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INQUIRY INTO THE CHRONIC NEPHRITIS OF QUEENSLAND.

Precis of certain aspects of the Official Inquiry into the Chronic Nephritis of Queensland, with special reference to Plumbism in children.

BY

R. W. CILENTO, M.D., B.S., D.T.M. & H.

Chief Quarantine Officer (General), North-Eastern Division,
Commonwealth Department of Health.

IN April, 1930, an inquiry into the frequency of chronic nephritis at early ages in Queensland was authorized and entrusted to the writer, the work being carried out in the Commonwealth Health Laboratories and the Central Office of the North-Eastern Division of the Commonwealth Department of Health.

Much of the available material was unsatisfactory, since most of the evidence from which previous conclusions had been drawn was originally incomplete, and was found no longer accessible. As a consequence, the findings here recorded are arrived at on a basis of compromise, or are necessarily expressed in indeterminate form.

A comparison of the death rates from chronic nephritis in the States of the eastern seaboard of Australia and in New Zealand, provided the following comment.

There was a very considerable increase in death rates of persons under 40 years from chronic nephritis in Queensland, as compared with the other States; there was, nevertheless, an equally marked drop as compared with other States in the death rates from 40 years onwards. When the total Queensland figures for all ages were compared with those for Victoria, for example, the average death rates per 100,000 for the period 1928-1930 inclusive, were almost identical, being as 52 : 51; but in the one instance the mass of the cases died before 40 years of age, and in the other, after 40.

The averages for Queensland and Victoria are consistently above that for New South Wales, and all the States of the eastern seaboard are very markedly above the figures for New Zealand.

Taking the death rate for New Zealand as normal, it would appear that if there exists in Queensland an obvious problem in chronic nephritis with death before the age of 40, there exists an equally obvious problem in chronic nephritis, with death after the age of 40, in, say, Victoria.

If the figures for the Eastern States are taken as normal, on the other hand, whatever cause is acting in Queensland would appear to act not as a separate and independent factor, but as something which accelerates by some twenty years the deaths of a certain number who would normally die of chronic nephritis. (See Figure I.)

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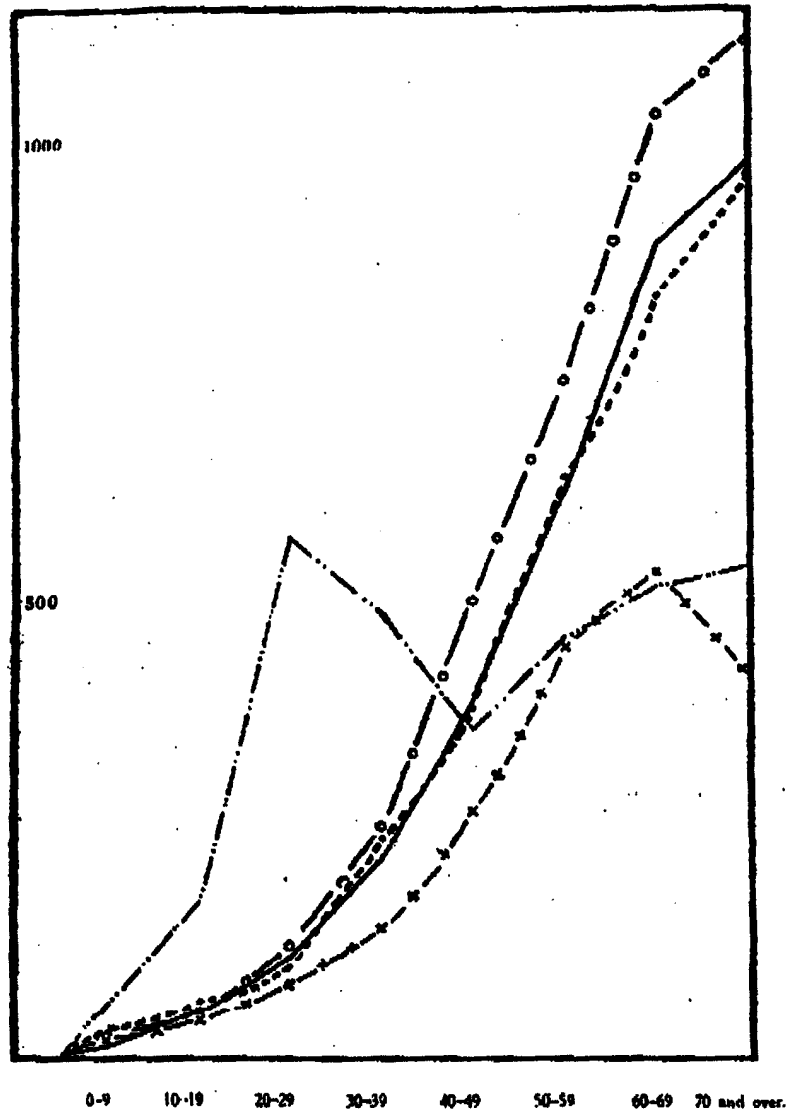


FIGURE I.
Total deaths from Chronic Nephritis, 1917-26, per 800,000 of population in decennial age-groups.

