According to Harriman (Alaskan expedition), there were at Unalaska only 8 days in the year, during several years' record, which were entirely clear, the remaining 312 being cloudy and 271 of these were rainy or snowy.

The Yukon district, or Northern Alaska, comprises that vast region of the Yukon Valley which extends from the Alaskan Mountains to the Arctic Ocean on the north and Behring Sea and Strait in the west.

In the interior of this region the climate becomes colder and drier—extremely rigorous during the long winter and relatively hot in the short summer. As one continues north arctic conditions of climate begin. On the Behring Sea coast, north of the Aleutians Islands, the winter climate is much more severe than that of temperate Alaska on the Pacific coast, but in summer the difference is less marked. At St. Michaels, on the south side of Norton Sound, the mean summer temperature is 50° F., which is but 4° below that of Sitka; and at Point Barrow, on the Arctic Ocean, the most northerly point in the United States, the mean summer temperature is 36.8° F. Furthermore, the winter on the Behring Sea coast about the mouth of the Yukon River and the Seaward Peninsula is somewhat less protracted and severe than in the interior, although it is still long, and from October to May the temperature rarely rises above the freezing point.

Extreme cold, however, as one knows from the experience of Arctic explorers, is not detrimental to health, and at Nome, the most populous mining town in Alaska, the winter is said to be the most agreeable season of the year, in spite of the fact that in midwinter there are but few hours of daylight, the shortest days giving but about three and a half hours of dusky light. "With hands and feet warmly protected, and winter underwear and windproof outer clothes *and exercise*, one can comfortably weather a degree of cold which, in lower latitudes, would immediately transform him to an icicle. This is due to the dryness of the cold." ("The Land of Nome," by Laurie McKee, New York, 1902.)

The following table, compiled from observations of the United States Weather Bureau, gives the annual and monthly mean temperatures and the extremes for St. Michaels, which is on the southern side of Norton Sound; it also may be utilized for ascertaining approximately the yearly temperature of Cape Nome, which is one hundred and fifty miles distant on the northern shore of Norton Sound, at its junction with Behring Sea. In the same table will be found a statement of the mean number of rainy and snowy days. As will be seen, the rainfall is very light, and is about fourteen inches annually, a striking contrast to that of Southern Alaska.

St. Michaels.

	Jan.	Feb.	Mar.	Apr.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.	Year.
Mean maximum (degs. Fahr.)	33.5°	38.0°	32.0°	40.5°	48.5°	62.5°	77.0°	65.0°	56.0°	47.5°	37.0°	34.0°	....
Mean minimum	−34.0	−20.0	−17.0	−20.5	−7.0	27.0	40.0	37.0	25.0	6.5	−4.0	−24.0	....
Mean monthly	−8.0	−2.3	8.9	19.9	33.1	46.3	53.6	51.9	43.9	30.5	15.6	4.8	26.1°
Extreme maximum	44.0	41.0	43.0	46.0	57.0	75.0	75.0	69.0	69.0	54.0	42.0	45.0	75.0
Extreme minimum	−47.0	−41.0	−39.0	−27.0	−2.0	22.0	33.0	32.0	18.0	3.0	−24.0	−43.0	−47.0
Mean number of rainy and snowy days	7	4	6	8	9	9	12	14	14	11	9	5	108

From observations made on the Yukon, not far from the site of the gold discoveries, by the United States Coast and Geodetic Survey for a series of six months, the following temperatures are noted: From October, 1889, to April, 1890, the mean temperature was as follows: October, 33° (above zero); November, 8° (above zero); December, 11° (below zero); January, 17° (below zero) February, 15° (below zero); March, 6° (above zero); April, 20° (above zero). "The daily mean temperature fell and remained below the freezing point (32° F.) from November 4th, 1889, to April 21st, 1890, thus giving one hundred and sixty-eight days as the length of the closed season. The lowest temperatures registered during the winter were: 32° below zero in November; 47° below zero in December; 59° below zero in January; 55° below zero in February; 45° below zero in March; 26° below zero in April. "The greatest continued cold occurred in February, 1890, when the daily mean for five consecutive days was 47° below zero. The weather moderated slightly about the 1st of March, but the temperature still remained below the freezing point. Generally cloudy weather prevailed, there being but three consecutive days, in any month, with clear weather, during the whole winter. Snow fell upon one-third of the days in winter, and a less number in the early spring and late fall months. The change of temperature from winter to summer is rapid owing to the great increase in the length of the day." (Bulletin of the United States Weather Bureau, July 29th, 1897.)

Yukon River at International Boundary, Lat. 65°, Long. 141°.

	Jan.	Feb.	Mar.	Apr.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.	Year.
Mean temperature (degs. Fahr.)	−17.0°	−10.0°	7.0°	24.0°	45.0°	57.0°	60 0°	52.0°	39.0°	31.0°	3.0°	−16.0°	23.0°
Extreme maximum	25.0	37.0	38.0	56.0	74.0	84.0	87.0	74.0	66.0	52.0	39.0	17.0	87.0
Extreme minimum	−60.0	−55-0	−45.0	−26.0	8.0	30.0	35.0	31.0	14.0	4.0	−35.0	−49.0	−60.0

One cannot be sure of reaching Nome by sea much before the middle of June on account of the ice in Behring Sea, or of getting away from there after the latter part of September or 1st of October. The prevailing winds are from the north, and severe blizzards with strong northeast gales are frequent in winter. In comparing the climate of Nome with that of the Klondike region to be spoken of directly, it may be said that in general the climate of the latter is rather more favorable than that of the former. The most trying climatic element is the continual wind.

Fifteen hundred miles in the interior, to the east of Nome City, is the Klondike region, also famed and frequented for the gold discovered there. It is reached either overland—the common passenger route from Skagway by rail for about one hundred miles by the White Pass and Yukon Railroad, and thence by steamer on the upper Yukon to Dawson—or by the longer all-water route, which is principally used for freight, by way of the lower Yukon. The distance from Skagway to Dawson, the principal city of the Klondike (in Canadian Territory), is five hundred and eighty miles.

The general characteristics of the Klondike climate are similar to those of Nome—long, extremely cold winters, with much snow and "brief but relatively hot summers." "In midwinter the sun rises from 9:30 to 10 A.M., and sets from 2 to 3 P.M., the total length of daylight being about four hours." (United States Weather Bureau report.) In June the sun rises about 1:30 in the morning and sets at 10:30 P.M., "giving about twenty hours of daylight, and diffuse twilight the remainder of the time." "During the warmer days of summer the heat feels almost tropical; the winter cold is, on the other hand, of almost the extreme Siberian region." "Yet a beautiful vegetation smiles not only over the valleys, but on the hilltops, the birds gambol in the thickets, and the tiny mosquito pipes out its daily sustenance to the wrath of man." (Heilprin, "Alaska and the Klondike.")

The following observations of mean and extreme temperatures of the United States Weather Bureau made at the Yukon River at the international boundary, about eighty miles north of Dawson, from September, 1889, to June, 1891, will indicate approximately the temperature conditions of the Klondike.

Such a climate, although severe, is said to be a healthy and invigorating one to most people, for the cold is uniform and dry, and there is very little wind, a contrast, in this respect, to Nome. In a report by Capt. W. P. Richardson, Eighth Infantry, U. S. A., the fact is stated that when the thermometer rises to zero, as it sometimes does in midwinter, it is too warm for comfortable travel. The best temperature, he states, is from 10° to 25° or 30° below zero. "With this temperature the sleds run easily, dogs work with spirit, and one can exercise with the warm clothing necessary at all times in Alaska without discomfort." The ground is frozen deeply, and in the warmest season only thaws to the depth of a foot or two.

