## Distribution of Hypertension and Renal Disease in Oregon

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SINCE RENAL DISEASE and systemic hypertension frequently co-exist clinically (1, 2), a positive correlation in their geographic distributions should not be unexpected. Such a positive correlation is evident in table 1, which lists the States with the highest and lowest mortality rates for both hypertensive heart disease (International Classification of Diseases (ICD) rubrics 440-443) and chronic nephritis (ICD 592-594). In 1960, 9 of the 11 States with high rates for hypertensive heart disease also had high rates for chronic nephritis. Peak mortality for both categories of disease occurred in the southeastern United States and in Hawaii. The "Southeast" in this instance did not include Florida but did extend as far north as
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Delaware, which suggests that population characteristics might be more important than simple geographic or climatologic variables in determining this pattern.

McDonough, using 1959-61 mean mortality rates for hypertension and hypertensive heart disease (ICD 440-447), showed that the increased risk of hypertension mortality in the Southeast was greater for blacks than for whites and that the peak hypertension mortality for white females occurred in the Middle Atlantic States of New York, New Jersey, and Pennsylvania (3). Earlier, Rose had also reported that the nation's highest rates were found in the southeastern States during 1949-51 for both white and black persons aged 40-64 years (4). Thus, this geographic pattern of increased hypertension mortality has persisted for at least a decade, although it has been more consistent for blacks than for whites.

At the other end of the scale in table 1, the geographic agreement for low mortality risks for hypertension and chronic nephritis is less complete. Alaska and the northwest Great Plains States of Nebraska, South Dakota, and Wyoming are found in both columns. McDonough's 1959-61 data showed that the Rocky Mountain region was lowest in hypertension mortality for all four groups: white and nonwhite males and females (3). Rose's 1949-51 agetruncated data were in agreement, showing the same geographic pattern of least hypertension mortality for both whites and nonwhites (4).

Several reports have documented the falling nephritis mortality rates and stable kidney infection mortality rates in the United States and in

England and Wales (5-9). Rothenberg and Heymann published 1949-51 mortality rates for nephritis and nephrosis in pediatric age groups by State which indicated that both acute and chronic renal mortality rates tended to be highest in the Southeast in both white and nonwhite children, although rates were usually higher among the nonwhites (10). A later study of the nephrotic syndrome among children in Erie County, N.Y., documented declining incidence and mortality rates between 1946 and 1961, confirmed that the rates were higher among nonwhites than whites, and showed that incidence rates were inversely
correlated with socioeconomic status in both whites and nonwhites. (11, 12). National patterns of geographic variation in nephritis mortality have been reported from Australia (13) and Canada (14) and undoubtedly exist elsewhere.

Stimulated by the geographic pattern of hypertension and chronic nephritis mortality rates in the United States and by the identification of an apparently meaningful geographic pattern of hypertension distribution in Colorado (15), we decided to investigate the epidemiology of hypertension in Oregon and additionally to search for correlations with the distributions of renal dis-

Table 1. States with highest and lowest mortality rates per $\mathbf{1 0 0 , 0 0 0}$ for hypertensive heart disease and chronic nephritis, 1960


[^0]ease. This report is concerned with data from our pilot statewide survey of mortality statistics and Selective Service medical examinations.

## Methods

Mortality data for hypertensive and renal diseases were obtained from the Health Division of the Oregon State Department of Human Resources and were agestandardized by the direct method with the 1970 Oregon population as the age distribution standard. To obtain rates which were relatively free from excessive sampling variation, counties were grouped into somewhat homogeneous geographic regions (see map), and 5 -year mean annual rates were used. The 1955 and 1965 population bases were the sums of the age-specific midpoints between the 1950 and 1960 censuses and the 1960 and 1970 censuses, respectively, by county. Mortality statistics by-age-by-cause-bycounty prior to 1950 are unavailable in Oregon.

