1	0.6	47	0.4
150–159 Digestive	8	4.7	496	4.2
160–165 Respiratory	2	1.2	168	1.4
170–181 Genitourinary	2	1.2	302	2.5
190–199 Other, unspecified	1	0.6	120	1.0
200–205 Lymphatic and haematopoietic	3	1.8	144	1.2
210–229 Benign neoplasm	1	0.6	31	0.3
230–239 Unspecified neoplasm	1	0.6	19	0.2
Total	19	11.2	1327	11.1

*Underlying cause and contributory causes of death from death certificates.

the two groups (74.6% for asthma and 75.1% for non-asthma).

Mortality from neoplasms in selected sites is compared in the asthma and non-asthma cohorts in table II. When conditions mentioned on the death certificate are considered, no significant differences between the two cohorts are evident. The overall rates are nearly identical (i.e., 11.2 per 100 for asthma and 11.1 per 100 for non-asthma). Age-specific death-rates from neoplasms (underlying and contributory cause of death) were also compared for the asthma and non-asthma cohorts. Comparing rates for age 20–59, 60–69, 70–79, and 80+ (including only the birth decade cohorts, 1850–79, whose members had a chance to reach 80), there were no significant differences between the asthma and non-asthma cohorts.

The overall frequency of asthma in this series (1.4%) is not inconsistent with population estimates of 1–2%.³ The similar overall mortality in the asthma and non-asthma groups (table I) is perhaps not unexpected, since about 50% of children with asthma reportedly become symptom-free before adult life and only 5–10% continue to have severe disability.⁴ The slightly higher death-rate from respiratory disease in the asthma groups, if real, may represent the small proportion of childhood asthmatics with disability or sequelae. In contrast, Alderson's series¹ included patients over the age of 25 with a history of wheezing dyspnoea. Although patients with chronic bronchitis and obvious cardiovascular disease were excluded, mortality from respiratory disease and from all causes was higher in the asthma patients than in the general population.¹

The inaccuracies of death-certificate diagnoses are well known, but such data give a fairly accurate indication of the presence of malignant neoplasms at death as judged by comparison with necropsy data.⁵ The present findings do not support the hypothesis of a reduced risk of cancer in persons with a history of asthma. Mortality-rates from neoplasms were similar in the asthma and non-asthma cohorts.

"Extrinsic" asthma of childhood or young adulthood involves immunological changes; some asthmatic children show moderately or strikingly elevated serum levels of IgE.^{6,7} The present subjects, reporting asthma as young adults, may be considered to comprise largely "extrinsic" cases. Thus, the negative findings reported here do not appear to support the hypothesis of an allergic or immunological mechanism in attempting to explain Alderson's findings.¹ Further examination of the results of both studies also suggests that an immunological mechanism is unlikely. Primary immunodeficiency and immunosuppression predispose particularly to lymphatic-system malignancies and leukaemia;^{8,9} in adult

acute leukaemia immunocompetence and tumour-specific immunity are directly related to prognosis.^{3,4} Alderson,¹ however reported no reduction in mortality from leukaemia and Hodgkin's disease in asthmatic patients relative to expected figures; the ratio of observed to expected deaths in asthmatics was 1.09 for leukaemia and 2.56 for Hodgkin's disease. In the present series, albeit small, lymphatic and haematopoietic cancers were slightly more frequent in the asthma than in the non-asthma cohort (i.e., 1.8 v. 1.2 per 100, table II). If an immunological mechanism protected asthmatic patients from cancer, it would seem most likely to be evident in these tumours, but the findings are to the contrary.

In the present asthma cases, information on treatment was not obtained, but presumably many had recovered before adult life and received no treatment. Alderson¹ thought it unlikely that conventional treatment of asthma was involved in his findings. Madsen,¹⁰ and Wybran and Govaerts,¹¹ however, suggested that a pharmacological effect involving cyclic A.M.P. could be a factor. Further studies should consider the possible protective effect of bronchodilators on cancer susceptibility.

Part of this work was done while the author was research associate, Department of Nutrition, Harvard School of Public Health, Boston, Massachusetts.

The late Dr Albert Damon provided access to the records on the subjects of this study.

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SHORT PRACTICE OF CLINICAL PSYCHIATRY

SIR,—Your review of my book (Nov. 1, p. 853) was generous, but in parts misleading. Allow me to present what I wrote:

"Lithium is the drug of choice for hypomania but takes a few days to act. It should be started at the same time as tranquillizers" (p. 58).

"Barbiturates remain the most effective hypnotics, but they are addictive and potent suicide agents. In some patients barbiturates cause depression in others prolonged clouding . . . Used judiciously they add an increment of drowsiness to clouding and in practice produce refreshing sleep in some, but not all, patients" (p. 102).

The table you refer to as "most unfortunate" (p. 18) compares and contrasts endogenous with reactive depression. In no way does it suggest that leucotomy is indicated for reactive depression.

The book is a practical guide for medical students, psychiatrists, and general practitioners, not a mini-encyclopædia. I am sorry that a combination of tables, charts, endpapers, footnotes, photographs, and inflation makes it so expensive.

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*.*It is not established that lithium carbonate offers any advantages over major tranquillisers in the treatment of hypomania. To prescribe both lithium salts and tranquillisers simultaneously is unnecessary and may be dangerous if impaired renal function is not excluded. The more established use of lithium as a prophylactic drug in affective psychoses is not mentioned in Dr Barton's book. Dr Barton tells us six times that barbiturates are "the most successful hypnotics" and recommends them for sedation in depression, hypomania, delirium, puerperal psychoses, neuroses, and in the confused elderly patient. He mentions alternatives, but always as second-line treatments. The table on p. 18 states that 60% of

3. National Center for Health Statistics. Chronic Conditions and Activity Limitation: U. S., July 1961—June 1963. Series 1000, No. 17, Washington, D.C.
4. Howell, J. B. L. in Cecil Loeb Textbook of Medicine (edited by P. B. Beeson and W. McDermott); p. 885. Philadelphia, 1971.
5. Abramson, J. H., Sacks, M. I., Cahana, E. J. *chron. Dis.* 1971, **24**, 417
6. Johansson, S. G. O. *Lancet*, 1967, **ii**, 951.
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8. Kersey, J. H., Spector, B. D., Good, R. A. *Int. J. Cancer*, 1973, **12**, 333.
9. Shackelford, G. D., McAlister, W. H. *Am. J. Roentgen. rad. Ther. nucl. Med.* 1975, **123**, 144.

10. Madsen, S. N. *Lancet*, 1975, **i**, 166.
11. Wybran, J. Govaerts, A. *ibid*, 1975, **i**, 341.