The vegetation in the Klondike region is, comparatively speaking, far more luxuriant than at Nome, where it is of arctic character, chiefly mosses and lichens, and the tundra or thick peat moss, or grass which renders foot travelling wearisome and slow. In the Klondike region the country is well wooded, principally with the spruce, although the aspen, birch, balsam, and poplar are found, and this region of forest extends with breaks several hundred miles northward of Dawson. In the summer the country is green and variegated, with a rich flora. Grass grows abundantly, and all the hardy vegetables are said to grow without trouble. Grain, vegetables, and fruit have been raised in small quantities. The native strawberry is found in many parts of the Yukon valley, and so also are various native berries, especially the blueberry. In the Yukon valley, near Dawson, celery, lettuce, potatoes, turnips, etc., have been successfully grown, as well as oats and wheat, and this in a latitude which runs through Greenland and Iceland! Of course such results would be impossible were it not for the fact that the summer days, though few, are very hot and the sun is almost continually above the horizon.

Fish, furs, and gold are the principal industries of Alaska. The discovery of gold has naturally attracted the most attention, but the fisheries form one of the most important industries, and next in importance to the fur trade is the salmon industry. The population was 63,592 at the census of 1900, of which number over 45,000 were males and about half were whites.

Nome City is the largest town, with a population of over 12,000, and next comes Skagway, with a little over 3,000. Dawson, the principal town of the Klondike region, in Canadian Territory, had in 1899 16,000 inhabitants.

The testimony is somewhat conflicting regarding the mosquitoes, but they are apparently pretty abundant, and at certain times and places constitute a veritable scourge. The gnats are also very annoying.

The accommodations, especially in the mining towns, are naturally not of the best, and are expensive; still, any one possessed of robust health need not be deterred either by the climate or by the poor accommodations from a journey to, or a permanent abode in, Alaska. The steamer accommodations from San Francisco, Seattle, or Vancouver are by some lines quite satisfactory. A summer excursion to the southeastern coast of Alaska—the iceberg region—is a favorite one.

References.—Various government reports from the Interior Department; Department of Commerce and Labor; Department of Agriculture, and the Weather Bureau; yearly reports of the governor of Alaska; Harriman, "Alaska Expedition"; Heilprin's "Alaska and the Klondike"; "The Land of Nome," by Laurie McKee; "The Pacific Coast Pilot," and many other special works.

Edward O. Otis.

ARSENIC, POISONING BY.—It has been estimated that of all the recorded deaths due to poisoning, at least three-fourths have probably been due to arsenic in some form. So powerful a factor has this poison proved in the history of the world's progress, and so instructive are the records, that a glance at the past seems necessary.

The knowledge of the poisonous nature of the compounds of arsenic seems to have first come from out the mysterious East. Long before the dawn of written history the Asiatics, the Egyptians, and doubtless other Eastern peoples, seem to have been well acquainted with the sulphides of arsenic and probably also with the method of preparing the trioxide by roasting arsenical ores. The oldest of the manuscripts and papyri dealing with materia medica, etc., which have come down to us (the famous Leyden collection), include the name "arsenic" in the list of substances with medicinal or poisonous action. We are ignorant, however, of the part this substance played in the world's drama prior to the first century A.D. But about this period the writings of Dioscorides and of Olympiodorus caused the method for the preparation of the oxide to be well known and its poisonous property to be well understood. From this time on, arsenic trioxide, because of its being colorless, odorless, tasteless, and insidious in its action, became a factor in history. Professional poisoners soon appeared on the stage and succeeded in developing poisoning into an art—if this be not a prostitution of this term. The first of these artists in crime seems to have been Locusta. Attached to the court of Agrippina she caused the death of Claudius by means of arsenic; later, as the tool of Nero, she accomplished the death of Britannicus and threw Rome into consternation and terror. Put to death by Galba in 68 A.D., she was succeeded by an adept disciple, Canidia, who administered arsenic to a large number of victims. From this time on, Italy proved a hotbed for schools of crime in which arsenic was always the chief agent. Until the thirteenth and to a certain extent to the fifteenth century, murder by means of arsenical preparations knew no bounds and overflowed all Europe. Standing forth among the many historical lesser criminals, we have Charles the Bad, King of Navarre, whose remarkable letters patent (fourteenth century), giving directions for the poisoning of his brothers and uncles by means of white arsenic to be mixed with food or wine, are still extant; the Pope Alexander VI., murdering out of revenge those of his cardinals who disagreed with him, and the wealthy cardinals, his friends, in order that he might seize their property, meeting death himself through the

mistake of a servant who gave him a bottle of wine containing arsenic intended for Cardinal Corneto. Alexander left two of his five children, Lucretia and Cæsar Borgia, of hideous memory, demons in human form, to distribute arsenic in all directions. About this time arose the famous Council of Ten of Venice, spreading murder abroad largely through the agency of an adept in the use of arsenic, de Ragubo, the Franciscan.

A century later makes us acquainted with the celebrated "Acqua Toffana," which probably consisted of a solution of arsenic trioxide in aqua cymbalaria, to which a small amount of tincture of cantharides was added. The woman Toffana, living in Palermo and Naples, discoverer and dispenser of this liquid, is credited with the death of over five hundred victims, including several crowned heads and two popes. While she practised her nefarious trade for years unsuspected, the Acqua Toffana struck terror throughout all Italy, and even throughout France and Austria. After her detection she fled for safety to a convent, where for twenty years longer her horrible trade flourished in secret. Upon her final detection and execution La Toffana's clients were supplied with the arsenical fluid by Spara, who had obtained the secret of its preparation from Toffana. Spara sold her preparations under a variety of other names, as, for example, "Manna di S. Nicolas di Bari," "Acquetta di Napoli," etc. Since Spara's clients seem to have been mostly of the middle class, records are lacking as to the extent of her criminal practices. Toffana and Spara also prepared and dispensed another poison even more potent, in which arsenic was combined with putrefactive compounds (the arsenical ptomaïns of Selmi). This preparation, sold under the name of "Acquetta di Peruzia," was prepared by sprinkling the viscera of a freshly killed pig with white arsenic, hanging them up and then collecting in a vessel the drippings of the putrefying material.

France now became the theatre of the great arsenical crimes of history through Catherine de Medici, who brought with her to France arch poisoners from her native land. The court of Louis XIV. was fairly smothered in crime. Among the hundreds of petty poisoners two remarkable women, doubtless the greatest criminals France has ever seen, stand head and shoulders above the rest. The first of these, Marguerite d'Aubray, Marquise de Brinvilliers, angelically beautiful, with winning voice and saint-like appearance, yet addicted to unspeakable vices from a child up, had been taught the methods of mixing arsenic with foods and beverages by her paramour Saint Croix. This woman had set her heart upon inheriting the family estate, and to gain her end it became necessary to cause the death of her father, brothers, and sister. In order to accomplish this she obtained entrance to the great hospitals of Paris under the guise of a Sister of Mercy. Here as a nurse she administered arsenic with food and medicine, noted the effects, going each day to the internes to learn the progress of the disease. Finding that her practices were unsuspected and undetected, she carried her experiments still further by giving arsenic in food to the poor who called at her door, dosing her servants and visiting the hospitals to which they were taken in order to learn the effect. Having at last satisfied herself that she knew the dose and the rate at which the poison should be administered, she proceeded with the execution of her long-planned crime. During a period of over eight months she administered arsenic to her father, thirty times by her own hands and a number of times by a servant. At the end of this period the murder was accomplished by giving a double dose. In a like manner she disposed of two brothers and attempted the life of her sister. If her servants displeased her, a little arsenic, carefully dispensed, put them out of her way. It has been impossible to ascertain the number of her victims, so cunningly did she work. Not only did she use arsenic trioxide alone, but also a carefully compounded mixture of this substance with mercuric chloride. Her crimes being discovered upon the death of Saint Croix, she was tried, found guilty, and executed in July, 1676. Thus it was that the Marquise de Brinvilliers invented the infamous "poudre de succession," which in the hands of the second of the women, La Voisin and her accomplices, literally powdered Paris and part of France with arsenic.

