Water-borne Diseases*

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THE subject about which I have been asked to speak today is an all-inclusive one, involving directly and remotely a disturbance of function or structure of any organ, tissue, or fluid of the body by water. This is such an enormous task that the time allocated permits only an abbreviated discussion of the subject with emphasis on one phase in which we have been particularly interested.

During the latter part of the 19th century, enteric bacteriology became a reality. Between 1880, the time when Eberth described the etiologic agent of typhoid fever, and the first few years of the 20th century, many of the present-day enteric pathogens were recognized; but it was not until the latter part of the first decade of the 20th century that much was known about the epidemiology and control of these diseases. At this time water played a very important role in their spread.

With improvements in the sanitary handling of food, the use of modern treated water supplies, and the sanitary disposal of body wastes, a marked progressive reduction in the incidence of enteric disease and of carriers has taken place. Public water supplies have been improved and so closely guarded that water-borne outbreaks of enteric disease from this source are becoming a rarity. Of recent years those few outbreaks reported in the United States, in which public water supplies were incriminated,[1] have been traced to accidents or breaks in sanitation such as back siphonage, inadequate chlorination, cross-connections, surface pollution, defective well casings, and ruptured sewage mains contaminating the supply. This speaks well for the marked advances made in sanitary engineering.

Many elderly practising physicians in Iowa have informed me that in the early part of the 20th century they saw many more cases of typhoid fever than obstetrical cases and that treatment of cases of typhoid constituted the greater share of their practices. "In 1900 over 23,000 people in the United States died of typhoid fever[2] and more than 100,000 deaths were due to diarrhea-enteritis and dysenteries. In 1944, the deaths from typhoid fever were less than 600 and deaths from the other enteric diseases dropped to about 15,000." Today, few Iowa physicians see a case of typhoid. During 1947, 3,062 cases of typhoid fever were reported to the U. S. Public Health Service,[3] of which 46 were reported for Iowa. Up to September 1, 1948, 20 cases have been reported in Iowa all due to heretofore unrecognized carriers. Five new carriers were responsible for these cases. In so far as I can find, outbreaks of typhoid in Iowa during the past 20 years have been traced to food handling carriers. In several borderline instances either soft drinks, water, or food may have been the source. "For the past thirteen years not one case of typhoid has been traceable to a public water supply in Michigan," [4] according to the Michigan Department of Health.

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The importance of a water supply is so well fixed in the minds of the laity that when a case of typhoid is discovered, the tendency still remains for checking the water and milk supply for typhoid organisms before any consideration is given to the human carrier who transmitted the infection to the vehicle. In such instances, testing the water and milk in most instances is rarely warranted, except when epidemiologic evidence indicates.

During World War II, a marked increase in enteric and other diseases spread by water occurred in war-ravaged areas, save for England where safe water was maintained. Many water supplies and sanitary installations were bombed out, and in many instances replacements were impossible. With this breakdown and inadequate substitutions, typhoid fever, salmonellosis, and dysentery increased in alarming proportions. With the increase in cases, there was a proportionate increase in carriers. This increase in cases and carriers, lack of adequate sanitary disposal of body wastes, and food contamination led to marked pollution of wells, springs, and streams, resulting in a vicious cycle. So polluted became the streams that typhoid bacilli could be isolated from them and vaccination of human beings against typhoid was not entirely effective. Bathing in the streams added to the gravity of the situation. The finding of many carriers [5] following mild and asymptomatic typhoid and salmonellosis was not unusual.

When the improvised water supplies were chlorinated but without filtration, amebic dysentery and other intestinal parasitic diseases increased. It has been stated that cysts of *Entamoeba histolytica* resist ordinary chlorination [6] and that their removal can be accomplished by filtration. In some of our observations on embryonated ascaris ova it was noted that the embryos will resist 10 per cent formalin, and 10 p.p.m. chlorine for as long as four years under icebox storage. Apparently the shell membrane becomes impervious to these agents. To what extent other parasitic pathogens will resist chlorination needs further study.