Queensland — · · · · · South Australia — · · · · · Victoria — o — o — o — o —
New South Wales — — — — — New Zealand x — x — x — x —

Note: The Curve for New Zealand represents only the period 1925-1926 averaged out as a ten-year period.

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It had been suggested by Nye that there was a "definite association" between the prevalence of chronic nephritis and the percentage prevalence of wooden houses painted with white lead paints, as follows:—

TABLE I.
(From Nye's Figure IX.)

State.				Wooden Painted Houses.	Deaths from Nephritis per 100,000.
				Per cent.	
Queensland	..	..	..	85	322
Victoria	..	..	..	68	108
New South Wales	..	..	..	48	102
South Australia	..	..	..	8	97

In this table, it is not known what is included in the term "Nephritis". The deaths per 100,000 from chronic nephritis over the five-year period mentioned above, are, respectively: Queensland, 52; Victoria, 51; New South Wales, 48; South Australia, 43.

When, moreover, the comparison is extended to include New Zealand, it is found that while 90 per cent. of the buildings in that country are of wood (that is, a greater percentage than any other in the series, including Queensland), the figures for chronic nephritis are exceptionally low, and lead poisoning among children is unknown.

In respect of the further contention that the important factor is not the matter of residence in a painted wooden house, but certain climatological factors affecting the weathering of paint, examinations in this regard were instituted. From these it became apparent that in New Zealand, which, as stated, with the highest percentage of painted wooden houses in Australasia, has the lowest percentage of chronic nephritis and no lead poisoning, the lead paint nevertheless does "weather", and soluble lead in proportions and quantities similar to those found in Queensland, is present on the walls, verandah rails, and so forth, of houses.

Outside Brisbane and certain coastal towns in Queensland, where children reside in painted wooden houses set on piles, in hot and humid climates, cases of lead poisoning do not figure in the hospital records.

For the actual distribution of chronic nephritis in Queensland, the figures were so small in certain individual towns that one is scarcely justified in drawing any conclusion whatever from them, but, taken in the aggregate, certain interesting particulars emerge.

The figures for deaths from chronic nephritis in the city of Brisbane, set out in decennial age groups, are closely imitated by those of Ipswich, Rockhampton, and Townsville, with which the Charters Towers group, though small enough to conceal many fallacies, may perhaps be included. The figures for the Maryborough group, on the other hand,

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including Maryborough, Cairns, Gympie, Mackay, Bundaberg, and Toowoomba, provide a very different picture, comparable with that found, for example, in Victoria (*see* Figures II. (a) and (b)).

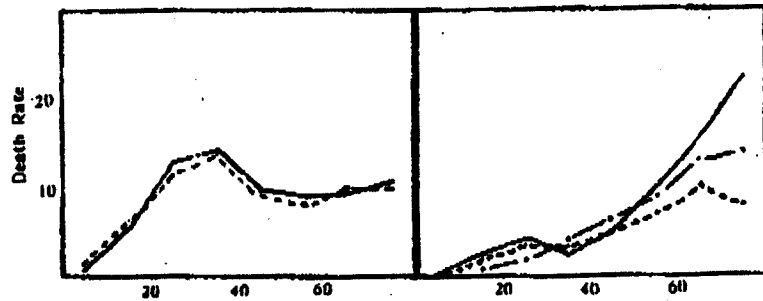


FIGURE IIa.

Death rate in decennial age groups per 100,000. For cities of Brisbane type — (Brisbane - - - - -) 1928 and 1929.

Brisbane
Ipswich
Rockhampton
Townsville
Charters Towers

Population
400,000.

FIGURE IIb.

Death rate in decennial age groups per 100,000 for cities of Maryborough type and rest of Country Districts 1928 and 1929 compared with Victorian average 1917-26.

