Summary

Personal observation of a woman suffering from diabetes insipidus over a period of 26 years has enabled me to ascertain the complete adaptation of such a patient to a normal life when the lesion is not fatal. It has also enabled me to study one more case of uterine atony undoubtedly related to hypothalamic-posterior-pituitary insufficiency.

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NEPHRITIS IN TEXTILE WORKERS

BY

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On May 24, 1947, in a leading article in the *British Medical Journal* (p. 727) on the subject of oxygen poisoning, the statement is made that "the inhalation of textile dusts is a steady source of death from nephritis and a group of conditions whose common factor is hyperpiesia."

It is the purpose of this paper to examine the evidence on which this statement is based. The information is obtained from the Registrar-General's Decennial Supplement for 1931, which shows a considerably increased mortality due to nephritis in certain groups of textile workers. In this fascinating document the causes of death are compiled from death certificates in the years 1930-2. I do not happen to have a textbook of medicine of that date readily available, but the fifth edition of Price's *Medicine*, published in 1937, still describes chronic interstitial nephritis as an entity and does not mention malignant hypertension at all. We must therefore from the outset be on guard against the assumption that nephritis appearing on a death certificate in 1931 bears any relationship to nephritis as commonly understood among physicians in 1947. Fortunately we can investigate this initial difficulty fairly easily.

The age incidence of deaths from nephritis and similar causes in 161 cases of which I have personal records is shown in Table I. From this it is quite clear that the main

TABLE I.—Deaths from Renal Disease: Author's Cases

Age (years):	0-14	15-24	25-34	35-44	45-54	55-64	65-69	70+
Nephritis, type I (Ellis)	7	17	15	6	5	0	0	0
" type II (,,)	8	14	5	5	4	3	0	0
Renal dwarfism type ..	1	5	2	0	0	0	0	0
Polycystic kidney ..	0	0	1	0	1	0	0	0
Chronic pyelonephritis ..	0	3	4	10	1	1	1	1
Amyloid kidney ..	0	0	0	4	0	0	0	0
Pregnancy kidney ..	0	1	1	2	0	0	0	0
Diabetic kidney ..	0	0	1	1	3	5	0	0
Miscellaneous renal deaths (polyarteritis nodosa, renal Tb., hydronephrosis, etc.)	0	5	2	4	4	6	2	0
Total renal diseases ..	16	45	31	32	18	15	3	1
Malignant hypertension ..	0	0	0	12	25	8	0	0

The above figures do not include non-renal deaths from essential hypertension or deaths from prostatic obstruction.

incidence of death from nephritis is under the age of 45. Only when deaths from malignant hypertension are added does mortality become considerable at 45 to 55 years of age.

The Registrar-General's figures for the age incidence of death from nephritis are totally different, as shown in Table II, where it will be seen that the main mortality is

TABLE II.—Deaths from Nephritis (Registrar-General, 1930-2; Death Rate per 100,000)

Age (years):	16-24	25-34	35-44	45-54	55-64	65-69	70+
Males ..	15	9	18	49	108	219	389
Married women ..	11	9	18	40	86	160	239
Single women ..	15	9	17	36	74	128	247
Total ..	41	27	53	125	268	507	875

over the age of 45 and must largely consist of deaths from hypertension. It is my experience in consulting practice that many cases of hypertensive heart failure with albuminuria are diagnosed by their doctors as chronic interstitial nephritis. There is a slight difference in age distribution between the various social classes, in that the lowest class has a rather higher percentage of deaths in younger persons; these are probably deaths from true nephritis related to infections.

The mortality from so-called nephritis for the combined social classes III and IV (into which the textile workers fall) is shown in Table III, and in Table IV are listed the deaths from nephritis in those groups of textile workers in which the mortality from this complaint is said to be excessive.* It will be seen that the increased mortality

TABLE III.—Deaths from Nephritis in Social Classes III and IV combined (Registrar-General, 1931)

Age (years):	16-	20-	25-	35-	45-	55-	65-	75+
Total ..	166	272	573	858	1,940	3,134	4,070	2,417
Percentage	1.2	2.0	4.3	6.4	14.5	23.4	30.4	18.0

TABLE IV.—Textile Workers with Excessive Incidence of Deaths from Nephritis (Registrar-General, 1931)

Age (years):	16-	20-	25-	35-	45-	55-	65-	75+
Group 29 (textile spinners—cotton)	3	2	3	5	17	19	39	15
" 30 (textile spinners—wool worsted, etc.)	0	1	2	2	3	3	4	3
" 32 (textile weavers—wool worsted, etc.)	0	0	2	1	11	6	11	7
" 33 (textile strippers and grinders—cotton)	0	0	1	0	5	5	0	0
" 34 (textile dyers) ..	0	2	2	2	11	19	19	6
Totals ..	3	5	10	10	47	52	73	31
Percentage ..	1.3	2.2	4.3	4.3	20.3	22.5	31.6	13.4

occurs between 45 and 55, the age of essential hypertension, not that of nephritis.

If it is in fact hypertension which causes excessive mortality from "nephritis" in cotton workers one would expect an increased incidence of other effects of hypertensive disease. The standardized mortality ratio (S.M.R.) of cerebral vascular lesions, compared with that of nephritis for the occupational groups concerned, is therefore shown in Table V, and it is clear that mortality from cerebrovascular disease is about as much in excess of normal as that from so-called nephritis.

At this stage the impatient reader might remark that whatever name is given to the disease there is an increased

* Analysis of Tables III and IV by the χ^2 test shows $P > 0.9$. The differences in age incidence of nephritis in textile workers and in the population from which they come is therefore not significant.