Prevalence data were obtained from the Oregon Selective Service System for all registrants born during 1939, 1940, and 1941, the same birth year cohort that was studied in Colorado. In Oregon, the record review was conducted at the State headquarters office instead of at each local board office, as had been done in Colorado. The record review began in April 1971 and continued for a year, as all registration folders were forwarded from the 32 local boards to State headquarters for destruction. Among the 37,101 Oregon registrants born in these 3 years were 15,887 (43 percent) whose files contained medical examination forms or medical diagnosis statements, or both; all of these documents were retained for this study. We were able, therefore, to check the accuracy of coding, which was a distinct advantage over the Colorado study. As in the Colorado study, the study was legally limited to an epidemiologic compilation, and registrants could not be contacted for information nor their identity revealed to others.

Although the local boards can grant medical deferment from military duty for obvious gross and serious medical problems, the overwhelming majority of medical deferments in Oregon were based on the results of medical examinations at the Armed Forces Entrance Examination Station (AFEES). The Oregon AFEES, located in Portland, serves all of the State except the Snake River counties, which are served by the Boise, Idaho, AFEES. Although many men had the results of more than one examination in their files, descriptive data including body size and blood pressure were coded as of the initial examination. They ranged in age from 17 to 26 years at first examination with 22 the modal age (mean $=21.52$ ). We found that a considerable number of registrants had been processed after July 1965, the cut-off date for the Colorado survey of this birth year cohort, and this later termination date constitutes a modest
difference in sampling bias between the two State surveys.

Blood pressure had been measured with the subject in the sitting position, and the lowest values obtained were recorded and coded. The majority of men with elevated blood pressure measurements had been retested after varying periods of rest, often after several days or weeks had elapsed. Usually those whose elevated initial blood pressures had not been rechecked were men whom the Armed Forces considered undesirable for other reasons besides their blood pressure. An AFEES examination probably constitutes more of a stress situation than the average medical examination, but to compensate for stress, blood pressure is measured after rest periods and with the subject in a recumbent position and it is remeasured after several weeks or months before a diagnosis of hypertension is made.

Table 2 shows the categories we


Table 2. Classification of blood pressure data for Oregon Selective Service System registrants born in 1939-41 and examined during 1957-69

| Code and category |
| :--- | :--- | :--- | :--- |

NOTE: AFEES - Armed Forces Entrance Examination Station, SBP — systolic blood pressure, DBP — diastolic blood pressure.
used to classify the combinations of blood pressure values and diagnoses encountered. Although Army regulations, effective beginning in 1961, lowered the systolic blood pressure criterion from 150 to 140 mm Hg for men 35 years or younger, Karpinos has clearly shown that AFEES medical examiners have generally tended to continue to make diagnoses of hypertension based on the $150-\mathrm{mm}$ standard and to underrecord seriously blood pressure values from 142 to 154 mm Hg since that date (16). Because these Oregon data exhibit the same characteristics that Karpinos described for the entire country, it seems reasonable to regard the systolic criterion listed in table 2 as the basis for a diagnosis throughout the examination period, despite the change in regulations. For only 26 (4.9 percent) of the 529 men with
diagnoses of essential hypertension was the diagnosis based on a minimum systolic pressure value of $142-150 \mathrm{~mm} \mathrm{Hg}$ and a recorded diastolic value in the normal range ( 90 mm or less). (These 26 men are not separately identified in table 2.) These statistics nicely illustrate that regulations do not always have their intended effects.

## Results

Table 3 shows that the mortality rate for hypertension in Oregon fell dramatically over the past 23 years. It should be remembered that between 1967 and 1968 a change in cause-of-death coding reduced the deaths coded as hypertension by a factor of slightly more than 2. Despite the coding change, it is evident that the decline in hypertension mortality was not uniform across the State. Perhaps the most striking change occurred in the

Portland Metropolitan area which had Oregon's highest rates during 1950-57 but, during 1963-72, was below the statewide rate. The consistency of that trend is certainly important. The difference in mean hypertension mortality between western and eastern Oregon steadily diminished and has virtually vanished during the 1968-72 period. The overall decline in hypertension mortality was least evident in the counties of the southern Cascade, the over $4,000-\mathrm{ft}$ elevation, and Snake River regions, where the rates were lowest initially and where the decline appeared to be arrested during 1968-72, if one allows for the effects of the 1968 coding change. That this is not an artifact attributable to the coding change is suggested by the high rates in the over $4000-\mathrm{ft}$ region, which have continued since 195357 , and the very slight relative

