of barium have been made in animals to which barium had been repeatedly administered for long periods.

No study appears to have been made of the amounts of barium that may be tolerated in drinking water or of effects from prolonged feeding of barium salts from which an acceptable water standard may be set. A rational basis for a water standard may be derived from the threshold limit of 0.5 mg Ba/m³ air set by the American Conference of Governmental Industrial Hygienists (*10*) by procedures that have been discussed (*11*). By making reasonable assumptions as to retention of inhaled barium dusts and absorption from the intestine (and including a safety factor) 1 mg/1 is derivable as a limit that should constitute a "no effect" level in water. Concentrations of barium in excess of 1 mg/1 are grounds for rejection of the supply because of the seriousness of the toxic effects of barium on the heart, blood vessels, and nerves.

LIMITS AND RANGES RELATIVE TO BARIUM STANDARD

1. Average U.S. urban air concentration ..... 0.025 ug Ba/m³ (*12*)
2. Surface and ground waters ..... Not usually present
3. Concentrations harmful to fish 400 mg/1 (*13*)
4. Concentrations harmful to Daphnia Magna 30 mg/1 (*14*)
5. Barium content of Brazil nuts (Only food with barium in considerable amounts) 0.06–0.3% (*15*)
6. Concentrations of various natural anions required to reach solubility product of barium salts:

	Solubility product moles/l at 25° C	*Milligrams anion required per liter to attain solubility product at 1 mg barium*	
$BaSO_4$	1×10^{-10}	1.3	SO_4
$BaCO_3$	8×10^{-9}	66	CO_3
BaF_2	1.7×10^{-6}	9000	F

The solubility of relatively insoluble barium salts such as the sulfate may be increased in the presence of iron, magnesium, and aluminum salts, so that in the presence of the latter, calculations of solubility from the solubility product may not apply.

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CADMIUM

As far as is known, cadmium is biologically a nonessential, nonbeneficial element. On the other hand, cadmium is recognized to be an element of high toxic potential. Slight cognizance has been taken of this in water quality control as evidenced by the fact that only the USSR, and in the United States, North Dakota, have set a permissible water standard for cadmium, 0.1 mg/1 by the former and 0.4 mg/1 as a tentative value by the latter. Recognition of the serious toxic potential of cadmium when taken by mouth is based on: (*a*) poisoning from cadmium-contaminated food (*1*) and beverages (*2*); (*b*) epidemiologic evidence that cadmium may be associated with renal arterial hypertension under certain conditions (*3*); (*c*) long-term oral toxicity studies in animals.

The possibility of cadmium being a water contaminant has been reported in 1954 (*4*); seepage of cadmium into ground water from electroplating plants has resulted in cadmium concentrations ranging from 0.01 to 3.2 mg/1. Other sources of cadmium contamination in

water arise from zinc-galvanized iron in which cadmium is a contaminant.

Several instances have been reported of poisoning from eating substances contaminated with cadmium. A group of school children were made ill by eating popsicles containing 13 to 15 mg/1 cadmium (*1*). This is commonly considered the emetic threshold concentration for cadmium. It has been stated (*5*) that the concentration and not the absolute amount determines the *acute* cadmium toxicity; equivalent concentrations of cadmium in water are likewise considered more toxic than equivalent concentrations in food probably because of the antagonistic effect of components in the food.

Chronic oral toxicity studies in rats, in which cadmium chloride was added to various diets at levels of 15, 45, 75, and 135 ppm cadmium, showed marked anemia, retarded growth, and in many instances death at the 135 ppm level. At lower cadmium levels, anemia developed later; only one cadmium-fed animal had marked anemia at the 15 ppm level. Bleaching of the incisor teeth occurred in rats at all levels except in some animals at 15 ppm. A low protein diet increased cadmium toxicity. A maximal "no effect" level was thus not established in the above studies (*6*). A dietary relation to cadmium toxicity has been reported by others (*7*).

Fifty ppm cadmium administered as cadmium chloride in food and drinking water to rats resulted in a reduction of blood hemoglobin and lessened dental pigmentation. Cadmium did not decrease experimental caries (*8*).

In a study specifically designed to determine the effects of drinking water contaminated with cadmium, five groups of rats were exposed to drinking water containing levels from 0.1 to 10 mg/1. Although no effects of cadmium toxicity were noted, the content of cadmium in the kidney and liver increased in direct proportion to the dose at all levels including 0.1 mg/1. At the end of one year, tissue concentrations approximately doubled those at six months. Toxic effects were evident in a three-month study at 50 mg/1 (*9*).

Thus, all levels of dietary cadmium so far tested have shown cadmium accumulation in the soft tissues down to and including 0.1 mg/1 (in drinking water). Because the presence of minute amounts (5×10^{-6}M) of cadmium in rat liver mitochondria has been shown (*10*) to interfere with an important pathway of metabolism (uncoupled oxidative phosphorylation), and because suspicion has been cast on the presence of minute amounts of cadmium in the kidney as responsible for adverse renal arterial changes in man (*3*), concentrations of cadmium in excess of 0.01 mg/1 in drinking water are grounds for rejection of the supply.

Further evidence that a concentration of 0.01 mg/l can be tolerated is found in a study made on long-continued cadmium absorption, without history of symptoms, in individuals whose drinking water had an average cadmium content of 0.047 mg/l (*11*).

LIMITS AND RANGES RELATIVE TO CADMIUM WATER STANDARD

U.S. average urban air concentration (1954–56) (*12*)	0.005 ug Cd/m³
U.S. range urban air concentration (1954–56) (*12*)	0–0.599 ug Cd/m³
Cd concentration lethal to minnows (*13*)	1,000 mg/l
Cd concentration lethal to stickleback (*14*)	0.20 mg/l
Cd concentration in tobacco	Not known
Cd concentration in foods	Not known

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CARBON CHLOROFORM EXTRACT

The use of Carbon Chloroform Extract (CCE) (*1*) as a practical measure of water quality and as a safeguard against the intrusion of

excessive amounts of potentially toxic material into water has been discussed elsewhere (*2*). It is proposed as a technically practical procedure which will afford a large measure of protection against the presence of undetected toxic materials in finished drinking water.

The most desirable condition is one in which the water supply delivered to the consumer contains no organic residues. Residual organic matter in the treated water clearly represents man-made or natural pollutants which have not been removed in water treatment or material such as lubricants inadvertently introduced by the water plant. In view of a general inability to clearly define the chemical and toxicological nature of this material, it is most desirable to limit it to the lowest obtainable level. Analysis of data available indicates that water supplies containing over 200 micrograms CCE/1 of water represent an exceptional and unwarranted dosage of the water consumer with ill-defined chemicals. It is recommended that 200 ug CCE/1 be the limiting concentrations in drinking water.