The revolting story of La Voisin is foreign to the topic under discussion. Suffice it to say that the poison she dispensed was generally white arsenic; but what is noteworthy, is that she followed the de Brinvilliers' method, which had been experimentally developed, namely, slow poisoning. Poisoning became such an epidemic in France, following the publicity of these cases, that the notorious special court (La Chambre ardente—Court of Poisoners) was established. This body proceeded so perseveringly, thoroughly, and mercilessly that since 1682 there have been no systematic poisonings by professional poisoners, clubs, or bands. It has been recently stated that in modern times no Locusta, Toffana, nor de Brinvilliers can exist, that the illness of the victims would promptly be diagnosed as arsenical poisoning and the criminal discovered. Unfortunately, this belief is not justified, for there are many cases which demonstrate that slow poisoning by arsenic is not readily diagnosed. Take, for example, the case of the monomaniac Hélène Jegado, who from 1833 to 1852 poisoned some 47 persons, about 30 of whom died, all of arsenic poisoning; or again there is the woman Van der Linden, who in 1887, acting as a nurse and at the same time agent for an accident insurance company, poisoned, or attempted to poison, 102 persons. Twenty-seven died; 47 were made seriously ill. As a nurse she dosed her victims, in whose names she herself had made out policies. In due time she collected from the company the amount of the policy for illness or death. So carefully did she proceed that the illness of her victims was not diagnosed as arsenicism. Again, take the cases of great mass-poisoning by wine containing arsenic at Hyères in 1887, due to the circumstance that a wine merchant, with the intention of plastering his wine, had dropped into the fermentation vat by mistake a quantity of white arsenic. There were over 400 victims and 4 deaths. All sorts of diagnoses were made by the physicians, and it was not until many months afterward that the true cause was discovered. Similar errors were made in 1900–02 in the famous beer poisonings in Great Britain. In this epidemic there were over 4,000 victims and 300 deaths. The first cases of illness were reported in the latter part of April and early in May, but it was not until the following November, seven months later, that the proper diagnosis of arsenical poisoning was surely established, and it was shown that the trouble came from impure sulphuric acid used for the inversion of sugar and starch, which materials under the Gladstone bill could be used for the manufacture of beer.

Within a few months two cases of slow poisoning by arsenic have come to the author's attention, in each of which the attending physician failed properly to diagnose the illness. One of these cases was homicidal.

The lessons to be drawn from the historical cases and others cited above seem quite plain. The physical diagnosis of subacute or chronic arsenical poisoning is often uncommonly difficult.

Most Important Compounds with Reference to Poisonings.—*Arsenic trioxide*, As_2O_3 (synonyms: arsenious acid, arsenious anhydride, white arsenic, arsenic, ratsbane, rough-on-rats, etc.) exists in two allotropic modifications, the amorphous and the crystalline. The crystalline form is of a dead white, opaque, and porcelain-like appearance; the amorphous, on the other hand, is clear, transparent, and glass-like, and for this reason is often termed vitreous, and has a tendency to pass slowly into the crystalline state. Because of this change, commercial samples of lump white arsenic are mixtures of the two modifications. Save in India, where it is sometimes colored yellow with cow's urine, arsenic trioxide occurs in commerce as a heavy white powder, or in the form of opaque white lumps, which when broken show a transparent glassy centre. The solubility of the commercial samples varies within quite wide limits; this is due to the fact that the solubility of the two modifications of As_2O_3 are different, that of the pure crystalline being about 1 in 350 of water at 15° C., and that of the pure amorphous 1 in 108. Cooled from boiling, 1 part in 45 of the crystalline remains in solution and 1 part in 30 of the amorphous. No two observers agree, however, as to the solubility of As_2O_3.

Arsenites.—All the samples of commercial dry arsenite of sodium and of potassium which have been examined by the author seem to correspond most closely to the formulas $NaAsO_2$ and $KAsO_2$. Their solubility in water is quite variable, but is always much greater than that of the trioxide. The majority of cases of poisoning from alkaline arsenites have been from the medicinal preparations of these salts, such as Fowler's solution, etc. Only a very few of these cases have been criminal.

Arsenates.—These salts correspond to the general formula R_3AsO_4. They are of little toxicological interest save for the fact that arsenic acid has been largely used in certain industries (*e.g.*, manufacture of aniline dyes) as an oxidizing agent, and the products placed on the market in an impure condition contain arsenic in enormous quantities.

Sulphides of Arsenic.—The disulphide, As_2S_2, occurs in nature as realgar, and was known to the ancients under the name sandarac or sandaracque. Realgar, both the mineral and the artificial compound, has been used as a pigment, in pyrotechnics, etc. The trisulphide, As_2S_3, long known as the mineral orpiment or auripigment, has had a variety of commercial uses. It was formerly used in printing, in tanning as a depilatory, in medicine, as a pigment, etc. The pentasulphide, As_2S_5, is of no medicinal importance. All the sulphides met with in commerce are amorphous yellow powders, varying in color from a light lemon yellow to a deep golden or even orange tint. When pure they are insoluble in water, hence their toxicity is very low. Some authorities hold that the pure sulphides are not poisonous. Experimental evidence shows, however, that these compounds are acted upon by the fluids of the body, and that arsenic is resorbed. Doubtless a long sojourn of these sulphides in the alimentary canal would lead to serious results. The commercial sulphides are invariably violent poisons, since they contain arsenic trioxide, sometimes to the extent of as much as thirty per cent. Le Prince and Chabenat report the case of a young girl who used an ointment consisting of 65 parts of butter and 35 parts of orpiment as a dressing for a tumor of the breast. Acute arsenic poisoning resulted, with death on the fifth day after the application of the dressing. As a depilatory, orpiment has been used from earliest times. It was a mixture of orpiment with lime or chalk, which was used by the ancients for the removal of the pubic hairs—a practice said to be still extant, at least in the case of women, in certain Mussulman tribes of the East.

Cases of poisoning from organic compounds of arsenic have not yet been reported, if we except those due to the notorious Acquetta di Peruzia. But considerable experimental work has been done on animals with kacodyl, kacodylic acid, diphenylarsine, monophenylarsine, arsendimethylcyanide, etc.

Coloring matters, pigments, containing arsenic, are responsible for a large percentage of cases of poisoning, both acute and chronic. Scheele's green (synonym: mineral green, Swiss green) is copper arsenite of somewhat variable composition. It is insoluble in water, and corresponds to the formulas $CuHAsO_3$ or $Cu_3(AsO_3)_2 2H_2O$. This beautiful compound is used as a pigment in painting, printing, etc., and as an insecticide. Paris green (synonyms: Vienna green, mitis green, Schweinfurter green) is a double salt, copper aceto-arsenite, corresponding to the formula $Cu(As_2O_4)_2,Cu(C_2H_3O_2)_2$. The commercial product is of variable composition according to the method employed in its manufacture. It is now used in enormous quantities as an insecticide; rarely as a pigment in painting, coloring, etc. Aniline dyes of the rosaniline group, which have been made by the old arsenical process, usually contain arsenic in large amount. The most important of these are the magentas and reds, but the blues, violets, purples, etc., may also be arsenical. In fact, it may be said that there is scarcely a shade which cannot be obtained by blending arsenic-containing dyes. Practically all the manufacturers of aniline dyes have now abandoned the arsenical process, but the author is informed that there is still one firm in New York State that uses arsenic acid in the preparation of fuchsin. Closely related to the aniline dyes are the arsenical lakes, such as Cochineal red, Vienna red, etc. King's yellow consists of impure arsenic trisulphide, hence is essentially orpiment. It often contains eighty to ninety per cent. As_2O_3. Mineral blue, a pigment in which copper arsenite and potassium arsenite enter in about equal parts, contains about thirty-nine per cent. arsenic.