Table 3. Mortality rates for hypertension (ICD 440-447 in 1950-67 and ICD 400-404 in 1968-72) by regions of Oregon

| Region | Age-standardized mean annual rates per 100,000 |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  | 1950-52 | 1953-57 | 1958-62 | 1963-67 | 1968-72 |
| Western | 89 | 65 | 44 | 32 | 10 |
| Coastal | 83 | 65 | 42 | 35 | 10 |
| Portland Metropolitan | 100 | 70 | 45 | 31 | 8 |
| Willamette Valley . . | 77 | 58 | 43 | 32 | 9 |
| Southern Cascade | 69 | 52 | 41 | 35 | 22 |
| Eastern | 70 | 51 | 39 | 28 | 11 |
| Columbia River | 76 | 48 | 32 | 27 | 7 |
| Central | 70 | 56 | 40 | 21 | 7 |
| Over 4000 ft elevation | 65 | 68 | 56 | 43 | 19 |
| Snake River | 66 | 40 | 37 | 26 | 13 |
| Oregon. | 87 | 63 | 43 | 32 | 10 |

NOTE: geographic regions contain the following counties: Coastal-Clatsop, Columbia, Coos, Curry, Lincoln. Tillamook; Portland Metropolitan-Clackamas, Multnomah, Washington; Willamette Valley, Benton, Lane, Linn, Marion, Polk, Yamhill; Southern Cascade-Douglas, Jackson, Josephine; Columbia River-Gilliam, Hood River, Morrow, Sherman, Umatilla, Wasco; Central-Crook, Deschutes, Grant, Jefferson, Wheeler; Over 4,000 ft. elevation-Harney, Klamath, Lake; Snake River-Baker, Malheur, Union, Wallowa.

Table 4. Mortality rates for hypertension and associated vascular diseases, by regions of Oregon, 1968-72

\left.|  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
|  |  | Mean annual crude rates per 100,000 |  |  |$\right]$

${ }^{\text {I }}$ ICD 400-404. ${ }^{2}$ ICD 410.0, 411.0, 412.1, and 412.2. ${ }^{3}$ ICD 430.0, 431.0, 432.0, 433.0, 434.0, 435.0, 436.0, 437.0, 438.0.
elevation in the rate for the southern Cascade counties in 196367.

The cause-of-death coding change in 1968 may have rendered judgment of hypertension mortality trends more difficult, but it now allows identification of hypertensive persons who died of ischemic heart disease and cerebrovascular disease, approximately doubling the number of readily identifiable hypertension deaths. In table 4 the consistency of the geographic distributions of hypertension-
hypertensive heart disease, hypertension-ischemic heart disease, and hypertensioncerebrovascular disease are compared. The geographic pattern of hypertension mortality is entirely consistent for these three disease categories in western Oregon but much less so in eastern Oregon, where the hypertensive heart disease and the cerebrovascular disease categories closely resemble each other. The combined hypertension mortality rate suggests that the mortality risk is
definitely greater in the southern Cascade counties and perhaps is unremarkable elsewhere in the State.
Oregon's nephritis and nephrosis mortality rate also fell dramatically between 1950 and 1972, a period when the rate for eastern Oregon consistently exceeded that for western Oregon (table 5). However, rates for county groups in the two halves of the State have not been as consistent, and the major part of eastern Oregon's excess nephritis mortality rate has been contributed by the Snake River counties, which appear to constitute a high-risk region. The most important impression gained from tables 4 and 5 is that Oregon's nephritis mortality rates have not varied in association with the State's hypertension mortality rates as much as the national mortality data would lead one to expect. We cannot explain Oregon's geographic pattern of hypertension mortality by the distribution of nephritis and nephrosis fatalities.