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2. Ettinger, M. B. A proposed toxicological screening procedure for use in the water works. J. Am. Water Works A.52: 689–694, 1960.

CHLORIDE, SULFATE, AND DISSOLVED SOLIDS

The importance of chloride, sulfate, and dissolved solids as they affect water quality hinges upon their taste and laxative properties. There is evidence that excessive amounts of these constituents cause consumer reactions which may result in individual treatment or rejection of the supply. Therefore, limiting amounts for these chemical constituents have been included in the Standards. The bases for developing these limits are described below.

Taste

The literature contains a number of reports on the taste threshold of various salts. Whipple, (*1*) using a panel of 10 to 20 persons, found the range of concentration of various salts detected as shown in Table 1. Richter and MacLean (*2*) studied the response of a larger panel to sodium chloride in distilled water. Table 2 summarizes their results.

Lockhart, Tucker, and Merritt (*3*) also studied the taste threshold of the ions in distilled water by studying the effect of ions in water on the flavor of brewed coffee. Using a triangular test with panels of 18 or more, they found results which are summarized in Table 3.

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In the Triangular taste test, the panel members are asked to taste three samples. Two of the samples may contain either the salt being tested or distilled water, while the third is different from the other two. The panel member is asked to identify the odd one. Using this test procedure, the threshold concentration is arbitrarily defined as the concentration at which the number of correct separations is 50 percent above the chance probability of one-third correct separations. i.e., when two-thirds of the panel make the separations correctly.

The results shown in Table 1 and Table 3 are in surprisingly good agreement, considering the difference in methods used. The Richter and MacLean study found taste thresholds considerably below those of the other two studies. They support reasonably well the recommended limits of 250 mg/1 for chloride and sulfates and 500 mg/1 for total solids.

It should be emphasized that there may be a great difference between a detectable concentration and an objectionable concentration of the neutral salts. The factor of acclimatization is particularly important. More than 100 public supplies in the United States provide water with more than 2,000 mg/1 of dissolved solids. Newcomers and casual visitors would certainly find these waters almost intolerable and, although some of the residents use other supplies for drinking, many are able to tolerate if not to enjoy these highly mineralized waters.

Relatively little information is available on consumer attitudes toward mineralized water. In this connection, the findings of a survey made by the California State Department of Public Health (*4*) showed that in five communities where the public supplies were highly mineralized, about 40 percent of the families surveyed purchased bottled water and about 50 percent stated they were dissatisfied with the water. These supplies had dissolved solids contents in the range of 500 to 1,750 mg/1. Calcium, sulfate, and magnesium were the dominant ions present, with sulfate concentrations in the range of 300 to 700 mg/1.

The taste threshold for magnesium is said to be 400–600 mg/1 (*5*).

Laxative Effects

Both sodium sulfate and magnesium sulfate are well known laxatives. The laxative dose for both Glauber salt ($Na_2SO_4 \cdot 10H_2O$) and Epsom salt ($MgSO_4 \cdot 7H_2O$) is about two grams. Two liters of water with about 300 mg/1 of sulfate derived from Glauber salt, or 390 mg/1 of sulfate from Epsom salt, would provide this dose. Calcium sulfate is much less active in this respect.

This laxative effect is commonly noted by newcomers and casual users of waters high in sulfates. One evidently becomes acclimated to use of these waters in a relatively short time.

The North Dakota State Department of Health has collected information on the laxative effects of water as related to mineral quality. This has been obtained by having individuals submitting water samples for mineral analysis complete a questionnaire which asks about the taste and odor of the water, its laxative effect (particularly on those not accustomed to using it), its effect on coffee, and its effect on potatoes cooked in it.

Peterson (*6*) and Moore (*7*) have analyzed part of the data collected, particularly with regard to the laxative effect of the water.

Peterson found that, in general, the waters containing more than 750 mg/l of sulfate showed a laxative effect and those with less than 600 mg/l generally did not. If the water was high in magnesium, the effect was shown at lower sulfate concentrations than if other cations were dominant. Moore showed that laxative effects were experienced by the most sensitive persons, not accustomed to the water, when magnesium was about 200 mg/l and by the average person when magnesium was 500–1,000 mg/l.

Moore analyzed the data as shown in Table 4. When sulfates plus magnesium exceed 1,000 mg/l or dissolved solids exceed 2,000 mg/l, a majority of those who gave a definite reply indicated a laxative effect.

Other Effects

Highly mineralized water affects the quality of coffee brewed with it. Lockhart, Tucker, and Merritt (*3*) found that from 400 to 500 mg/l of chlorides or 800 mg/l of sulfate as $MgSO_4$ affected the taste of coffee. Gardner (*8*) studied the effect of ions in water on the brewing time of drip coffee and hence on the quality of the product since prolonged contact with the grounds makes the coffee bitter. Sodium had a distinct deleterious effect.

At high enough mineral concentration, water becomes completely unusable for drinking. These concentrations are in the range above 5,000 mg/l and need not be considered here.

Conclusion

It is recommended that waters containing more than 250 mg/l of chlorides or sulfates and 500 mg/l of dissolved solids not be used if other less mineralized supplies are available. This is influenced primarily by considerations of taste. Cathartic effects are commonly experienced with water having sulfate concentrations of 600 to 1,000 mg/l, particularly if much magnesium or sodium is present. Although waters of such quality are not generally desirable, it is recognized that a considerable number of supplies with dissolved solids in excess of the recommended limits are used without any obvious ill effects.

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TABLE 1.—*Range of concentration of various salts detected by taste in drinking water by panel of 10 to 20 persons*

Salt	Concentration detected—mg/l			
	Median		Range	
	Salt	Anion	Salt	Anion
KCL	525	250	350–600	167–286
NaCl	300	182	200–450	121–274
$CaCl_2$	250	160	150–350	96–224
$MgCl_2$	500	372	200–750	149–560
Sea water		[1] 300		[1] 150–600
$NaSO_4$	350	237	250–550	169–372
$CaSO_4$	525	370	250–900	177–635
$MgSO_4$	525	419	400–600	320–479

[1] In terms of mg/l chloride.

Source: Whipple, G. C., The value of pure water. Wiley (1907).

TABLE 2.—*Taste threshold concentrations of panel of 53 adults for NaCl*

	Concentrations mg/l					
	Mean		Median		Range	
	NaCl	Cl	NaCl	Cl	NaCl	Cl
Difference from distilled water noted	160	97	100	61	70– 600	42– 364
Salt taste identified	870	530	650	395	200–2,500	120–1,215

TABLE 3.—*Taste threshold concentration of salt and ions in water*

	Threshold concentration—mg/l		
	Salt	Cation	Anion
NaCl	345	135	210
KCl	650	340	310
$CaCl_2$	347	125	222
$MgSO_4$	500	100	400
$NaHCO_3$	1,060	290	770

Source: Lockhart, E. E., Tucker, C. L., and Merritt, M. C. The effect of water impurities on the flavor of brewed coffee, Food Research, *20*, 533–605 (1955).