Arsenical insecticides are responsible for a large number of deaths, accidental, suicidal, and criminal. Of these substances the most important is Paris green. Its composition and quality are now controlled by statutory enactment in several States of the Union. When sold as an insecticide the statutes call for a product containing combined arsenic in an amount which shall be not less than fifty per cent. calculated as As_2O_3, not over four per cent. of which shall be soluble in cold water. Most of the cases of poisoning by this material occur in the United States and in Russia. London purple (synonyms: English purple, Paris purple), formerly much used, is obtained by treating a by-product of the aniline dye industry with lime. The arsenic is present both as calcium arsenite and as calcium arsenate, the latter in excess of the former. Calculated as arsenic the average amount present in London purple is about twenty-nine per cent. Arsenoids, substitutes for Paris green, have been placed on the market in recent years. Green arsenoid contains on an average about sixty per cent. As_2O_3, and has been incorrectly called Scheele's green. White arsenoid contains barium arsenite; thirty-three per cent. of the arsenic present is water-soluble. Para green is a mixture of Paris green with about thirty-six per cent. gypsum. Laurel green consists essentially of calcium arsenite (thirteen per cent.), gypsum, calcium carbonate, and magnesum carbonate.

Vermin Killers.—Of the many arsenical poisons on the market for the purpose of destroying rats, mice, etc., only one needs discussion, *i.e.*, rough-on-rats. This preparation has been employed in many crimes in the United States and consists of arsenic trioxide about seventy-two per cent., barium carbonate twenty-six per cent., sand two per cent., with usually a little carbon to color it. In case of a question of poisoning by this vermin killer, barium should always be searched for. The author has, however, in his possession a sample of this material purchased some years ago which contains no barium.

Arsenic in Foods, Beverages, etc., occurs (1) as a preservative, (2) in coloring matter added, or (3) as an accidental impurity. In official reports issued by the German Government the claim is made that there are annually exported from the United States to European ports enormous quantities of dried apples which have been treated with weak arsenical solutions to preserve them from decay and from the attacks of insects. Moscow, Russia, suffered severely some years ago from a cholera-like epidemic, the cause of which was eventually traced to sturgeon flesh which had been sprinkled with white arsenic to arrest decomposition. The addition of arsenical colors to foods and beverages has given rise to a large number of cases of arsenical poisoning. Sausage, jellies, preserved fruit, wines, fruit syrups, confectionery, cakes, etc., have all been found to have caused trouble. Usually this has been due to arsenical aniline dyes, but in a few instances Paris green has been present. Accidents arising from the presence of arsenic as an impurity in some product used in the manufacture of the food or beverage are usually confined to beers, ales, or glucose preparations. While we are considering poisonous food materials, the fact must not be overlooked that cattle feeding in the neighborhood of industries throwing out arsenic may become poisoned or else relatively immune from

habitual use, and their flesh when eaten by man may lead to very serious results. The same holds true for milk secreted by an animal which has taken arsenic into its system.

Arsenic in Colored Papers, Inks, Clothing, etc.—Many cases of very serious chronic arsenical poisoning have been traced to these sources. A decade ago the amount of highly arsenical wall papers was appalling, and in spite of the denial of eminent chemists and hygienists, the evidence is such that chronic poisoning from arsenical wall papers must be accepted as an established fact, providing that the room so papered is small, damp, and ill ventilated, and the alleged victim spends much time in the room. Contrary to popular opinion, green-tinted papers are rarely arsenical; those which have caused trouble in the United States have almost invariably been tinted in shades of red. In an investigation conducted by the author some years ago upon an undoubted case of arsenicism from wall paper, it was found that over ninety per cent. of the red and maroon papers obtainable of the same date of manufacture as that on the walls of the room were highly arsenical. Many samples of wall papers manufactured within the last six years have since been examined, but none of these modern papers have thus far been found which contain more than mere traces of arsenic. In all probability the cause of arsenicism from wall papers is largely due to the formation, by moulds and bacteria, of arsine and of a volatile organic arsenic compound. This peculiar action is so well marked in the case of Penicillium brevicaude that advantage is taken of the fact for the purpose of testing for the presence of arsenic. Mucor mucedo, Aspergillus glaucus, Aspergillus virescens also possess this same property, but to a less degree. Colored papers other than wall papers have also given rise to poisoning: as, for example, the chewing of such papers by children, the moistening with the tongue of many colored gummed labels. In 1889 several cashiers in German banks were poisoned through handling with moistened fingers large numbers of Swiss banknotes printed with arsenical ink. Several cases are on record of poisoning from using copying inks, typewriter ribbons and indelible pencils made with arsenical aniline dyes. Graffsky has described several very serious cases of chronic poisoning from handling colored chalk in one of the schools of Germany. A violet-colored crayon was found to contain over seven per cent. arsenic. Clothing dyed with impure aniline colors has been the cause of many a sad case of illness. These cases have usually been caused by such wearing apparel as comes in direct contact with the skin, such as stockings, trousers, waists, etc. In Germany copper arsenite has been used for coloring tarlatan and several cases of serious poisoning have resulted therefrom. Two of the most striking cases of arsenical paralysis with which we are acquainted were due to green tarlatan ball gowns. Zinreck states that he found in twenty yards of this material used for a ball gown 300 gm. of Paris green.

Medicinal preparations have given rise to poisoning, either from overdosage or because of the presence of arsenic as an impurity in the substance administered, as, for example, in bismuth subnitrate and other bismuth preparations, sodium phosphate, glycerin, chloroform, phosphoric acid, sulphuric acid, etc. For the composition and dosage of arsenical medicines, see *Arsenic*, Vol. I., of REFERENCE HANDBOOK.

Industrial poisoning by arsenic is by no means rare, for in a large number of industries the workmen are exposed to compounds of this element in one form or another. In the following table will be found a list of trades or articles manufactured which are credited with cases of arsenicism, and a brief statement of the cause of the illness.

Name of Industry or Article Manufactured.	Causes of Poisoning.
Paints in which the basis is orpiment, copper arsenites, mineral blue, etc.	Arsine, arsenical dust.
White enamel glass, stained glass and certain other glasses.	Arsine, arsenical dust.
Copper, cobalt, nickel, zinc, tin.	Arsenic dust in air from roasting arsenical ores.
Copper refined in the wet way.	Arsine.
Hydrogen for balloons, limelights, when made from iron or zinc and an acid.	Arsine.
Phosphoric acid.	Arsenic trioxide.
Sulphur and allied industries.	Arsenical dust, arsine.
Arsenic and arsenical preparations.	Arsenical dust in mines, charging and emptying retorts, handling products.
Galvanized iron, tinned iron.	Arsine.
Zinc chloride.	Arsine.
Copperas (ferrous sulphate).	Arsine.
Aniline dyes by the arsenical method.	Arsenic acid, arsenous acid in dust, in air or on hands.
Artificial flowers, leaves, etc.	Arsenical pigments or dyes by contact, or in dust in air.
Stuffed animals, birds, preparation of natural history specimens.	Arsenic trioxide, arsenical soap and fluids absorbed by hands or in dust.
Bird shot (arsenic used to harden the lead).	Arsenical dust.
Aniline from nitrobenzene.	Arsine.
Indigo blue.	Arsine.
Embalming fluids.	Arsenic trioxide, arsenites of sodium, potassium calcium.
Preservation of wood.	Arsenical dust.
Wall papers, colored papers.	Arsenical dyes and pigments in dust or by handling.