Maryborough
Cairns
Gympie
Mackay
Bundaberg
Toowoomba

Population
75,000

Maryborough type cities — Rest of
Country - - - - - Victorian average
10 years per 100,000

It was demonstrated that in the cities of the Brisbane series, with a population of 400,789 people, there was a total death rate for the period 1928-1929 inclusive, of 265 persons before the age of 40; in the rest of the State, whether city or country, with a population of 511,497, there was a total loss of only 83 lives before the age of 40 years. That is to say, in this particular, these five cities alone, compared with the rest of the State (which has a population some 25 per cent. larger), show a ratio of 33 : 8 per 100,000 per annum in respect of chronic nephritis.

It appeared perfectly obvious, therefore, that in these particular cities, a definite factor was causing a mortality more than four times as great as elsewhere for chronic nephritis before the age of 40 years. It was just as apparent, however, that the same circumstances did not occur in other cities where conditions of life were apparently similar, but that, on the contrary, in such places, very definite similarities not only with the rest of Queensland, but with the rest of Australia were found, irrespective of the fact that they included every range of climate in Queensland.

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Thus, for the Brisbane series, with a population of approximately 400,000 persons, 204 deaths are recorded, while the rest of the State, with a population of approximately 511,000, showed a total of 404; or, in other words, the total death rate over the age of 40 shows a ratio of 25.5 : 41.5 per 100,000 per annum for the Brisbane type of city and the rest of the State respectively.

Here, again, therefore, it might be suggested that whatever factor is operating manifests itself as a local factor that culls those persons liable to death from chronic nephritis at an age considerably earlier than they would otherwise die, and not as a new factor adding large numbers of deaths from chronic nephritis to the absolute total of the series.

As to such selective factors operating to produce kidney damage, the first that suggested itself was naturally scarlet fever.

The notifications for this disease in Queensland are considerably lighter than in several other States, though doubtless on account of the minor manifestations of rash, &c., in scarlet fever in the tropics, many cases otherwise notifiable are overlooked. When, however, the deaths from scarlet fever during the intense epidemics of 1890 and 1898 are considered in reference to the death rates for nephritis over the period 1890 to 1930, definite impressions appear to be made at the three periods at which nephritis manifests itself in relation to scarlet fever: that is to say, firstly, those cases which occur as a fatal nephritis complicating the attack of scarlet fever; secondly, those which are set up by an attack of scarlet fever, become chronic, and run their course, producing death approximately in five years; and, thirdly, those which set up initial kidney damage at the time of the attack of scarlet fever, and which, perpetuated by various causes, continue, finally to produce death 25 to 35 years after the attack.

Figures for deaths from chronic nephritis for the whole period of 40 years up to the present, show suggestive phases in this regard.

The fact that no similar series of figures is recorded for other States perhaps arises from the fact that those had their epidemic incidences of scarlet fever, to a degree comparable with the epidemics in Queensland quoted, at a period ten to twenty years earlier, and had already passed at the period under review from a stage of mass susceptibility into the present more general phase of adult mass immunity.

Various other prevalent disorders were discussed in this connexion without providing much information, with the exception of plumbism in childhood.

In the clinical series, the doubtful standards of diagnosis in use at different periods in Brisbane suggested the most rigid scrutiny of all cases claiming to have been "leaded" as children, and as a result there remained only an admittedly selected series for clinical examination; very gross evidences of plumbism were required before the diagnosis as such was admitted, and such cases of plumbism are not

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unnaturally followed by chronic nephritis, so that to some extent the clinical series begs the question.

Prior to the clinical examination of old cases—none new came to my observation—the history of plumbism in Queensland was reviewed in some detail, together with the various suggestions as to the sources of lead.

It was not found possible to relate the seasonal occurrence suggested by the monthly rate of admissions to hospital to the rainfall or to several other factors which had been suggested as important in etiology.

In connexion with the clinical observation of cases, 100 cases who showed definite signs and symptoms of chronic nephritis were made a series for particular examination, and comprise the following age groups: Under 10 years, 5; 10 to 20 years, 48; 20 to 30 years, 26; 30 to 40 years, 21. During the course of the investigation, 18 of the cases under observation died.

The various types of cases, case histories, and types of kidney involvement were set out and studied in detail. As results, a possible familial factor was suggested; a syphilitic factor was definitely indicated in cases with nephritis affecting successively several members of the family at much about the same age; and a close association with scarlet fever, measles, and diphtheria—three diseases often difficult to distinguish in Queensland—was also made out. It was considered permissible to suggest that all these matters were important factors of selectivity.

Of all etiological factors investigated, none was as commonly suggested as plumbism among 100 cases of the Brisbane series, 46 per cent. stating that they had been treated at an earlier period for "plumbism". In 30 of these cases hospital records indicated typical plumbism, and in 16 indefinite signs. Country cases of chronic nephritis had no related history.