Pyelonephritis has also long been associated with hypertension, although it is probably best regarded as one of the results rather than a cause (2, 17-20). Mortality trends for kidney infection in Oregon during 1950-72 showed a geographic pattern quite different than the hypertension and nephritis deaths considered previously (table 5). This rate increased gradually until 1958-67 and then declined everywhere except in the Snake River counties. The Portland Metropolitan area consistently had the highest kidney infection death rate in Oregon over the 23-year period, a ranking which might reflect more urologic facilities and staff, better survival from other renal and urologic conditions, better diagnosis, or a host of other factors. Thus, the mortality rate patterns for these renal diseases neither reflect nor correlate with the recent pattern of hypertension mortality in Oregon.

The statewide morbidity data in table 6 show the geographic distributions of all registrants listed by the Oregon Selective Service

System, the proportion of registrants for whom we had medical examination forms or other medical information, and those registrants for whom there was evidence of past or present concern about hypertension or renal disorders. The sampling fraction for whom we had medical data is fairly uniform around the State (varying
from 39 to 46 percent in table 6 and from 38 to 52 percent by individual local boards), as would be expected by the medical requirements of a national law and by the existence of State and national review procedures. The prevalence of men with some evidence or past history of hypertension was 79 per 1,000 registrants coded ( 34 per 1,000 of

Table 5. Mortality for nephritis and nephrosis (ICD 590-594 in 1950-67; 580-584 in 1968-72) and for kidney infections (ICD 600 in 1950-67; 590 in 1968-72),by regions of Oregon

|  |  | Age-standardized mean annual rates per 100,000 |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Region' | $1950-52$ | $1953-57$ | $1958-62$ | $1963-67$ |  |

${ }^{1}$ See footnote to table 3.
those listed) for the entire State. The variation in hypertension prevalence agrees with the 1963-67 geographic pattern of hypertension mortality in western Oregon, but not in eastern Oregon. The discrepancy in eastern Oregon between mortality and prevalence patterns consists of (a) high mortality rates and for males a low prevalence rate in counties over 4,000 ft elevation and (b) low mortality rates and for males a high prevalence rate in the Snake River counties. Since the mortality rates are for the total population, these discrepancies might be due to major sex differences in hypertension mortality in these two regions as well as to sampling error, and we expect to investigate this further. Renal disorders as a group were consistently more prevalent among the registrants in the eastern than in the western regions of Oregon.

Classification of men into diagnostic categories of hypertension is shown in table 7. Of the $1,-$ 248 with potential hypertension, only 529 ( 42 percent) were diagnosed as hypertensive. The distribution of definite hypertensives was geographically similar to the 196367 mortality pattern. The excess of borderline hypertensives in the Snake River counties may have been a function of different measurement procedures in the Boise AFEES, of the region's high nephritis mortality rates, or of

Table 6. Hypertension and renal disorders among Selective Service registrants born 1939-41 and examined 1957-69, by regions of Oregon

| Region' | Registrants |  |  | Hypertension, all categories |  | All renal disorders |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Listod | Coded |  |  |  |  |  |
|  |  | Number | Percent | Cases | Rates ${ }^{2}$ | Cases | Rates ${ }^{2}$ |
| Western | 30,972 | 13,444 | 42 | 1,030 | 78 | 406 | 31 |
| Coastal | 3,621 | 1,575 | 43 | 130 | 83 | 52 | 33 |
| Portland Metropolitan | 13,850 | 6,012 | 43 | 454 | 76 | 174 | 29 |
| Willamette Valley . . . . | 9,552 | 3,998 | 42 | 317 | 79 | 132 | 33 |
| Southern Cascade | 3,949 | 1,559 | 39 | 129 | 83 | 48 | 31 |
| Eastern | 6,129 | 2,743 | 45 | 218 | 79 | 115 | 42 |
| Columbia River | 1,993 | 904 | 45 | 70 | 77 | 43 | 48 |
| Central | 1,223 | 511 | 42 | 37 | 72 | 22 | 43 |
| Over 4,000 ft elevation | 1,369 | 626 | 46 | 42 | 67 | 22 | 35 |
| Snake River ......... | 1,544 | 702 | 45 | 69 | 98 | 28 | 40 |
| Oregon. | 37,101 | 15,887 | 43 | 1,248 | 79 | 521 | 33 |