TABLE 4.—*Solids and ion concentration of wells as related to presence or absence of laxative effects*

Determination	Range mg/l	Number of wells in range	Laxative		Effects present not stated	Percent of yes answers [1]
			Yes	No		
Total dissolved solids	0–1,000	51	5	37	9	12
	1,000–2,000	72	12	45	15	21
	2,000–3,000	62	25	21	16	54
	3,000–4,000	30	13	11	6	54
	over 4,000	33	14	4	15	78
Magnesium plus sulfate	0–200	51	9	34	8	21
	200–500	45	7	27	11	21
	500–1,000	56	11	38	17	28
	1,000–1,500	36	18	10	8	64
	1,500–2,000	14	6	4	4	60
	2,000–3,000	21	13	3	5	81
	over 3,000	14	5	1	8	83
Sulfate	0–200	56	10	36	10	22
	200–500	47	9	28	10	24
	500–1,000	56	13	26	17	33
	1,000–1,500	34	16	10	8	62
	1,500–2,000	16	9	4	3	69
	2,000–3,000	20	9	3	8	75
	over 3,000	8	3	0	5	100

[1] This percentage is based only on the total of yes and no answers. It is probable that a large proportion of the wells for which no statements were made were not regularly used as water supplies.

Source: Moore, Edward W., Physiological effects of the consumption of saline drinking water, a progress report to the Subcommittee on Water Supply of the Committee on Sanitary Engineering and Environment, National Research Council (1952).

CHROMIUM

The limit of 0.05 mg/1 for chromium as hexavalent chromium ion appearing in the U.S. Public Health Service 1946 Drinking Water Standards was based on the lowest amount analytically determinable at the time it was established. At present, the level of chromate ion that can be tolerated by man for a lifetime without adverse effects on health is unknown. A family of four individuals is known to have drunk water for periods of 3 years at a level as high as 1 mg chromate/1 without known effects on their health, as determined by a single medical examination (*1*). The family continued to drink the water which, when sampled later, contained

25 mg/1. No continued medical observation of these individuals was made.

When inhaled, chromium is a known cancerigenic agent for man (*2*, *3*). It is not known whether cancer will result from ingestion of chromium in any of its valence forms. According to Fairhall (*4*), trivalent chromium salts show none of the toxicity of the hexavalent form, particularly the highly insoluble salts. Trivalent chromium moreover, is believed not to be of concern in drinking water supplies.

Chromium is not known to be either an essential or beneficial element in the body.

The most recent study by MacKenzie, Byerrum, et al. (*5*) was designed to determine the toxicity of chromate ion (and chromic ion) at various levels in the drinking water of rats. This study, like a number of previous ones, showed no evidence of toxic response after 1 year at levels from 0.45 to 25 mg/1 by the tests employed, viz., body weight, food consumption, blood changes, and mortality. However, significant accumulation of chromium in the tissues occurred abruptly at concentrations above 5 mg/1. Unfortunately, no study was made of the effect of chromate on a cancer-susceptible strain of animal. It would appear, however, from this and other studies of toxicity (*6*, *7*, *8*), that a concentration of 0.05 mg/1 is sufficiently low to cause no effect on health.

The possibility of dermal effects from bathing in water containing 0.05 mg/1 would likewise seem remote, although chromate is a recognized and potent sensitizer of the skin (*9*).

Chromium is not known to be a common or significant element in food sources. That which may be found in small quantities in foods is in trivalent form, is usually adventitious, and arises chiefly from cooking in stainless-steel ware. Neither the amounts nor the assimilability are known to be of any hygienic significance (*8*, *11*).

LIMITS AND RANGES RELATIVE TO CHROMIUM WATER STANDARDS

Threshold range for color (*12*)	1.4–11 mg/1
Threshold range for taste (*12*)	1.4–25 mg/1
U.S. urban air conc'n range (1954–56) (*13*)	0–0.29 ug/m³
Average urban air conc'n (1954–56) (*13*)	0.007 ug/m³
Chromium content of cigarette tobacco (*14*)	1.4 ug/cigarette
Chromium in foods cooked in stainless-steel ware (*9*)	0–0.35 mg/100 g
Chromate conc'n toxic to fish (*15*, *16*, *17*)	5–200 mg/1
Chromate conc'n toxic to Daphnia Magna (*17*)	0.05 mg/1
Chromate conc'n range in surface water (*12*)	0–2.3 mg/1

CALCULATED MAXIMAL DAILY INTAKE OF CHROMIUM FROM VARIOUS SOURCES

(Approximate Values)

Food, cooked in stainless-steel ware	10–25 ug
Water	2 ug
Air	0.3 ug
Cigarettes	10–15 ug

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COPPER

In the Public Health Service 1942 Drinking Water Standards, the permissible concentration of copper in drinking water was raised from 0.2 mg/1 to 3.0 mg/1.

Copper is an essential and beneficial element in human metabolism, and it is well known that a deficiency in copper results in nutritional anemia in infants. The daily requirement for adults has been estimated to be 2.0 mg (*1*). The children of preschool age require about 0.1 mg daily for normal growth. The average daily urinary excretion is in the order of 1.0 mg, the remainder being eliminated in the feces. Since the normal diet provides only a little more than is required, an additional supplement from water would ensure an adequate intake. The distribution of copper in the body is fairly uniform, except for the liver where it appears to accumulate.

Copper imparts some taste to water but individuals vary in the acuity of their taste perception and the detectable range varies from 1–5 mg/1 (*2*). Small amounts are generally regarded as nontoxic but large doses may produce emesis and prolonged oral administration may result in liver damage.

Inasmuch as copper does not constitute a health hazard but imparts an undesirable taste to drinking water, it is reasonable to establish the concentration of 1.0 mg/1 as the recommended limit.

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CYANIDE

The U.S. Public Health Service Drinking Water Standards for 1946 contain no limit for cyanide. Since 1946, standards have been developed for cyanide by other agencies as shown in the following tabulation.

Standard set by	*Limit for cyanide mg/1*
International Standards for Drinking Water, Geneva (1958)	0.01
Netherlands (1959)	0.01
USSR Standard (1951)	0.2
Ohio Water Pollution Control Board (1952)	0.15
Adv. Bd. Lake Erie-Ontario Sect. I.J.C. (1953)	0.1
N.Y. Water Pollution Control Bd. (1952)	0.1
Pacific N.W. River Basin (1952)	0.05

The cyanide standards appear to be based on the toxicity for fish and not for man, as is shown by a comparison that follows of the safe, toxic, and lethal doses for fish and for man. Cyanide in reasonable doses (10 mg or less) is readily converted to thiocyanate in the body. Usually lethal toxic effects occur only when the detoxifying mechanism is overwhelmed.