In attempting to follow up from their origins cases of plumbism ending in chronic nephritis, records were examined carefully. The admissions for plumbism to the Brisbane Hospital for Sick Children from 1891 to 1931 are set out in Figure III. Specialized information is available only from 1915, but from this it was readily apparent that the crude figures in themselves were of little value. In each year there was a certain proportion of re-admissions, a number of doubtful cases, and many cases recognized to be indefinite and so recorded. These last vary from probable cases of lead poisoning to cases which almost certainly are not lead poisoning. Irrespective, however, of difficulties of diagnosis, there were certain cases in the series which were undoubtedly cases of plumbism. On the other hand, from the middle of 1930, when this investigation began, until it ended, not one case of frank plumbism associated with paralysis came under observation.

Among the disorders clouding the diagnosis of plumbism occasionally appeared mal-nutrition; vitamin deficiencies, such as scurvy or less obvious manifestations; congenital syphilis; and infantile paralysis.

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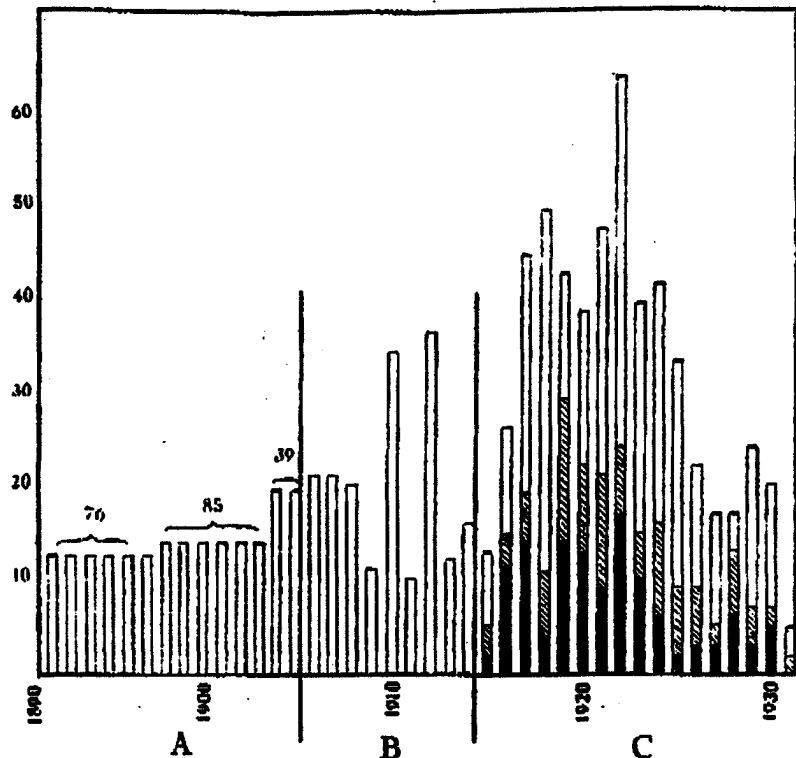


FIGURE III.

Crude admissions for Plumbism, Hospital for Sick Children, Brisbane, 1891-1931 showing :

- A. (1891-1905) Crude Average Admissions (estimated) ;
- B. (1906-1914) Crude Annual Admissions ;
- C. (1915-1931) Annual Admissions corrected to show new cases (black), doubtful cases (cross-hatched) and re-admissions, operation cases, &c. (white).

Of 80 severe and typical cases of plumbism in childhood selected from the records of the Hospital for Sick Children, it was only possible to get in touch with 28. In 19 of these, chronic nephritis was found at varying intervals after the recorded plumbism with paralysis. There was thus demonstrated here, as elsewhere, a definite relationship between the occurrence of marked plumbism and the subsequent occurrence of chronic nephritis.

The suggestive apparent relation between the violent epidemics of scarlet fever of 1890 and 1898 and the death rates from chronic nephritis from 1890 to 1930; the demonstration (incomplete as it was) of the importance of familial syphilis; the frequency of a history of familial nephritis; the very frequent history of mal-

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nutrition preceding plumbism (often evidenced and accompanied by pica); and so forth, are conformable with an assumption that plumbism may not act to any marked extent as an independent cause of chronic nephritis, but may be evidence that where for any reason familial or morbid influences have damaged the kidney, ingested lead hastens the incidence of death from chronic nephritis.