Channels of Introduction.—Accidental poisonings are the result of swallowing the poisonous material, breathing arsine or arsenical dust, or occasionally absorption through wounds or through the sound skin. But in criminal cases every conceivable channel has been used, the usual mode of introduction being in food or drink. A number of cases are recorded in which the poison has been administered to the victims by enemas or vaginal douches. The legend that Ladislas, King of Naples, met his death in 1414 by arsenic introduced into the vagina of his mistress by a jealous courtier, and that Calpurneus murdered his wives by digital introduction of arsenic into the vagina are probably not mere myth and fiction, for we find several modern imitators of Calpurneus. In one instance the arsenical material was introduced by placing the poison under the prepuce, and introducing it into the vagina in this manner, so as to avoid all danger of detection. In another instance three wives were successively murdered by placing in the vagina a mixture of flour and arsenic trioxide. Experiments on animals have shown, beyond the possibility of a doubt, that death may result from arsenical poisoning through such an introduction of the poison into the body. A few cases are found in which arsenical vapors have been employed with criminal intent, as for example the vaporization of arsenic trioxide and the roasting of arsenical pyrites in a room.

Fatal Dose.—Brouardel's investigations have shown that the toxicity of arsenic varies according to: (1) The method of administration; (2) the kind and age of animal; (3) the nature of the food material present in the alimentary canal. Owing to the fact that some persons have, to a certain degree, a natural or acquired resistance to arsenic it is difficult arbitrarily to set any definite figure as representing a fatal dose. Moreover, when the stomach contains a large amount of fatty material a huge dose can apparently be ingested without fatal results. It is to be said in this connection that a very large dose is seldom as dangerous as one consisting of close to what may be said to be the normal fatal dose, for the reason that vomiting is apt to be immediate and the poison is removed at once without having undergone resorption. For As_2O_3 the toxic dose for man can be set as lying between 5 mgm. and 50 mgm. (0.07–0.77 grain), and the fatal dose 100–300 mgm. (1.54–4.63 grains). For Paris green the doses are probably somewhat higher than twice these quantities. The above figures refer to per-os administration. When the poison comes in direct contact with the blood, as for example through wounds, the abraded skin, or the direct introduction into the body by some sharp instrument, it is about ten times as active. Rouyer has attempted to calculate the toxic and fatal doses on the basis of the amount of poison resorbed rather

than the total amount taken. According to his experiments and calculations 0.6 mgm. As_2O_3 resorbed per kilogram body weight will give rise to serious symptoms; 2.5 mgm. per kilogram may cause death in twenty-four to twenty-five hours, while 3 mgm. per kilogram will always cause death in about eight hours. It would seem that Rouyer's figures are probably too high, for they would require the resorption of about 200 mgm. to cause the death of a man of average weight. The actual dose to be ingested would therefore be considerably higher.

In the case of animals we find great variation in the intensity of the action of arsenical compounds, not only with respect to species, but also in different individuals of the same species. Von Gohier has seen a horse remain to all appearances in good health after a dose of 30 gm. of arsenic trioxide, while on the other hand 3 gm. has caused death. Not only do we have this variability of action on different individuals, but veterinarians are fairly well agreed that, particularly in the case of cattle and sheep, an animal may at one time stand a dose as high as 20–30 gm., while the same identical animal will suffer severe illness from 5 gm. at another time. The following table, while necessarily more or less arbitrary, can be taken as probably as nearly in accord with experimental data as is possible with our present knowledge.

	Fatal Dose.	
	When taken internally.	When by wounds.
Horned cattle	10.0 to 30.0 gm.	2.0 gm.
Horses	10.0 to 25.0 gm.	2.0 gm.
Sheep, goats	5.0 to 15.0 gm.	.2 gm.
Swine	.5 to 1.0 gm.	.2 gm.
Dogs	.1 to .2 gm.	.02 gm.
Fowl	.1 to .15 gm.	.01 gm.
Pigeons	.05 to .1 gm.	.005 gm.

It will be seen from the above that sheep and goats are, per kilogram weight, remarkably resistant. A similar resistance is frequently met with in certain dogs.

Habitual Use, Tolerance, Arsenicophagia.—Aside from certain species of fungi and bacteria, arsenical compounds are toxic to all other forms of life. But by repeated small doses a certain immunity is at last acquired, after which enormous doses can be swallowed with impunity. This fact was known in most ancient times, for we are told that Mithridates (first century B.C.), who was well versed in the poison lore of his time, acquired immunity in this manner to protect himself against poisoners. By far the most striking cases of acquired tolerance are to be found among the mountaineers of Tyrol, Styria, Carinthia, the Punjab, and in some parts of the mountain ranges of South America. These mountaineers start taking arsenic, in the form of orpiment or the trioxide, from their youth; the men believing that it enables them to endure greater fatigue, wards off the mountain sickness and makes breathing easier; the women in order to improve their complexion, to acquire shining eyes, a rosy tint, and a well-rounded form. These arsenicophages seem to enjoy perfect health and reach an advanced old age. They exist, however, in a pseudo-normal state, for the suppression of the daily dose of arsenic leads to serious illness. The majority never reach a very high daily consumption of the drug, but instances are not lacking in which the amount taken daily reaches 300–400 mgm. We also have proof positive that certain individuals have taken this same amount at a single dose without suffering any inconvenience thereby. This tolerance and habit seem in many cases to be hereditary. A somewhat similar resistance is met with in patients who have been subjected to arsenical treatment with increasing dosage. After a time considerably over the maximum dose is tolerated. In Europe and in America certain waters are found containing arsenic. These waters have been long used by the inhabitants of the district, but no evil effects have been reported.

The administration of arsenic to horses by grooms in increasing doses has been practised for many years, especially in England, the object being to improve the animal's coat, aid the assimilation of food, and to give them a plump, well-rounded appearance. The arsenic is either mixed with the food or is tied in a tiny piece of cloth and attached to the bit. This latter procedure results in the production of a slight salivation and the appearance of white foam about the mouth, the animal holding its head erect and champing at the bit, conditions which are supposed to indicate a spirited animal. Horses subjected for any length of time to this treatment require a continuance of the daily dose, without which they rapidly lose flesh and condition.

There is as yet no satisfactory explanation for the acquired tolerance of arsenic. The theory that there is an abnormal rate of elimination by the kidneys is not well established, and is insufficient to account for all the facts.

SYMPTOMS. ACTION.—With the possible exception of the gaseous compounds all other arsenical substances may be said to have a like action on man. No matter, therefore, in what form arsenic is administered the symptoms observed will not differ to any appreciable extent. These symptoms can be grouped under four different heads. Those typical: (1) Of a mineral acid—acidismus. (2) Of a mineral salt—metal toxicosis. (3) Of action on the nervous system: (*a*) splanchnic paralysis, resulting in hyperæmia of all abdominal organs; (*b*) neuritis. (3) Of inflammation of the intestinal tract and of fatty degeneration, especially of the heart. The result of the ingestion of the poison may lead to either acute or chronic poisoning. In acute poisoning it is possible to distinguish three forms: A gastro-enteric or ordinary form, paralysis arsenicalis, and asphyxia arsenicalis.