Oral toxicity of cyanide for man

Dosage	Response	Literature citations
2.9–4.7 mg/day	Noninjurious	(5)
10 mg, single dose	Noninjurious	(6)
19 mg in water	Calculated from threshold limit for air to be safe	(7)
50–60 mg, single dose	Fatal	(8)

Toxicity of cyanide for fish

Cyanide in mg/l	Time of exposure	Fish species	Response	Literature citations
0.05	120 hours	Trout	Death	(1)
0.1–0.2	1–2 days	do	do	(2)
0.126	170 minutes	do	Overturned	(3)
0.176		Bluegills, Sunfish	Toxic limit	(3)
1.0	20 minutes	Trout	Death	(1)
10.0	90 minutes	Carp	do	(4)
0.02	27 days	Trout	Survival	(1)
0.4	96 hours	Bluegills	do	(3)
0.5	96 hours	Bullheads	do	(3)

Because proper treatment will reduce cyanide levels to 0.01 mg/1 or less, it is recommended that concentrations in water be kept below 0.01 mg CN/1.

For the protection of the health of human populations, concentrations above 0.2 mg CN/1 constitute ground for rejection of the supply. This limit should provide a factor of safety of approximately 100 and is set at this level because of the rapidly fatal effect of cyanide. Proper chlorination under neutral or alkaline conditions will reduce cyanide to a level below the recommended limit. The acute oral toxicity of cyanogen chloride, the chlorination product of hydrogen cyanide, is approximately one-twentieth that of hydrogen cyanide (*9*).

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FLUORIDE

Fluoride in drinking water will prevent dental caries. When the concentration is optimum, no ill effects will result and caries rates will be 60–65 percent below the rates in communities using water supplies with little or no fluoride (*1, 2*).

Excessive fluoride in drinking water supplies produces objectionable dental fluorosis which increase with increasing fluoride concentration above the recommended upper control limits.[1] In the United States, this is the only harmful effect observed to result from fluoride found in drinking water (*3, 4, 5, 6, 7, 8, 9*). Other expected effects from excessively high intake levels are: (a) bone changes when water containing 8–20 mg fluoride per liter (8–20 ppm) is consumed over a long period of time (*5*); (b) crippling fluorosis when 20 or more mg of fluoride from all sources is consumed per day for 20 or more years (*10*); (c) death when 2,250–4,500 mg of fluoride (5,000–10,000 mg sodium fluoride) is consumed in a single dose (*5*).

The optimum fluoride level for a given community depends on climatic conditions because the amount of water (and consequently the amount of fluoride) ingested by children is primarily influenced by air temperature (*11, 12, 13, 14*). Many communities with water supplies containing less fluoride than the concentration shown as the lower limit for the appropriate air temperature range[1] have provided fluoride supplementation (15, 16, 17). Other communities with excessively high natural fluoride levels have effectively reduced fluorosis by partial defluoridation and by change to a water source with more acceptable fluoride concentration (*18, 19*).

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IRON

Both iron and manganese are highly objectionable constituents in water supplies for either domestic or industrial use. The domestic consumer complains of the brownish color which iron imparts to laundered goods. Iron appreciably affects the taste of beverages (*1*).

The taste which iron imparts to water may be described as bitter and astringent. Individuals vary in their acuity of taste perception,

and it is difficult to establish a level which would not be detectable for the majority of the population. A study by the Public Health Service (*2*) indicates that the taste of iron may be readily detected at 1.8 mg/l in spring water and at 3.4 mg/l in distilled water.

The daily nutritional requirement is 1 to 2 mg but intake of larger quantities is required as a result of poor absorption. Diets contain 7 to 35 mg per day and average 16 (*3*). The amount of iron permitted in water by quality control to prevent objectionable taste or laundry staining (as much as 0.3 mg/l) constitutes only a small fraction of the amount normally consumed and is not likely to have any toxicologic significance.

Whereas the U.S. Public Health Service 1946 Drinking Water Standards set a limit of 0.3 mg/l for iron and manganese combined, it is recommended that a limit be established for each and that the concentration of iron be limited to 0.3 mg/l.

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LEAD

Lead taken into the body can be seriously injurious to health, even lethal, if taken in by either brief or prolonged exposure. Prolonged exposure to relatively small quantities may result in serious illness or death. Lead taken into the body in quantities in excess of certain relatively low "normal" limits is a cumulative poison. Poisoning may result from an accumulation in the body of lead absorbed in sufficient quantities from any one or all of three common sources: food, air, and water, including that used in cooking and in beverages. A fourth, but variable source of intake is inhaled tobacco smoke. Except in certain occupational conditions, absorption of lead through the skin is not of general public health importance.

The total amount of lead taken into the body from these sources as modified by absorption and elimination, determines whether the sources of exposure have been excessive and produce poisoning, or may be tolerated without effect throughout a lifetime.

The daily intake of lead that may be tolerated without effect throughout each decade of life is not precisely known, but a value may be determined from the following information.

1. The amount of lead ingested in food and beverage by adults in good health in various parts of the United States has been shown by

Kehoe and associates (*1*) to vary from less than 0.1 mg to more than 2.0 mg/day with a mean value of about 0.32 mg/day. At these levels, excretion keeps pace with intake, and if any accumulation of lead occurs it is intermittent and of no hygenic significance.

2. When, under experimental condition, the daily intake of lead from all sources amounted to 0.5–0.6 mg over a long period of time (1 year or more), a small amount is retained in normal healthy adults but produced no detectable deviation from normal health. Indirect evidence from industrial workers exposed to known amounts of lead for long periods was consistent with these findings (*2*).

3. Appreciable increases in the daily intake of lead above 0.6 mg daily result in body accumulation at rates that increase as the daily dose increases. Extrapolations from data from balance experiments over a 5-year period indicate, but do not prove, that an intake appreciably in excess of 0.6 mg/day will result in the accumulation of a dangerous quantity of lead in the body during a lifetime.

4. The intake of lead from food sources is probably approaching an irreducible minimum; on the other hand, the number of sources and the extent of lead exposure are increasing. The atmosphere is one of these. Over the past decade, the amount of atmospheric lead in many cities has increased more than tenfold, from a few tens of micrograms (ug) per cubic meter (m^3) of air to more than 15 ug/m^3 in some cities on repeated occasions (*3*). The national average for urban atmosphere is presently 1.4 ug/m^3. Wide variations in these values exist throughout the nation because the sources are largely unregulated and are increasing at different rates in different areas from vehicular traffic. If the average daily intake of air of an adult is 20 cubic meters, then the daily addition to the body burden of lead from the atmosphere could be of the order of several micrograms to a few tens of micrograms, depending on the location. This assumes a modest 10 percent retention of that which the individual inhales.