[^1]| Region' | Definite hypertension |  | Borderline hypertension |  | Hypertension history but normal blood pressure |  | Secondary hypertension |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Cases | Ratos ${ }^{\text {2 }}$ | Cases | Rates ${ }^{\text {a }}$. | Cases | Rates ${ }^{2}$ | Cases | Rates ${ }^{2}$ |
| Western | 446 | 34 | 391 | 30 | 166 | 13 | 27 | 2 |
| Coastal | 65 | 41 | 43 | 27 | 22 | 14 | 0 | 0 |
| Portland Metropolitan | 181 | 30 | 187 | 31 | 70 | 12 | 16 | 3 |
| Willamette Valley . . . . | 131 | 33 | 118 | 30 | 59 | 15 | 9 | 2 |
| Southern Cascade | 69 | 44 | . 43 | 28 | 15 | 10 | 2 | 1 |
| Eastern | 83 | 30 | 97 | 35 | 34 | 12 | 4 | 1 |
| Columbia River | 27 | 30 | 27 | 30 | 15 | 17 | 1 | 1 |
| Central . . . . . | 13 | 25 | 17 | 33 | 7 | 14 | 0 | 0 |
| Over 4,000 ft elevation | 17 | 27 | 19 | 30 | 5 | 8 | 1 | 2 |
| Snake River | 26 | 37 | 34 | 48 | 7 | 10 | 2 | 3 |
| Oregon. | 529 | 33 | 488 | 31 | 200 | 13 | 31 | 2 |

'See footnote to table 3 . ${ }^{2}$ Rates per 1,000 registrants with medical records.
other factors unknown at present. None of the rates for the three other categories of hypertension (borderline, past history only, and secondary) shows a geographic pattern which matches the patterns of hypertension mortality or of definite hypertension prevalence. One view of this phenomenon is that the frequencies of persistent hypertension and of its fatal outcome vary geographically and probably temporally, although the underlying (constitutional) predisposition might not.

Of the 529 men diagnosed as having essential hypertension by the Selective Service System, only 183 (35 percent) were known
previously, and only 35 (7 percent) gave a history of previous or current antihypertensive treatment for any period (table 8 ). Only 19 percent of those previously known to be hypertensive had received antihypertensive therapy, according to the available records. These proportions were relatively uniform around the State except that in the Central counties there seems to have been a greater tendency to measure blood pressure and to initiate antihypertensive treatment in this age group. Reluctance to begin treatment was particularly notable in the southern Cascade and eastern Columbia River counties. Among the 529 cases were four men

Table 8. Previously known disease and treatment of 529 men with hypertension diagnoses among Selective Service registrants born 1939-41 and examined 1957-69, by regions of Oregon

| Region' | $\begin{gathered} \text { Number } \\ \text { with } \\ \text { deflinite } \\ \text { hypertension } \end{gathered}$ | Hypertension known before AFEES |  | History of antihypertonsive treatment |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  |  |  | Number | Percent of- |  |
|  |  | Number | Percent |  | Total | Known before AFEES |
| Western | 446 | 151 | 34 | 29 | 7 | 19 |
| Coastal | . 65 | 15 | 23 | 3 | 5 | 20 |
| Portland Metropolitan | .. 181 | 64 | 35 | 13 | 7 | 20 |
| Willamette Valley . . . . | .. 131 | 51 | 39 | 11 | 8 | 22 |
| Southern Cascade ... | . 69 | 21 | 30 | 2 | 3 | 10 |
| Eastern | 83 | 32 | 39 | 6 | 7 | 19 |
| Columbia River | . 27 | 10 | 37 | 1 | 4 | 10 |
| Central .... . . . | . 13 | 8 | 62 | 2 | 15 | 25 |
| Over 4,000 ft elevation | . 17 | 5 | 29 | 1 | 6 | 20 |
| Snake River . . . . . . . . | . 26 | 9 | 35 | 2 | 8 | 22 |
| Oregon. | . . 529 | 183 | 35 | 35 | 7 | 19 |

'See footnote to table 3.
Note: AFEES-Armed Forces Entrance Examination Station.
with electrocardiographic evidence of left ventricular enlargement; none had received treatment. Only 1 of the 529 was diagnosed as having malignant hypertension, and this young man manifested grade 3 retinopathy, a double left kidney and ureter, radiographic evidence of renal artery stenosis, asthma, and obesity. His was the only identified case of renovascular hypertension as well.