Acute Poisoning.—Ordinary Form. Soon after swallowing the poison the victim complains of a disagreeable metallic taste in the mouth, of an annoying burning, itching, or tickling dryness of the throat, there being usually frequent spitting and more or less salivation. This is generally, but not always, followed by pain in the upper part of the alimentary canal which gradually passes downward, increasing in severity. In most cases in from one to two hours, rarely later, after swallowing the poison, nausea, vomiting, and purging set in. In the event of Paris green being taken, vomiting sets in at an earlier stage than is the case with other arsenical compounds. The vomited matter may be streaked with blood, or green if copper compounds are present. In a short time the stools which were at first colored with bile, become frequent and rice-water-like. Usually there is tenesmus. The patient finds more or less difficulty in swallowing, suffers from dizziness, headache, pains in the limbs, a burning unquenchable thirst, and often a distressing hiccough. The abdomen is distended and is painful on pressure, the secretion of urine is diminished, and there may even be anuria. The patient's voice becomes hoarse, features pale and haggard, eyes sunken, skin cold and dry. The pulse becomes slow and weak; respiration difficult and oppressed, and death usually takes place in fifteen to twenty-four hours, but often on the second day. The patient is ordinarily conscious to the last, but there may be coma, paralysis, or convulsions. If death does not occur before the second or third day, there is usually an apparent general improvement, save for the persistence of the dryness of the mouth and severe thirst. The temperature remains about normal or very little above. There is apt to be extensive erythema or an erysipelas or œdematic swelling which is most marked on the face and genitals, but there are patches more or less scattered over the body. There may be falling out of the hair. Blebs or pustules may appear on various parts of the body, or there may be circumscribed efflorescences. In many instances recovery is difficult and very protracted. Arsenical applications to the sensitive skin ordinarily give rise to severe dermatoses; as, for example, arsenical eczema of the legs and thighs from contact with trousers or stockings colored with arsenical aniline dyes. There may even be gangrene.

Arsenical paralysis is seen in both acute and chronic

poisoning, but an extensive paralysis is rare. Seisser in the famous Würtemberg mass-poisoning observed only 1 case in 373. Alexander has collected the records of 55 cases of arsenical paralysis; of these 17 resulted from chronic arsenicism, while 38 were acute. In the great mass-poisoning in Great Britain in 1900, a very large proportion seem to have been troubled with paralytic symptoms. When arsenical paralysis results from acute poisoning it does not usually appear prior to the fourth day, or even the end of the first week. The normal termination of the seizure is the first half of the second week. The beginning of the trouble is marked by pain and paranæsthesia; soon there is numbness of the hands and feet, extending to the forearm and lower leg; very rarely is the paralysis of greater extent. Pain, which is seldom absent, may be very severe (anæsthesia dolorosa); it may be either constant or intermittent. The paranæsthesia has been variously designated by patients as a "sleepy," "furry," "itching," or "tingling" sensation. The hands and feet are cold, the tactile sense is disturbed as well as the temperature sense, cold water is mistaken for warm, while hot water is not recognized as such. As a rule loss of motility follows in a few days, but may also occur simultaneously with the paranæsthesia. In the majority of cases the patient cannot walk or stand for weeks, or even months, after the attack, but occasionally the effects soon disappear. The muscles first and chiefly affected are the extensors and abductors of the toes, later those of the fingers, and finally those of the lower and upper limbs. According to the investigations of Imbert-Gonebeyre the paralysis may be confined to the knees and elbows. Following the paralysis there is increasing muscular atrophy, which may reach an extraordinary degree of severity. Von Marik has described a case in which almost all the body muscles were affected.

Paralysis arsenicalis may be confused with tabes, alcohol neuritis, syringomyelitis, etc.

Asphyxia arsenicalis is a term given by several authorities to rare cases of exceptionally acute arsenic poisoning with unusually rapid death. In this form there is extraordinarily severe intestinal catarrh with vomiting, diarrhœa with rice-water stools, tenesmus, tonic convulsions first of the lower limbs, later of the upper limbs, cyanosis and death, with marked paralysis of the muscles of respiration in a few hours. This form of poisoning bears a striking resemblance to cholera asiatica of malignant type.

Chronic poisoning is seen most often in workmen in the metallurgical industries, in dye and paint works, among taxidermists, etc. A detailed list has already been given. Hill has also recorded a very unusual case, the alleged result of burning gas made from coal very high in arsenic.

According to Brouardel and Pouchet, who have made one of the most careful studies of arsenicism, there are four distinct phases in the progress of the disease. These phases correspond quite closely to the grouping given above under acute poisoning, but are more marked. The early stages of the disease can be characterized as protracted gastro-enteric catarrh. There are loss of appetite and vomiting of mucus and bile. Vomiting and pain may, however, be absent. The patient is apt to suffer from headache, faintness, irregularity of the bowels, depression, and loss of sleep. The breath is fetid, and the perspiration exhales an alliaceous odor like that of arsine. There is great emaciation. Fever and œdema may or may not appear. The urine is albuminous, contains hyaline cylinders, and substances reducing Fehling's solution. Sugar is rarely present. The urine contains arsenic, and on standing deposits magnesium-ammonium arsenate. A common accompanying symptom of much diagnostic value is obstinate conjunctivitis, together with redness and burning of the eyelids, and defective tear secretion. The throat and nasal passages are sometimes dry and irritated. The voice is hoarse. There may be slight cough, which, in cases arising from breathing arsenical dust, passes into more or less severe bronchitis. At other times the victims suffer from an influenza-like snuffle and the nasal secretions are flecked with blood. Many cases of arsenicism have been diagnosed as "grippe," "cold in the head," etc. The skin is far more severely affected than in acute poisoning. There is generally urticaria or an erythematous eruption, or desquamation. Sometimes papules or vesicles form on the breast, back, and face, while later pustules may appear in the arm pits, upon the scrotum, or in the vulva. Frequently the face takes on a dusky tint, and there is marked pigmentation of most of the exposed parts and the pressure areas of the body, or this pigmentation is confined to the knees, about the neck, and to the region of the stomach. The victim appears to be dirty. So dirt-like is this pigmentation that patients upon their admission to hospitals have been given several successive baths. The hair first turns gray, then falls. There may be growths on the nails; the latter may even be shed. Hyperkeratosis of the hands and feet of short duration is seen in not a few cases. Following the typical symptoms of cachexia and muscular atrophy there is wasting away of the muscles, especially of the lower limbs, though the arms may also be affected and the hands become claw-like. Icterus is neither constant nor characteristic, yet is frequently observed. It never reaches the intensity met with in poisoning by phosphorus. True paralysis arsenicalis seems to be of less frequence in chronic poisoning than in acute; but if it does appear its effects are of greater duration, lasting even a life-time. There are often hemianæsthesia and hemiplegia. According to the reports of cases of poisoning from arsenical beer, published in the *Lancet* in 1900–02, it would appear that a large percentage of the victims suffered from more or less marked paralysis, as shown by difficulty in walking, numbness, pricking, tickling, etc., of the lower limbs from the knees down, and from the elbows down, often accompanied by exquisite pain.

Mackensie has reported cases marked by paralysis of the vocal cords. Another peculiar form of paralysis has been studied by Charcot and Biett, and has received the designation anaphrodisia arsenicalis, because of the fact that the disease is characterized by a paralysis of the nerves of sexual sensation. Five typical cases are known. The physical diagnosis of chronic arsenical poisoning is very difficult. The most reliable method of reaching a decision consists in testing the urine for arsenic. This chemical diagnosis must never be omitted. The fatal period in chronic poisoning is too variable to permit of any statement.