5. The amount of lead in cigarette tobacco smoke has been reported (*4*) to be as high as 0.3 ug/puff. In a heavy smoker, a few micrograms per day could be added to the lead body burden assuming 10 percent retention of the total smoke inhaled.

Foods contain lead in widely varying amounts because of the natural and unavoidable content of lead in foods, the inevitable contamination with lead that results incidentally from processing and packaging, and the residue from insecticidal spraying and dusting. Certain foods, in particular those which are more seriously and unavoidably contaminated, are required by law to contain by analysis no more than a prescribed concentration of lead. The foods under regulation make up a relatively small portion of the average normal diet. Conse-

quently, only partial control is exercised over the lead intake from food sources. The foods that contribute the greater portion of the diet contain concentrations of lead which are considered to be normal (that is, natural or incidental) but in any case unavoidable (under 0.2 ppm, and usually well under 0.1 ppm). The total intake of lead from these foods is governed by the quantity and quality of the food ingested, and by contamination with lead in the handling and preparation of the food.

The lead concentration in surface and in ground drinking water sources in the United States in 1940 ranged from traces to 0.04 mg/1, averaging 0.01 mg/1. It is now not uncommon to find the lead content of water in urban supplies to be from one-half to one-fifth this value, provided the water is not stored in tanks painted with oil-base lead paint (Type I) or provided that the piping and fixtures are not of lead or lead alloys. However, a principal source of lead in municipal drinking waters is lead pipe and goosenecks in house services and plumbing systems. The practice of using lead pipe is still permitted by many plumbing codes. Normal adults in the temperate zone drink quantities of water, ranging from less than 1 to more than 3 liters/day, the average being taken as 2 liters. This is in addition to the water used in cooking and in other beverages. Thus, water can contribute a substantial proportion of the total daily intake of lead, depending upon the concentration of lead therein, the environmental temperature, and physical exertion.

Inasmuch as three of the four sources of lead intake in the human body—ingested foodstuffs, inhaled atmosphere, and tobacco smoke—are for the most part unregulated in their lead content, and because the total daily intake of lead which results in progressive retention of lead in the human body appears to be less than twice the average normal intake of lead in adults in the United States, concentrations of lead in drinking water greater than 0.05 mg/1 constitute grounds for rejection of the supply.

In consonance with this limit is the reported finding that bacterial decomposition of organic matter is inhibited by lead concentrations at or above 0.1 mg/1 (*5*). Lead in soft water is highly toxic to certain fish (*6*); 0.1 mg/1 is toxic to small sticklebacks, larger fish are somewhat less susceptible to lead. Calcium ion at a concentration of 50 mg/1 removes the toxic effect of 1 mg/1 lead for fish (*7*).

LIMITS AND RANGES OF LEAD AFFECTING HEALTH

Physiologically safe in water:	
Lifetime	**0.05 mg/1**
Short period, a few weeks	**2–4 mg/1**

Harmful range in water:

Borderline	2–4 mg/1 for 3 months.
Toxic	8–10 mg/1, several weeks.
Lethal	Unknown, but probably more than 15 mg/1, several weeks.

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MANGANESE

There are two reasons for limiting the concentration of manganese in drinking water: (a) to prevent esthetic and economic damage, and (b) to avoid any possible physiologic effects from excessive intake.

It has been reported that minute amounts of manganese cause difficulty in water quality control. The domestic consumer finds that it produces a brownish color in laundered goods and impairs the taste of beverages including coffee and tea (*1*, *2*).

From the health standpoint, there are no data to indicate at what level manganese would be harmful when ingested (*3*, *4*). The principal toxic effects which have been reported are the results of inhalation of manganese dust or fumes. It has been estimated that the daily intake of manganese from a normal diet is about 10 mg (*5*). In animals, at least, it has been shown to be an essential nutrient, since diets deficient in manganese interfere with growth, blood, and bone formation and reproduction. Hepatic cirrhosis has been produced in rats when treated orally with very large doses. As far as is known, the neurologic effects of manganese have not been reported from oral ingestion in man or animal (*6*).

The principal reason for limiting the concentration of manganese is to provide water quality control and thus reduce the esthetic and economic problems (*1, 3, 8*).

The U.S. Public Health Service Drinking Water Standards (1946) state that iron and manganese together should not exceed 0.3 mg/1. In a survey of 13 States reporting on levels of manganese giving rise to water quality problems, only three States recommended levels as high as 0.2 mg/1, two permitted 0.15 mg/1 and four each permitted 0.1 mg/1 and 0.05 mg/1 respectively. Domestic complaints arise when the level of manganese exceeds 0.15 mg/1 regardless of iron content. Griffin (*8*), in reviewing the significance of manganese as chairman of the task group on "Manganese Deposition in Pipelines", quoted the belief of certain water utility men that water to consumers should be free of manganese. For some industries, this is imperative. However, Griffin believes that concentration of manganese could be tolerated by the average consumer at 0.01–0.02 mg/1.

In view of the above and the difficulty of removing manganese to residual concentrations much less than 0.05 mg/1, and measuring such concentrations, manganese concentrations should be limited to a maximum of 0.05 mg/1.

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NITRATE

Serious and occasionally fatal poisonings in infants have occurred following ingestion of well waters shown to contain nitrate (NO_3). This has occurred with sufficient frequency and widespread geographic distribution to compel recognition of the hazard by assigning a limit to the concentration of nitrate in drinking water.

From 1947 to 1950, 139 cases of methemoglobinemia, including 14 deaths due to nitrate in farm well-water supplies, have been reported in Minnesota alone (*1*). Wastes from chemical fertilizer plants and field fertilization may be sources of pollution. The causative factor producing serious blood changes in infants was first reported in 1945 in polluted water containing 140 mg/l nitrate nitrogen (NO_3 –N) and 0.4 mg/l nitrite (NO_2) ion in one case; in the second case, 90 mg/l nitrate nitrogen and 1.3 mg/l nitrite ion (*2*). Since this report, many instances of similar occurrences have been recorded not only in this country but in Canada, Great Britain, Belgium, Germany, and other countries.

The International Drinking Water Standards of 1958 took cognizance of the problem in noting that ingestion of water containing nitrates in excess of 50 mg/l (as nitrate) may give rise to infantile methemoglobinemia but have included no limit. Taylor (*3*), in England, has suggested a limit of 20 mg/l nitrate nitrogen. Bosch, et al. (*1*), consider nitrate nitrogen concentrations in excess of 10–20 mg/l capable of producing cyanosis in infants. Various South American countries have recommended maximum permissible levels of from 0.5–228 mg/l nitrate (NO_3) (0.1–51 mg/l nitrate nitrogen) (*4*).