TABLE V.—Standardized Mortality Ratio at Ages 20-65
(normal 100)

Group		From Nephritis	From Cerebral Vascular Lesions
29	Cotton spinners	135	151
	Wives	<i>136</i>	<i>131</i>
33	Cotton strippers and grinders	220	217
	Wives	<i>233</i>	<i>175</i>
34	Textile dyers	171	170
	Wives	<i>93</i>	<i>144</i>
174	Cotton blow-room workers	300	200
	Wives	<i>200</i>	<i>33</i>

The figures in italics are not statistically significant owing to the small numbers of certified deaths concerned.

mortality among certain trades from some condition certified by doctors in 1930-2 as nephritis, and it is not a matter of first importance what nomenclature is used to describe it. But there is a fundamental difference in aetiology between nephritis and essential hypertension in that the one is, so far as we know, largely the result of environmental factors (reaction to infection), whereas in the other the main and overriding factor in aetiology is heredity (Weitz, 1923; Hines, 1937-8; Platt, 1947). In other words, one would look first for environmental factors such as humidity, conditions of work, nutrition, and liability to infection if true nephritis were proved to be common in a certain group of work-people, but one would look first for evidence of inbreeding if one were investigating essential hypertension. This does not mean that the liability to death from essential hypertension cannot be influenced by environment; it is merely a matter of the most likely major influence at work, for in general it will save time and mistakes to examine that first. It will be seen from Table IV that the wool workers (groups 30 and 32) together contribute less than a quarter of the deaths from nephritis to the total for the three years in question, and we will therefore confine our remarks to the cotton industry.

Inbreeding, of course, is well known to occur in cotton towns, and to a large extent we are dealing with hereditary trades. If hereditary influences are at work in a disease like hypertension with a roughly even sex distribution we should expect the wives of workers in the affected groups to be affected. That this is the case is also shown in Table V, and it is interesting to contrast these figures with those for paperhangers and painters, in which the possibility of lead poisoning arises. In these trades the high S.M.R. for nephritis (131) and cerebrovascular lesions (151) in the men is not shown among their wives, for whom the figures are 86 and 82 respectively. Before accepting this very striking demonstration as proof against an industrial cause of nephritis in the textile trades, however, it must be admitted (as indeed it is noted by the Registrar-General) that many of the married women are also engaged in the cotton trade and so may be exposed to the same influences as their husbands.

If heredity is at work one might expect some increase of hypertensive disease in the surrounding population. This is in fact the case, for the S.M.R. in the region North IV (Lancashire and Cheshire) in 1931 was 122 for nephritis and 119 for cerebrovascular lesions (normal 100). These are considerable increases, having regard to the size and heterogeneity of the population.

The percentage of persons suffering from any hereditary disease in a population will tend to remain stationary under conditions of random mating, so that communities relatively isolated and inbred may for a long while show a tendency to suffer more from a particular inherited trait than the population at large.

A difficulty arises over occupational group 31 (textile weavers—cotton), in which the mortality from nephritis

is not excessive (S.M.R., 84). There are two reasons for not accepting this discrepancy as weighing heavily in favour of an occupational factor, however. The first is that the figure of 84 is not statistically significant, the numbers of deaths being too small; and the second is that the mortality from nephritis in the 1921-3 report was actually a high one—i.e., the cotton weavers in previous years suffered the same mortality from so-called nephritis as their workmates in other branches of the trade. The mortality for wives in this group is unfortunately not stated.

In Table VI an estimate of deaths from degenerative cardiovascular diseases in various occupational groups has been made by combining deaths (at ages 35 to 70) shown as cerebrovascular lesions, myocarditis, angina pectoris, and nephritis. It is seen that group 70 (clergy) actually have the highest incidence because of a likelihood of dying from angina pectoris. The second column of figures shows the deaths from cardiovascular degenerative diseases omitting angina. This table merely illustrates how occupational groups differ with regard to degenerative diseases in general. If these figures do not prove that angina among clergymen is due to the inhalation of incense they also fail to prove that nephritis in cotton workers is due to the inhalation of textile dusts.

TABLE VI.—Percentages of Occupational Groups dying of Degenerative Cardiovascular Disease at Ages 35 to 70

	Deaths	The Same Without Angina
Group 31: Textile weavers—cotton (no abnormal mortality from nephritis; S.M.R., 94)	29.1%	27.1%
„ 34: Textile dyers (nephritis S.M.R., 171; total S.M.R., 123)	30.9%	28.7%
„ 35: Makers of textile goods, etc. (mortality generally normal; S.M.R., 93)	26.6%	22.6%
„ 70: Anglican clergy (S.M.R., 69)	35.4%	25.5%
„ 81: Draughtsmen, costing clerks, etc. (very healthy; S.M.R., 65)	23.5%	19.7%

Knowledge advances and fashions in certification change. The accuracy of all statistics is dependent upon the accuracy of the data from which they are compiled. Owing to the war the next decennial report will presumably be based on the 1951 census (1941 being omitted). I venture to predict that by that time there will be a sharp decline in nephritis among older persons and a corresponding increase in deaths from hypertension and hypertensive heart failure.

In certain textile trades there may be influences at work whose effect is to hasten or even to initiate cardiovascular and “renal” disease. This investigation of the evidence does not disprove such a possibility, but it suggests that before setting out on a laborious, time-consuming, and expensive investigation into the cause and prevention of nephritis in textile workers we should need more evidence that it exists. In particular we would require evidence that there is a renal syndrome among textile workers which is in some way different in its incidence, symptomatology, or course from hypertensive disease in the general population.

Conclusions

This paper proves nothing. It suggests that the deaths from so-called nephritis in textile workers are in all probability due to hypertension, and can be as easily explained by natural variations in the genetic constitution of an inbred population as they can by environmental influences.

This should be borne in mind before making the assumption that such deaths are due to “the inhalation of textile dusts.”

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