In advancing such an assumption, one was faced by the immediate necessity of admitting the accessibility and frequent ingestion of lead, presumably by a large number of persons in Brisbane, and as a corollary, with two further problems—(1) the source of the lead so ingested; and (2) the factors, if any, other than those indicated above, that determine the lead intake generally, and the occurrence of chronic or acute plumbism in individual cases.

All writers on lead poisoning are agreed as to the variable tolerance of individual members of the population for this metal, and to the fact that the degree of resistance is subject to fluctuation from many causes—for example, that it is to some extent a function of age, of sex, and of general health.

In 66 per cent. of the cases quoted, the illness commenced at four years or under; the disease was practically confined to children under nine years of age; and there was a marked concentration in the three to four years group. Closer investigation only served to reduce the average age of onset to from one and half to three years of age.

Investigations were made into the relation of nail-biting to the disease; to the importance of pica; to the amount of soluble white lead that could be determined in the environment; taken up on the surface of a moist palm; &c.

The three common sources of lead were—(1) lead arsenate on the only green vegetables readily accessible in Brisbane—cabbages and tomatoes; (2) white lead on houses, verandah railings, &c.; and (3) lead in plumbi-solvent waters.

Lead arsenate has only been commonly in use for fifteen years, while cases of lead poisoning have been reported for 40 years.

White lead paint is very generally distributed, and while this renders it the most accessible source of lead in poisonous quantities, it serves also to cast doubt as to its absolute significance, owing to the strict limitation of recorded plumbism to certain areas within the very wide range of the general distribution of white lead paint, and this is emphasized by the present absence of cases without material change in the environmental accessibility of white lead paint. On the other hand, a very strong chain of evidence incriminating white lead paint is recorded in individual cases, while indications in any other direction are extremely few.

In respect of *water supply*, the water of the Enoggera reservoir is plumbi-solvent, and there is a relationship, perhaps accidental, between the present apparent cessation of cases and the cessation of the supply of undiluted plumbi-solvent Enoggera water, while the

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diminution of cases and the diminution of the supply of soft water appear to be roughly coincident. On the other hand, the presence of cases from several towns in Queensland widely separated from Brisbane, and in particular the records of Ipswich, which, while producing cases, is supplied with water from Brisbane, but not from the Enoggera reservoir, militate against the assumption. On the other hand, the source of the lead in water, while admittedly speculative, cannot be disregarded without a considerable range of experiments, difficult to institute, but essential to conclusive evidence. (See Figure IV.)

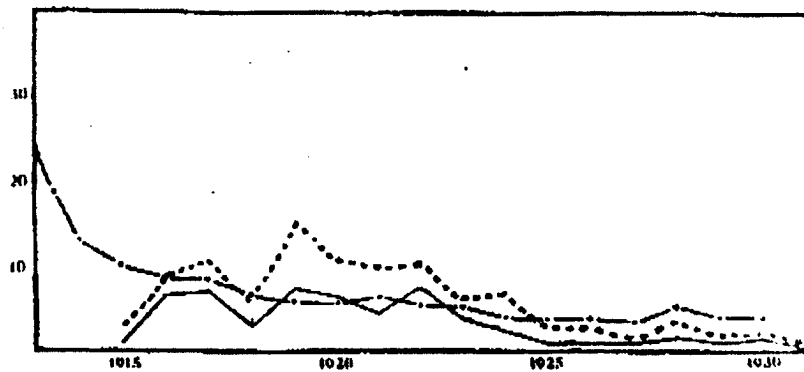


FIGURE IV.

New and suspected cases of Plumbism 1915-31 (inclusive) per 100,000 of population of Brisbane compared with percentage to total water supply of Enoggera (soft) Water.

Percentage of Enoggera Water to total — — — — — New cases of Plumbism
Doubtful cases of Plumbism - - - - -

The tentative conclusion reached is that the chronic nephritis common in Queensland arises, as elsewhere, as a result of many factors productive of kidney damage, and including principally familial weakness of the kidney fabric, damage produced by congenital syphilis, and conditions set up as the result of the acute or prolonged action of toxins derived from pathogenic organisms. In Brisbane, in cases where such predispositions exist, the concurrent ingestion of infinitesimal doses of lead from an undetermined source appears in a proportion of cases to precipitate a fatal issue many years earlier than would otherwise be the case, weeding out the great mass of chronic nephritics before they attain the age of 40. Since whatever factor is operating in Brisbane appears to be operative also in several other cities of the Brisbane type, the possibility of lead poisoning being a factor in these areas, though it does not figure in hospital records, cannot be entirely discounted, but must be investigated before findings can be conclusive.

The work on which these tentative findings are based is included in the full report submitted to the Director-General of Health, Canberra.