Cases of infantile nitrate poisoning have been reported to arise from concentrations ranging from 15–250 or more mg/l nitrate nitrogen (usually with traces of nitrite ion) in instances in which the water was analyzed up to 1952, according to Campbell (*5*). Campbell himself reported a case from ingesting water with 26.2 mg/l as nitrate nitrogen (116 mg/l nitrate ion).

According to methods of analysis commonly employed for nitrate in water, the presence of appreciable amounts of chloride would result in an erroneously low value for nitrate, and the presence of considerable amounts of organic matter would give an erroneously high value for nitrate. Insufficient attention has been given this important factor in evaluating permissible safe levels of nitrate in water.

Nitrate poisoning appears to be confined to infants during their first few months of life; adults drinking the same water are not affected but breast-fed infants of mothers drinking such water may be poisoned (*6*). Cows drinking water containing nitrate may produce milk sufficiently high in nitrate to result in infant poisoning (*5*). Both man and animals can be poisoned by nitrate if the concentration is sufficiently great.

Among the more acceptable hypotheses for the specificity of nitrate poisoning of infants is the following: the gastric, free acidity of infants is low (a *p*H of 4 or greater), permitting the growth of nitrate-reducing flora in a portion of the gastrointestinal tract from

which nitrite absorption can occur. It is also stated that foetal hemoglobin forms methemoglobin more readily than the adult form.

According to a recent study from Germany (*8*), the primary causes of toxicity are an elevated nitrate concentration and the presence of an unphysiologic amount of nitrite-forming bacteria, especially in the upper portion of the digestive tract. Members of the coliform group and the genus Clostridium are capable of reducing nitrate to nitrite. In infants whose diet is mainly carbohydrate, it is believed that the coliform organisms are the group responsible; organisms capable of reducing nitrite to nitrogen are not normally present in the infant. Careful measurement of a number of other constituents in 23 offending well waters, nitrite, ammonia, chloride, and organic substances, failed to reveal a casual relation of these substances to the injury.

There are no reports of methemoglobinemia in infants fed water from public water supplies in the United States, although levels of nitrate in some may be routinely in excess of 45 mg/l. This may indicate that well water for analysis has often been improperly sampled or that some other as yet unknown factor is involved. Practically nothing is known of the variation in nitrate concentration in the same well. Because samples associated with injury are taken after injury occurs, it is conceivable that this delay has resulted in failure to measure truly injurious concentrations.

Sodium nitrate has been fed to rats for a lifetime without adverse effects at levels below 1 percent (10,000 ppm) in the diet (*9*); two dogs tolerated for 105 and 125 days, respectively, 2 percent nitrate in the diet without effects on blood or other adverse effects.

Nitrite is equally dangerous in water supplies. Although concentrations that occur naturally are generally of no health significance, nevertheless, they may enter water supplies inadvertently as a result of intentional addition to private supplies as anticorrosion agents.

A limit of 200 ppm of nitrite (or nitrate) in "corned" products has been set by Federal regulation on the basis that 100g corned beef could convert maximally from 10–40g hemoglobin to methemoglobin (1.4–5.7 percent of total hemoglobin). Adult human blood normally contains on the average of 0.7 percent methemoglobin; the blood of "heavy" smokers may contain 7–10 percent carboxyhemoglobin, another blood pigment conversion product incapable of transporting oxygen. Carbon monoxide in urban atmosphere adds perceptibly to the total inactive pigment. The summated blood pigment conversion products represent about the maximum tolerated without headache.

Because of the great difference in molecular weight between sodium nitrite, 69, and hemoglobin, 64,000, small increments of nitrite produce large quantities of methemoglobin (1g nitrite converts 460-

1850g hemoglobin). The margin of safety is still further narrowed in infants whose blood volume is small, their total blood hemoglobin is decreasing after birth (from 17–20g to 10.5–12g), and their foetal hemoglobin is more readily converted to methemoglobin.

An instance of nitrite poisoning of children has been reported (10). The children ate frankfurters and bologna containing nitrite considerably in excess of the 200 ppm permitted.

Evidence in support of the recommended limit for nitrate is given in detail by Walton (7) in a survey of the reported cases of nitrate poisoning of infants in this country to 1951. The survey shows that no cases of poisoning were reported when the water contained less than 10 mg/1 nitrate nitrogen. Walton notes, however, that in many instances the samples for analysis were not obtained until several months after the occurrence of the poisoning.

In light of the above information and because of the uncertainty introduced by tardy analyses, the frequent lack of attention to possible interfering factors in the analysis, the health of the infant, and the uncertain influence of associated bacterial pollution, 10 mg nitrate nitrogen (or 45 mg nitrate) per liter of water is a limit which should not be exceeded.

At present there is no method of economically removing excessive amounts of nitrate from water. It is important, therefore, for health authorities in areas in which nitrate content of water is known to be in excess of the recommended limit to warn the population of the potential dangers of using the water for infant feeding and to inform them of alternative sources of water that may be used with safety.

LIMITS AND RANGES RELATED TO NITRATE WATER STANDARD

Average concentration adult human blood: 10 ug nitrate/100 ml (0.1 ppm).
Average daily urinary nitrate excretion: 500 mg (mainly from vegetables).
Strained baby foods: 0 (squash, tomatoes)—833 ppm nitrate (spinach).
Green Vegetables: 50 ppm nitrate (asparagus, dry weight), 3,600 ppm nitrate (spinach, dry weight).
Limit of nitrite (or nitrate) permitted in meat (or fish) products by Federal regulation: 200 ppm.

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PHENOLS

The term "phenols" is understood to include cresols and xylenols. Both the International Drinking Water Standards and those of the U.S. Public Health Service of 1946 recommended a limit of 1 ug/1 of phenol in water. This limit is set because of the undesirable taste often resulting from chlorination of waters containing extremely low concentrations of phenol. Phenol concentrations of 5 mg/1 or more are injurious to fish, whereas 1 mg/1 or less will not seriously affect most fish. Concentrations from 15–1,000 mg/1 in the drinking water were reported (*1*) without observable effect on rats for extended periods; 5,000 mg/1 appeared likewise to exert no effect on digestion, absorption, or metabolism, but 7,000 mg/1 arrested growth and resulted in many stillbirths. Thus, concentrations injurious to health are far removed from those which impart unpleasant taste or affect fish. Phenol is largely detoxified in the mammalian body by conjugation to far less toxic substances (*2*).

Although additional information has been developed (*3*) since the 1946 Standard was set, its nature indicates no need of a change in the former limit for phenols 0.001 mg/1 (1 ug/1).

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SELENIUM

The presence of selenium in water has heretofore been a matter of regional importance (*1*). The fact that it is now recognized as being toxic to both man and animals makes it essential that limits be set for all water intended for human consumption.