Table 9 presents the geographic distributions and regional rates for various renal disorders specified in the Selective Service records. The cases add up to more than the 521 men listed in table 6 because some were classified in more than one diagnostic category. A history of clinical glomerulonephritis was given by 116 persons. Their geographic distribution is unremarkable except that the relatively low prevalence rate for the Snake River counties contrasts strongly with the generally high nephritis mortality rates for that region. More frequent than nephritis among these registrants was a history of kidney infection; their geographic distribution is also unremarkable. A history of renal trauma (post-traumatic hematuria or worse) was relatively more frequent among registrants from coastal counties and parts of eastern Oregon, reflecting some but not all of the regions in the State where renal trauma is a common occupational hazard in activities

Table 9. Specified renal disorders in the medical records of Selective Service registrants born 1939-41 and examined 1957-69, by regions of Oregon

| Region' | Glomerulonephrtits |  | Pyelonephritis |  | Renal trauma |  | Nophroilthiasis |  | Albuminuria, NOS |  | Other andunspocitioddisordars |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Casos | Ratos ${ }^{\text {2 }}$ | Casos | Rates ${ }^{\text { }}$ | Cases | Ratos ${ }^{2}$ | Casos | Ratos ${ }^{2}$ | Cases | Ratos ${ }^{\text {2 }}$ | Cases | Rates ${ }^{2}$ |
| Western | 86 | 7 | 115 | 9 | 41 | 3 | 27 | 2 | 131 | 10 | 66 | 5 |
| Coastal | 14 | 9 | 14 | 9 | 9 | 6 | 3 | 2 | 12 | 8 | 13 | 8 |
| Portland Metropolitan | 35 | 6 | 43 | 7 | 15 | 2 | 13 | 2 | 64 | 11 | 17 | 3 |
| Willamette Valley . | 24 | 6 | 43 | 9 | 11 | 3 | 9 | 2 | 41 | 10 | 27 | 7 |
| Southern Cascade | 13 | 8 |  | 10 |  | 4 |  | 1 | 14 |  | 9 | 6 |
| Eastern | 30 | 11 | 26 | 9 | 16 |  | 13 |  | 37 | 13 | 23 | 8 |
| Columbia River | 9 | 10 | 9 | 10 | 6 | 7 | 2 | 2 | 14 | 15 | 13 | 14 |
| Central ...... | 7 | 14 | 5 | 10 | 2 | 4 | , | 6 | 6 | 12 | 5 | 10 |
| Over 4,000 ft elevation | 9 | 14 |  | 6 |  | 2 | 5 | 8 | 9 | 14 | 2 | 3 |
| Snake River | 5 | 7 | 8 | 11 | 7 | 10 | 3 | 4 | 8 | 11 | 3 | 4 |
| Oregon. | 116 | 7 | 141 | 9 | 57 | 4 | 40 | 3 | 168 | 11 | 89 | 6 |

'See footnote to table 3. ${ }^{2}$ Rates per 1,000 registrants with medical records. NOTE: NOS-Not otherwise specified.
such as logging, ranching, and rodeos. A history of renal or ureteral stones was distinctly more frequent among residents of the drier regions of eastern Oregon, especially the counties above $4,000 \mathrm{ft}$, than among residents of moist western Oregon. Past or present albuminuria not otherwise specified (NOS) was uniformly more frequent in eastern than in western Oregon, although the differences were not great.
Other and unspecified renal conditions included 19 diagnoses of hydronephrosis or other ureteral disorders and 9 major congenital renal malformations (3 of polycystic kidneys, 2 of unilateral absent kidney, 3 of unilateral double kidney, and 1 of horseshoe kidney joined at inferior poles). Of the total with renal disorders, 30 men (6 percent) had undergone surgical nephrectomy, most often as an aftermath of trauma.