In 1946, 32 outbreaks of water-borne disease were reported to the Public Health Service,[1] involving 4,512 cases with 2 deaths. Only 9 of these were traced to public water supplies while 22 were traced to private supplies and 1 to the use of a tidal basin for wading. In comparison with this, there were 318 outbreaks due to food products with 13,321 cases and 17 deaths reported.

Private water supplies continue to remain a source of minor outbreaks. The above figures reported I feel represent but a small portion of the actual occurrence of disease from private water supplies. Over a period of some thirty years, approximately 70 per cent of the farm wells in Iowa were found basically unsafe and were so proved on analysis of the water in our laboratories. Not only are these supplies contaminated from nearby privies, but they are subject to pollution via defective platforms and casings, from rains, and by fowls and animals permitted to run at large in the yard where the wells are located.

Infections by organisms of the *Salmonella* group are quite frequent among farmers whose water supplies are unsafe. Whether these infections can be traced solely to water, requires further detailed epidemiologic study of the entire farm population as well as of the water. It appears from meager epidemiologic data and cultures of stools in isolated instances that certain proteus and paracolon species found in farm wells in Iowa are pathogenic and are capable of causing diarrhea. Here again, more detailed studies are essential to eliminate animal sources of infection.

Although water supplies have not been definitely incriminated as a source of poliomyelitis, epidemiologic investi-

gations suggest that contaminated streams and improperly operated swimming pools may serve as a focus of infection. Poliomyelitis virus has been isolated from sewage, and it has been shown that the virus will resist ordinary doses of chlorine.

Some of the less common diseases that have been transmitted via water are those caused by chemical poisons such as lead, arsenic, and cadmium; poisoning from water-borne plants and shellfish; a wide variety of parasitic diseases; tularemia; brucellosis, shigella infections, epidemic jaundice, and Weil's disease. The status of minerals such as chlorides and manganese needs further study. The status of cyanogen compounds in water, traceable to algae, likewise needs further study.

A disease commanding a great deal of attention recently is that of fluorosis or mottling of the tooth enamel. It appears fairly well substantiated that dental caries is definitely decreased in areas where the well water supplies contain fluorine in concentrations of 1 p.p.m., and that supplies containing increased amounts above 1.2 p.p.m. show progressive increases in mottling. Control studies now being conducted in cities in which 1 p.p.m. fluorine is being added to the municipal water supply should give more definite information.

Cyanosis or methemoglobinemia in infants due to nitrates in farm well water continues to be a problem in Iowa since its recognition by Comly.[7] The actual incidence of this condition is unknown, due to lack of adequate reporting. Our attention is periodically called to cases in which parents of cyanotic babies or physicians are interested in tests of the well water involved as well as that of neighbors who have deep wells. Johnson and his associates [8] have reported the incidence of nitrate nitrogen in various types of private wells in Iowa. These data clearly indicate that a hazard to infant health exists in our rural areas from this source. Gilbert Kelso, Principal Water Analyst, and his staff in the Iowa State Hygienic Laboratory, have compiled data for 1½ years on 2,313 water supplies from private wells submitted for routine analysis. Of this number, 1,943 were less than 100 ft. deep and 645, or 28 per cent, contained NO_3N (nitrate nitrogen) above 20 p.p.m. Of 370 wells over 100 ft. deep 13, or 3.5 per cent, contained NO_3N above 20 p.p.m.

In another statistical study, involving 3,833 private wells, including 874 wells having above 20 p.p.m. NO_3N, 112, or 13 per cent, did not contain coliform organisms by standard methods of examination. Seven hundred and sixty-two, or 87 per cent, of these water supplies containing above 20 p.p.m. NO_3N also gave positive coliform tests. Therefore, it seems logical to conclude that the standard tests for coliform determination will rule out most of the nitrate-bearing waters as unsafe.

Of 1,475 well supplies containing from 0 to 10 p.p.m., NO_3N, 819 were under 50 ft. in depth, 304 were between 50 and 100 ft., 144 between 100 and 150 ft., 90 between 150 and 200 ft. and 118 above 200 ft.

Of 181 well supplies containing 11-20 p.p.m. NO_3N, 144 were under 50 ft., 32 were between 50 and 100 ft., 4 were between 100 and 150 ft., and 1 between 150 and 200 ft.