Selenium is known to produce "alkali disease" in cattle, and its effects, like those of arsenic, may be permanent (*1,2*). Recent reports indicate also that selenium may increase the incidence of dental caries in man (*3*). Of greater importance in limiting the concentration of selenium is its potential carcinogenicity (*4*). Rats fed a diet containing varying concentrations of selenium (3 to 40 mg/1) showed toxic effects at all levels, the outstanding pathologic lesion being hepatic cell tumors.

From very limited information (*5*) concentrations of selenium in water considered safe for man have been found toxic for fish.

In view of the potential seriousness of above reported effects, it is recommended that the limits for selenium be lowered from its present value of 0.05 mg/1 to 0.01 mg/1 and concentrations in excess of this lower value be used as grounds for rejection of the supply.

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SILVER

The need to set a water standard for silver (Ag) arises from its intentional addition to waters for disinfection. The chief effect of silver in the body is cosmetic, which consists of a permanent blue-grey discoloration of the skin, eyes, and mucous membranes which is as unsightly and disturbing to the observer as to the victim. The amount of colloidal silver required to produce this condition (argyria, argyrosis), and which would serve as a basis of determining the water standard, is not known, but the amount of silver from injected Ag-arsphenamine, which produces argyria is precisely known. This value is any amount greater than 1 gram of silver, 8g Ag-arsphenamine in an adult (*1, 2*).

From a review (*2*) of more than 200 cases of argyria, the following additional facts were derived. Most common salts of silver produce argyria when taken by mouth or by injection. There is a long-delayed appearance of discoloration. No case has been uncovered that has resulted from an idiosyncrasy to silver. There was, however, considerable variability in predisposition to argyria; the cause of this is unknown but individuals concurrently receiving bismuth medication developed argyria more readily. Although there is no evidence that gradual deposition of silver in the body produces any significant alteration in physiologic function, authorities are of the opinion that occasional mild systemic effects from silver may have been overshadowed by the striking external changes. In this connection, there is a report (*3*) of implanted silver amalgams resulting in localized argyria restricted to the elastic fibers and capillaries. The histopathologic reaction resembled a blue nevus simulating a neoplasm with filamentous structures and globular masses. Silver affinity for elastic fibers had been noted a half-century earlier (*5*).

A study (*5*) of the metabolism of silver from intragastric intake in the rat using radio-silver in carrier-free tracer amounts showed absorption to be less than 0.1–0.2 percent of the silver administered; but this evidence is inconclusive because of the rapid elimination of silver when given in carrier-free amounts. Further study indicated, however, that silver is primarily excreted by the liver. This would be particularly true if the silver is in colloidal form. Silver in the body is transported chiefly by the blood stream in which the plasma proteins and the red cells carry practically all of it in extremely labile combinations. The half-time of small amounts of silver in the blood stream of the rat was about 1 hour. A later report (*6*), using the spectrographic method on normal human blood, showed silver unmistakably in the red blood cell and questionably in the red cell ghosts and in the plasma. Once silver is fixed in the tissues, however, negligible excretion occurs in the urine (*7*).

A study (*8*) of the toxicologic effects of silver added to drinking water of rats at concentrations up to 1,000 ug/1 (nature of the silver salt unstated) showed pathologic changes in kidneys, liver, and spleen at 400, 700, and 1,000 ug/1.

A study (*9*) of the resorption of silver through human skin using radio-silver Ag^{111} has shown none passing the dermal barrier from either solution (2 percent $AgNO_3$) or ointment, within limits of experimental error (± 2 percent). This would indicate no significant addition of silver to the body from bathing waters treated with silver.

Great uncertainty, however, currently surrounds any evaluation of the amount of silver introduced into the body when silver-treated

water is used for culinary purposes. It is reasonable to assume that vegetables belonging to the family Brassicaceae, such as cabbage, turnip, cauliflower, and onion, would combine with residual silver in the cooking water. The silver content of several liters of water could thus be ingested.

Despite these uncertainties and the present lack of appropriate drinking water studies, it is possible to derive a tentative drinking water standard for silver by using silver deposited in excess of 1g in the integument of the body as an end point that must not be exceeded. Assuming that all silver ingested is deposited in the integument, it is readily calculated that 10 ug/1 could be ingested for a lifetime before lg silver it attained from 2 liters water intake per day; 50 ug/1 silver could be ingested approximately 27 years without exceeding silver deposition of 1g.

Because of the evidence (7) that silver, once absorbed, is held indefinitely in tissues, particularly the skin, without evident loss through usual channels of elimination or reduction by transmigration to other body sites; and because of the probable increased absorbability of silver as silver-bound sulfur components of food cooked in silver-treated waters, the intake for which absorption was reported in 1940 to amount to 60–80 ug per day (*10*); and because of the above calculation, a concentration in excess of 50 ug/1 is grounds for rejection of the supply.

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ZINC

Limits for concentrations of zinc in drinking waters have been established as follows: (a) USPHS Drinking Water Standards (1946), 15 mg/1; (b) Ohio and North Dakota, 1 mg/1; (c) International Drinking Water Standards (1958), permissible—5 mg/1 and excessive—15 mg/1; (d) various South American Countries, 5 to 15 mg/1.

Zinc is an essential and beneficial element in human metabolism (*1*). The daily requirement for preschool-age children is 0.3 mg Zn/kg. Total zinc in the adult averages 2g. Zinc content of human tissues ranges from 10–200 ppm wet weight, the retina of the eye and the prostrate containing the largest concentrations (500–1,000 ppm). Three percent of all blood zinc is in the white blood cells. The daily adult human intake averages 10–15 mg; excretion of zinc averages about 10 mg daily in the feces and 0.4 mg in the urine. Zinc deficiency in animals lead to growth retardation that is overcome by adequate dietary zinc. The activity of several body enzymes is dependent on zinc.

A group of individuals stationed at a depot used a drinking water supply containing zinc at 23.8 to 40.8 mg/1 and experienced no known harmful effects. Communities have used waters containing from 11–27 mg/1 without harmful effects (*2, 3*). Another report (*4*) stated spring water containing 50 mg/1 was used for a protracted period without noticeable harm. On the other hand, another supply containing approximately 30 mg/1 was claimed to cause nausea and fainting.

Zinc salts act as gastrointestinal irritants. Although the illness is acute, it is transitory. The emetic concentration range in water is 675–2, 280 mg/1. In tests performed by a taste panel, 5 percent of the observers were able to distinguish between water containing 4 mg/1 (when present as zinc sulfate) and water containing no zinc salts (*5*). Soluble zinc salts at 30 mg/1 impart milky appearance to water, and at 40 mg/1, a metallic taste (*6*).

Inasmuch as zinc in water does not cause serious effects on health but produces undesirable esthetic effects, it is recommended that concentrations of zinc be kept below 5 mg/1.