The prevalence of definite hypertension among registrants with various renal diseases is compared in table 10 to hypertension prevalence in the absence of known renal disease. There is a highly significant increased prevalence of definite hypertension among registrants with any previous renal disorder compared to those with none. Although the prevalence of hypertension is increased in each renal diagnostic category, only for glomerulonephritis is the difference
significant at $\boldsymbol{a}=.01$. If these observed rates persisted in a larger population, then more cases of renal disorder would eventually cause the elevated hypertension prevalences for the other disorders to become significant too. However, only a small proportion (7 percent) of cases of definite hypertension were associated with known renal disorders among young men examined for the Armed Forces in Oregon in 1957-69.

## Discussion

Although the southeastern States and Hawaii showed peak mortality for hypertension which coincided with peak mortality for nephritis in 1960, the same geographic relationship could not be found in a
more detailed study of hypertension and renal disease mortality in Oregon over the 23 years from 1950 through 1972. Neither could we find a geographic association between hypertension prevalence and the frequencies of past glomerulonephritis or other renal conditions among a cohort of Selective Service registrants. Although both hypertension and nephritis mortality rates have fallen dramatically and persistently in Oregon during the 23 years, we have no comparable information about incidence or prevalence trends, since our morbidity data describe only a single cohort during a short period. However, if glomerulonephritis incidence and severity trends resemble those for

Table 10. Prevalence of definite hypertension among Oregon Selective Service registrants with previous renal disorders

| Renal disorders | Number of registrants | Hypertension |  | Significance of difference from none' |
| :---: | :---: | :---: | :---: | :---: |
|  |  | Number | $\begin{aligned} & \text { Rate per } \\ & 1,000 \end{aligned}$ |  |
| None | 15,366 | 492 | 32 |  |
| Any | 521 | 37 | 71 | . 0015 |
| Glomerulonephritis | 116 | 14 | 121 | . 003 |
| Pyelonephritis | 141 | 9 | 64 | . 12 |
| Renal trauma | 57 | 4 | 70 | . 26 |
| Nephrolithiasis | 40 | 2 | 50 | . 60 |
| Albuminuria, not otherwise specified | 168 | 11 | 65 | . 08 |
| Other and unspecified . . . . . . . . . . . . | 89 | 9 | 101 | . 03 |
| 'According to the computation where $B=$ the rate base used, for example, 1,000, and the |  |  | $\bar{x}_{1}$ | $\frac{\bar{x}_{2}(B-}{N_{2}}$ |

rheumatic fever, then they should be declining in Oregon because rheumatic fever mortality and reported incidence rates have declined dramatically during the past 23 years (unpublished tabulations by W. E. M.). In subsequent studies we hope to determine what changes, if any, have occurred in hypertension prevalence rates among other cohorts of Oregon Selective Service registrants.

The primary goal of this compliation of data was to identify geographic variations in the risk of hypertension occurrence within Oregon. However, this goal has been only partially realized because our morbidity data antedate the 1968-72 period during which the geographic pattern in hypertension mortality became clearly evident. The section of Oregon which seems most likely to warrant description as a relatively high-risk region for hypertension is the southern Cascade counties, a mountainous area containing the Rogue and Umpqua Rivers and the small cities of Medford, Grants Pass, and Roseburg. Like many other parts of Oregon, the economy is based on lumber, livestock, orchards, and transportation and has been slowly expanding. In this region the expected decline of the hypertension mortality rate was arrested during 1968-72, and the prevalence of definite hypertension was relatively but not significantly elevated among Selective Service registrants prior to that. Although rigorous medical management of hypertensive episodes in adolescence and childhood might alleviate the prevalence of hypertension among young adults, there is, as yet, no evidence for such an effect. The southern Cascade region's very low proportion of registrants with hypertension known to have received therapy is not an explanation of the region's risk status, since it was not unique in this regard. There is no recent evidence of a shortage of generalist or specialist physicians in this region compared to the rest of the State (21).