Poisoning by arsenic may sometimes be confused with poisoning by phosphorus, antimony, ricinus, croton, abrus, robinia, podophyllum, etc.

Elimination of arsenic takes place chiefly by the kidneys; but there is reason to believe that in addition to the urine, the bile, milk, and perspiratory fluid play an important part, and that a certain portion of the poison is still further thrown out through the intestines. Arsenic can be detected in the urine in from two to eight hours after resorption, and is usually completely eliminated from the body, save from the bones, in from eight to twenty days. The normal period of elimination seems to be from twelve to fifteen days. This holds true only for acute poisoning, since in chronic poisoning, followed by recovery, the liver seems to retain arsenic for a long period; just how long is not known, but in animals it has been shown to be beyond forty days after the ingestion of arsenic ceased. Wood asserts that he has found arsenic eliminated eighty to ninety days after ingestion. On the other hand, Kunkle reports a case of poisoning by copper arsenite followed by death in seventeen hours, yet no arsenic could be detected in the liver, the stomach, or the intestines.

Saveri has shown that the arsenic eliminated by the urine is in the arsenous condition. Selmi has gone a step farther and has shown that part at least of the arsenic is eliminated in combination with organic matter as an arsenical organic compound, and that doubtless another portion of the arsenic is eliminated as arsine. The question of the elimination of arsenic through the milk is one

of very great importance. In the light of experimental evidence and of cases collected, it is now possible to assert positively that an infant nursing at the breast may die of arsenical poisoning when its mother is undergoing arsenical treatment, or has swallowed some arsenical compound yet not in sufficient amount to give rise to illness of the mother; or, in case of a toxic dose having been ingested, there may be death of the infant before the appearance of symptoms of poisoning in the mother.

For information as to the localization of arsenic and its distribution in the body after death, the reader is referred to Vol. VI., p. 722, of the REFERENCE HANDBOOK.

Post-mortem Appearances.—In acute poisoning the findings are but slightly characteristic. In rare cases negative results will be obtained, but only when death has been unusually rapid, as for example in cases of arsenical asphyxia. No matter through what channel the poison has been introduced, the mucosa of the stomach and intestines will generally be found to be swollen, covered with bloody foam, more or less uniformly red, intensely inflamed, or hemorrhagic. In some cases there is ulceration, even necrosis. A few cases of perforation are known. Corrosion may take place in a few hours (five or six), and after ten hours may be very severe; this may not be confined to the stomach alone, but may be seen in the intestines. The small intestine is commonly more or less filled with colorless, watery, offensive contents, while the large intestine contains fæcal matter colored with bile. The liver, though apparently normal, shows on section marked fatty degeneration. Similar conditions obtain in the kidneys and heart. The blood shows no specially marked change, but its alkalinity is greatly decreased. In all but a few cases there is a remarkable preservation of the body with the internal organs fresh and of almost life-like consistence, or there may be mummification. Either of these conditions existing in an exhumed unembalmed body should lead to a strong presumption of death by arsenic, but the practitioner must never lose sight of the fact that preservation and mummification are far from being constant or peculiar to this poison. In deaths following chronic poisoning the findings are quite uncertain. The most constant are atrophy of the muscles, a dusky skin often marked with blackish patches, alopecia, gastro-adenitis, and hepatic, renal, and cardiac steatosis.

The *mechanism* or action of arsenic compounds can be briefly summed up as follows: (1) Local cauterization; (2) vaso-motor paralysis of splanchnics; (3) degeneration of albumin of the body and disturbance of metabolism; (4) disturbance of activity of the central nervous system and of the skin; (5) paralysis of the heart.

Following the paralysis of the peripheral extremities of the splanchnic nerves there results great hyperæmia of the abdominal organs, giving rise to an appearance of inflammation. This condition extending to the mucosa becomes severe gastro-enteritis, explaining why it is that these lesions are seen, no matter what may have been the mode of entry of the poison. The disturbance of the metabolism leads to fatty degeneration of most of the internal organs, and especially of the glands. When this steatosis affects the walls of the blood-vessels, multiple hemorrhage results. As an immediate consequence of fatty degeneration of the heart muscles the ganglia become affected and cardiac paralysis results. The severe cerebral disturbances sometimes seen can be ascribed in part to the sinking of the blood pressure, and in part to degeneration of the ganglionic cells of the brain.

Normal Arsenic.—The question as to whether arsenic is a normal constituent of the body has been the subject of several investigations within the last few years. The weight of evidence seems to support the theory of Armand Gautier, that arsenic is always to be found in the healthy body at least in the thyroid, where it plays an important part, and in the hair, nails, bones, and mammary glands, and that this normal arsenic is eliminated chiefly by the skin and the milk. Before this theory can be accepted as proved more research is needed.

ANTIDOTES. TREATMENT.—The most effective antidote with which we are now acquainted—freshly precipitated ferric hydrate—was first proposed by Bunsen in 1834. It depends for its efficacy upon the fact that it unites with arsenous acid, arsenites, or arsenates to form insoluble compounds. With arsenous acid or arsenites a basic ferric arsenite results, mixed with a variable proportion of basic ferrous arsenite, while with arsenates a basic ferric arsenate is formed. The ferric hydrate must be freshly prepared, since it soon loses the power to unite to form an insoluble compound. The official method of preparation is as follows: To ammonia water (U. S. P.), 110 c.c., add water 250 c.c.; to solution ferric sulphate (U. S. P.), 100 c.c., add water 1,000 c.c. Pour with constant stirring the solution of ferric sulphate into the ammonia water, pour the mixture on a muslin filter, squeeze, and add to the precipitate sufficient water to make about 250 gm. Of this final preparation administer to an adult two to four tablespoonfuls every ten minutes. The dose for a child is one dessertspoonful every ten minutes. Hydrated magnesium oxide, proposed by Bussy in 1846, is also of much value. Like the iron antidote it must be freshly prepared. One part of freshly burnt magnesium oxide is suspended in twenty parts of water. Of this preparation four to six tablespoonfuls may be administered every fifteen minutes. As in the case of iron, insoluble basic arsenites and arsenates result. In Europe the two above antidotes are combined, as, for example. To Liquor ferri sulph. oxydat. (P. G.) 100 gm., add water, 250 gm. To water, 250 gm., add magnesia usta, 15 gm. Mix the two solutions, shake well, and throw upon muslin.

Both the iron and magnesium compounds must be removed from the stomach as soon as possible, since the insolubilized arsenic may be again resorbed through the solvent action of the fluids of the body. A recommendable procedure is to wash out the stomach with water holding magnesium oxide in suspension; then administer the antidote. After a few minutes induce vomiting, or use the stomach pump. Repeat this process several times. Demulcent drinks should be promptly given to retard resorption—milk, albumen in water, flour and water, etc. Following the antidote, the treatment must be symptomatic. It must never be forgotten that during convalescence the diet must be watched with great care, and only bland articles given.

In the absence of the above-mentioned antidotes, ferric hydrate, prepared in any way, can be given, or the following may be employed with more or less success: saccharate of iron, acetate of iron, citrate of iron, dialyzed iron, powdered or reduced iron, milk of lime, sulphur, sulphide of iron, etc. The administration of alkalies must be carefully avoided.