Of the 2,313, no wells over 200 ft. showed more than 10 p.p.m. NO_3N. In general, it may be said that the deeper the well the less likelihood there is that the water will contain NO_3N in sufficient quantity to cause cyanosis.

Out of 69 cases of cyanosis in infants traceable to water during this same period, 27 supplies contained 50 p.p.m. NO_3N or less, while 42 supplies contained from 51 to 801-plus p.p.m. Of the water supplies involving these cases, 53 were bacteriologically unsafe whereas 16 contained no coliform bacteria.

You may ask what concentration of NO_3N in water is capable of producing cyanosis in infants? This question I feel can be answered only after more complete statistical, clinical, and epidemiological data have been accumulated. From data submitted with well water specimens to our laboratory, cyanosis was associated with use of water in which at the time of the analysis 20 p.p.m. were found in 4 instances, 15 p.p.m. in 2 instances, 10 p.p.m. in 1, 9 p.p.m. in 1, 5 p.p.m. in 1, 2 p.p.m in 1, 0.4 in 1, and in 2 instances no detectable nitrates. Whether the cyanosis in the latter instances were due to congenital heart disease or other causes I cannot say. However, we are well aware of the fact that marked changes in nitrate levels in water will vary on periodic testing, from causes which we cannot define. It is possible that chemical fertilization of the soil and rains play an important role in the varying nitrate content of rural wells. In one instance, at the time of cyanosis in an infant the well water was found to contain 70 p.p.m. NO_3N whereas two weeks later analysis of another sample showed only 10 p.p.m.

There are apparently many problems which remain unsolved in relation to this disease. One of the mysteries is why cyanosis does not occur in all babies fed on artificial formulae diluted with high nitrate-bearing waters. It has been quite well established that when nitrate compounds in water are converted by bacterial action in the intestinal tract of certain infants to nitrites, the latter are absorbed into the blood, resulting in the oxidation of hemoglobin to methemoglobin, thus leading to cyanosis. A number of controversial theories have been advanced as to why certain infants who ingest these supplies do not contract the disease. These theories I will not discuss here as they are highly controversial.

Another question which might be asked is, whether adults suffer from the presence of the nitrates in water. The answer at this time is not definitely known. Diarrhea has been noted in some adults at the same time their offspring have diarrhea and cyanosis.

In spite of the enviable record made in reducing water-borne diseases, as health educators we fail in many respects to get over to the public the necessity of following accepted sanitary practices, which, if followed to the letter would for all practical purposes eliminate water-borne diseases. To maintain this record we must make certain that persons in charge of operating water plants and those who examine water for purity must be thoroughly trained and fully aware of the responsibilities of their positions. The job has been well done; let us strive to perfect it. We must continue to guard closely our water supplies in order to maintain the excellent record established.

CONCLUSIONS

1. Water-borne outbreaks of disease from public water supplies have been practically eliminated in the United States save for those due to accidents and breaks in sanitation.

2. Data are presented showing the relationship of nitrates in rural water supplies to the depths of the wells.

3. Sixty-nine cases of infant cyanosis associated with NO_3N in rural water supplies are reported.

4. There appears to be close correlation between the NO_3N content of rural water supplies and their coliform content.

5. Accumulated data suggest that in cyanosis emergencies, water from properly constructed drilled wells over 200 ft. in depth may be tentatively substituted in infant formulae until safety tests can be adequately checked.

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Medical Care Section Created in California School of Public Health

A Section in Medical Care Administration has been established by the University of California School of Public Health at Berkeley. The new curriculum complements the school's existing programs in hospital management and public health administration. Qualified graduate students may major in medical and hospital administration courses leading to the M.P.H. degree. Edward S. Rogers, M.D., dean of the school, heads the joint division and E. Richard Weinerman, M.D., is developing the new medical care curriculum. Drs. Sydney S. Norwick, Berkeley, and Dean A. Clark, new administrator of the Massachusetts General Hospital in Boston, are part-time visiting lecturers. A small research staff, headed by Charlotte F. Muller, Ph.D., is making medical economics field studies. The section will serve as a source for consultation in problems of medical care, offering technical aid and other help to interested persons or organizations and as a repository of factual data in medical economics.