A similar study of hypertension distribution in Colorado showed a
high-risk region, characterized by an arrested decline of hypertension mortality in the general population and a high prevalence of hypertension in young men; the region was also distinguished by high nitrate levels in municipal water sources (22). Because nitrate levels reflect pollution by organic materials and there is evidence that ground water nitrate concentrations may be rising in some regions (23-25), these facts seemed to be a reasonable basis to suspect an environmental effect upon blood pressure levels, particularly in view of the evidence that chronic exposure to organic aliphatic nitrates results in increased risks of diastolic hypertension and sudden death among explosives workers (26-29, references in 22). Unfortunately, there is no statewide compilation of the chemical quality of water in Oregon such as was available in Colorado, although a special study of nitrate levels is currently underway.

If we can identify and manipulate environmental factors which influence the manifestation and outcome of hypertension, then persons affected by environmental factors could be ranked with renovascular hypertensives as correctable, the principal difference being that the correction would operate at the community environmental level rather than the individual level and that the correction would not require surgical intervention. Surely this correction would be a worthy achievement.

Some may regard hypertension diagnoses by the Selective Service System as too stress-associated to be reliable. However, the proportion of draftees with apparent potential blood pressure abnormalities is less than 8 percent, a level which is quite compatible with frequencies measured in the national Health Examination Survey (30) and in a followup study of university students (31). Even if one wished to consider virtually all the draftees as having "borderline" or "labile" cases, the prevailing medical experience to date indicates definite increased risk of eventual persistent hypertension as
well as elevated morbidity and mortality rates and validates a cautiously active approach to medical management and followup of such cases (32). The 200 Oregon registrants with only a past history of medically recognized blood pressure elevation (table 7) are an index of a generally available epidemiologic and clinical opportunity in that the early stages of essential hypertension are still unclear and may consist of cyclical blood pressure elevations which eventually become persistent. What proportion of this group progresses to the establishment of persistent hypertension? Is the proportion affected by the environment? Can the proportion be favorably affected by early medical therapy and followup? These are important clinical questions whose answers will be essential to the development of effective community control of hypertension (33, 34).

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## STMOPSIS

MORTON, WILLIAM E. (University of Oregon Medical School), KNUDSEN, JOHN C., and PORTER, GEORGE A.: Distribution of hypertension and renal disease in Oregon. Public Health Reports, Vol. 90, January-February 1975, pp. 34-43.

Expecting to find agreement between the geographic distribution of hypertension and renal disease, we developed regional mortality rates for 1950-72 and prevalence rates for a Selective Service cohort born in 193941 and examined during 1957-69. For this purpose the State's counties were grouped into eight geographically homogeneous regions.

The general decline in hypertension mortality was most pronounced in Portland, Oregon's major urban center. However, the decline halted during 1968-72 in the southern Cascade region which has become an area of relatively higher risk within the State. During these 23 years nephritis mortality fell, kidney infection mortality was stable, and both syndromes showed peak mortality in other, different regions of the State. The geographic pattern of hypertension prevalence among the draftee cohort resembled the 1963-67 hypertension mortality pattern, but more recent morbidity data are needed to confirm the southern Cascade region's recent change to a
high-risk area.
Of 529 draftees with diagnosed hypertension, only 35 percent of the cases were previously known, only 7 percent had had any previous treatment, and only 7 percent were associated with known renal conditions. Among 521 registrants with a history of renal disorders, the prevalence of hypertension was increased for all categories of renal disease but was significantly high only for those with a history of glomerulonephritis.

To date in Oregon we have found no evidence that renal disorders are major determinants of hypertension morbidity or mortality.


[^0]:    ${ }^{1}$ Oregon 20.3.
    SOURCE:8 Age-standardized rates per 100,000 were adapted from "Cardiovascular Disease, 1960 Data on National and State Mortality Experience," U.S. Government Printing Office, Washington, D.C., 1964.

[^1]:    'See footnote to table 3. ${ }^{2}$ Rates per 1,000 registrants with medical records.