Cadmium and lead are common contaminants of zinc used in galvanizing. Assuming that zinc is dissolved from galvanized water pipe no less than cadmium, dissolution of zinc to produce 5 mg/1 would be accompanied by something less than the allowable 0.01 mg cadmium per liter when cadmium contamination of the zinc is as high

as 0.03 percent. Likewise, lead concentrations would likely be increased by something less than the allowable 0.05 mg/1 when lead contamination of the zinc is as high as 0.6 percent.

LIMITS AND RANGES RELATIVE TO ZINC WATER STANDARD

Food (7)—Milk, 4 mg/1
Egg (Hen)—1 mg
Cd content of galvanized pipe: 0.014–0.04 percent. Average 0.03 percent.
Pb content of galvanized pipe: 0.24–0.6 percent. Average 0.45 percent.
Urban air concentration: Average 2 ug/m^3 (8).
Concentrations toxic to fish: 0.3–4 mg/1, depnding on degree of water hardness (9).
Drinking water containing 50 mg/1 (as Sulfate) was not harmful to rats which used it for 6 weeks (3).

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E—RADIOACTIVITY

The effects of radiation on human beings are viewed as harmful and any unnecessary exposure to radiation should be avoided. In this discussion we are concerned with radiation from radioactive materials in the environment, particularly in water, food, and air.

The development of the nuclear industry has been attended by a small, unavoidable increase of radioactivity in the environment. Nuclear weapons testing causes an increase of radioactivity from fallout. Exposure of human beings to environmental sources of radiation

should be minimized insofar as is technically and economically feasible.

The Federal Radiation Council (*1*) has provided guidance for Federal agencies conducting activities designed to limit exposure of individuals of population groups to radiation from radioactive materials deposited in the body as a result of their occurrence in the environment.

The following recommendation of the Federal Radiation Council is considered especially pertinent in applying these Standards: (*2*)

> "There can be no single permissible or acceptable level of exposure without regard to the reason for permitting the exposure. It should be general practice to reduce exposure to radiation, and positive effort should be carried out to fulfill the sense of these recommendations. It is basic that exposure to radiation should result from a real determination of its necessity."

The Federal Radiation Council criteria (*1*) (*3*) (*4*) have been observed in establishing the limits for radioactivity in the Drinking Water Standards. It should be noted that these Federal Radiation Council guides apply to normal peacetime operations.

The Federal Radiation Council guides are predicated upon three ranges of daily intake of radioactivity. For each range, a measure of control action was defined, which represented a graded scale of control procedures. These are shown by the following table:

TABLE I.—*Graded scales of action*

Ranges of transient rates of daily intake	Graded scale of action
Range I	Periodic confirmatory surveillance as necessary.
Range II	Quantitative surveillance and routine control.
Range III	Evaluation and application of additional control measures as necessary.

The Federal Radiation Council (*4*) further defined the action to be taken by stating that: "Routine control of useful applications of radiation and atomic energy should be such that expected average exposures of suitable samples of an exposed population group will not exceed the upper value of Range II." Furthermore, they recommended, with respect to Range III, that "Control actions would be designed to reduce the levels to Range II or lower and to provide stability at lower levels."

The radionuclide intake ranges recommended are the sum of radioactivity from air, food and water. Daily intakes were prescribed with the provision that dose rates be averaged over a period of one

year. The range for specific radionuclides recommended by the Federal Radiation Council (*1*) are shown in the following table:

TABLE II.—*Ranges of transient rates of intake (micromicrocures per day) for use in graded scale of actions summarized in Table I*

Radionuclides	Range I	Range II	Range III
Radium-226	0-2	2-20	20-200
Iodine-131 [1]	0-10	10-100	100-1000
Strontium-90	0-20	20-200	200-2000
Strontium-89	0-200	200-2000	2000-20,000

[1] In the case of Iodine-131, the suitable sample would include only small children. For adults, the RPG for the thyroid would not be exceeded by rates of intake higher by a factor of 10 than those applicable to small children.

The Advisory Committee, in considering limits which should be established for drinking water, recommended limits for only two of the above nuclides, Radium–226 (3 uuc per liter) and Strontium–90 (10 uuc per liter). Iodine–131 is not found in significant quantities in public water supplies frequently enough to call for routine monitoring and Strontium–89 levels are not likely to be significant unless Strontium–90 levels also are high.

In the case of Radium–226, above-average levels of intake generally occur only in unusual situations where the drinking water contains naturally occurring Radium–226 in greater than average amounts, as in the case of certain ground waters, or from the pollution of the supply by industrial discharges of waste containing radium. With this in mind, a limit of 3 uuc/liter has been set for Radium–226 in drinking water. If one assumes a daily intake of such drinking water of about 2 liters per day, this would result in a daily intake from water of 6 uuc which falls in the lower portion of Range II in the above table. If there is evidence that Radium–226 from sources other than water is greater than usual, levels may have to be reduced below the above limit using the guides established by the Federal Radiation Council.

The principal source of Strontium–90 in the environment to date has been due to fallout from weapon tests, and human intake of Strontium–90 to date has been primarily from food. In recognition of this fact, the limit for Strontium–90 in water has been set at 10 uuc/liter, a limit substantially higher than the highest level found in public water supplies to date.

The Standards recognized the need to provide guidance for those situations where the limits are exceeded. In these instances, the Standards provide for the continued acceptance of the water supply if radioactivity from all other sources in addition to that from the water does not exceed intake levels recommended by the Federal Radiation Council for control action (the upper limit of Range II). It is essen-

tial in such instances for the certifying authority to determine with reasonable confidence that this latter condition is met.

Although a great variety of radionuclides may be present in drinking water, it has not been considered necessary to establish limits for general application to water supplies for other than the above two at this time. If significant concentrations of radioactivity are found in drinking water, an effort should be made to determine the radionuclides present and, where appropriate, to reduce their concentrations as much as feasible.

In assessing the hazard of radionuclides for which limits have not been set in these Standards, or for which guidance has not yet been provided by the Federal Radiation Council, it is suggested that the values (MPCw for the 168-hour week) in table I, of the report of the International Commission on Radiological Protection (*6*) or the National Committee on Radiation Protection (*7*), adjusted by a factor appropriate for exposure of the general population, be used. When mixtures of radionuclides are present the permissible concentration of any single nuclide must be reduced by an amount determined through applicable calculations in these reports.

In these Standards an upper limit of 1,000 $\mu\mu$c per liter of gross beta activity (in the absence [1] of alpha emitters and Strontium-90) has been set. If this limit is exceeded the specific radionuclides present must be identified by complete analysis in order to establish the fact that the concentrations of nuclides will not produce exposures above the recommended limits established in the Radiation Protection Guides. (*3*)

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