POISONING BY ARSINE is probably of greater frequency in the industries than is generally believed, though acute poisonings are quite rare. Arsine or arseniureted hydrogen is a heavy colorless gas of peculiar fetid odor. Its specific gravity is about 2.7. It is but slightly soluble in water, and burns with a characteristic bluish-white flame. The pure gas is a frightfully active poison, only a whiff or two causing death. Of some twenty cases of acute poisoning on record, practically all have been the result of chemical laboratory accidents, but subacute and chronic poisonings are frequent in the industries where small amounts of arsine are breathed in the air of ill-ventilated work-rooms. Although the odor of arsine is characteristic and very penetrating, the gas may be present in the air of a room in sufficient amount to lead eventually to poisoning, and still not be detected by the sense of smell, or at least not noticed. Poisoning occurs chiefly in the preparation of hydrogen and the manufacture of balloons or toy balloons; in the coloring or bronzing of brass and other alloys; in the desilvering of lead and subsequent treatment of the silver zinc with acids; in the galvanizing and tinning of sheet iron; in the reduction of nitrobenzene, etc., in the aniline industry; in the manufacture of many salts; in fact, in all industries or operations where hydrogen is given off in reactions between compounds containing arsenic as an impurity. Besides these sources there is the further possibility of

poisoning from wall papers in damp, ill-ventilated rooms through the formation of arsine by fungi and bacteria.

In strictly chronic poisoning the specific action of the poison manifests itself in anæmia or pernicious anæmia.

The effects of arsine on the system appear slowly. The victim shivers, complains of chills, and often of an indescribable feeling of sickness passing into great uneasiness and fear, resulting in exhaustion, weakness, and repeated fainting fits, during which the body is cyanotic, cold, and bathed in cold sweat. There is a blackness before the eyes and the pupils are dilated. There may or may not be nausea and vomiting. The pulse is somewhat accelerated but small and weak. Respiration is dyspnœic. The breath and mouth are fetid. After eight or ten hours, or even much later, the specific effects of the poison manifest themselves; the red blood corpuscles are destroyed and methæmoglobin is formed. All the urine passed is bloody, deep red, dark brown, or even black. There is sometimes anuria. Polycholia sets in and the skin becomes icteric or dark-colored. The stools are not watery as in arsenicism, but are colored dark with bile. The liver and spleen are swollen and painful on pressure. There are severe headache and a fear of death. Death occurs from œdema of the lungs or from paralysis of the heart. If death does not result the period of convalescence is generally very long. Spectroscopic examination of the blood for bands of methæmoglobin is of great value in diagnosis. These characteristic bands will also be noted after death, providing that an examination is promptly made.

The treatment of cases of arsine poisoning is difficult and unsatisfactory. Sodium bicarbonate should be administered to convert the methæmoglobin into alkaline methæmoglobin, which the body can readily change to oxyhæmoglobin. Transfusion of blood may often be imperative. The clogging of the canals of the kidneys has been successfully treated by injection of normal salt solution. The remaining treatment must be symptomatic. In the choice of stimulants to counteract the severe depression of the heart's action it should be remembered that alcohol is barred because of its action on the kidneys.

Clinical Tests.—For the rapid detection of arsenic in the urine, vomited matter, stools, etc., where much organic matter is present, there is nothing superior to the Reinsch method, *i.e.*, acidification with pure HCl and boiling with a tiny strip of pure copper foil or gauze. The stained copper should then be tested by heating in a glass tube in contact with air and the crystalline sublimate of As_2O_3 examined with a hand lens or microscope. For inorganic material the modified Gutzeit test will be found to be rapid and reliable. The gases evolved by the action of sulphuric or hydrochloric acid on zinc in the presence of a solution of the material to be tested are passed through cotton moistened with lead acetate (to hold back any hydrogen sulphide), and are tested either with filter paper moistened with mercuric chloride or with a dry crystal of silver nitrate. In the presence of arsenic the mercuric chloride spot turns brick-red, orange, or brown; the silver nitrate crystal first canary or lemon-yellow, then black. *Emile Monnin Chamot.*

ASIATIC CHOLERA.—(Synonyms: Epidemic cholera, Cholera asphyxia, Algid cholera, Malignant cholera, Cholera spasmodica, Pestilential cholera, Pestilential asphyxia, Oriental cholera, Choleric pestilence, Indian cholera, Ganglionitis peripherica et medullaris, Trisplanchnia, Hyperanthraxis, Morbus oryzeus [because supposed by Tytler to be due to damaged rice], Troussegalant, Cholera gravior, Vishucki or Vishuchiki [by Hindoo physicians], Haouwa [tornado] in Bagdad.) (For a wider discussion of terminology, see Macpherson, "Annals of Cholera," chapter ii., ed. 1884.)

The derivation of the word cholera is usually from χολὴ ῥεῦ (flow of bile), or χολὰς ῥεῦ (intestinal flux), but the correct one is probably that given by Jobard, of Brussels (*Gaz. Méd. de Paris*, 1832, p. 389), who considers the term to be made up from two Hebrew words, choli-ra (or morbus malus).

Asiatic, or epidemic, cholera is an acute infectious disease that is endemic in certain parts of India, and that has during the last century advanced out of that country to other parts of the world, where, in its epidemic form, it has produced great loss of life. It is characterized by its great fatality among the communities to which it may be transported, by the apparent ease with which it has been carried from place to place, and by its invariably following the lines of travel in its march from one place to another. It is distinctly a disease of the gastro-intestinal tract, produced, primarily, by a micro-organism and attended with secondary symptoms, due to the absorption of toxic principles elaborated during the development of this micro-organism.

There is only a difference of degree between cholera, choleraic diarrhœa, and cholerine—the disease is the same in all these forms provided that they are accompanied by the activity of the spirillum of Koch. So far as true cholera and cholera nostras are concerned, there is a very great similarity, or rather there may be, between the clinical symptoms of the two diseases, but the differentiation may be easily made by the isolation of the specific spirillum of the former. The same thing is true in regard to the differentiation of true cholera from an attack of indigestion, which, if severe enough, may take on many of the characteristics of true cholera. Cholera nostras is a seasonal disease and is not transportable, and it has been known for ages. True cholera made its first advance out of India in 1817, and since that time has been seen periodically in Europe. In all cases it has followed the line of travel, and has never been seen to be distributed in any other way. Therefore true cholera never makes its appearance except after other cases have been seen that might excite suspicion, while cholera nostras appears only in hot weather, in sporadic cases, and dies out if the weather becomes cooler.

The period of cyanotic chills, although one of the most striking in both diseases, gives no special indications for differential diagnosis, for the same thing is seen as an accompaniment of many other diseases, as acute catarrhal diarrhœas, acute poisonings, etc. The prodromic period exists in cholera nostras practically always, but not nearly always in true cholera.

The period of reaction is of importance in differential diagnosis. In cholera nostras it is usually benign—if the patient escapes the violence of the first attack, convalescence is as a rule easy and rapid; with true cholera, on the contrary, convalescence is exceedingly dangerous—full of pitfalls, and may terminate fatally at any time.

Mention of the disease is made in Sanscrit and Chinese writings. It is spoken of by Hippocrates (Epidemics), and successively by Aretæus, Celsus, Galen, Caelius Aurelianus, Aëtius, Paulus Æginatus, and Alexander de Tralles. All of these writers, with many others, refer to affections resembling the cholera, but it is not until the seventeenth and eighteenth centuries that we find descriptions of the *epidemic* disease. Genuine epidemics, analogous to cholera, are described by Rivière, who made his observations at Nîmes in 1564, and by Zacutus Lusitanus, who saw several in different parts of Europe in 1600. The most remarkable accounts by authors of this epoch are those of Willis ("Opera Gen.," 1680, t. xi., p. 74), describing epidemics in London, in 1670, of what he called "dysenterica aquosa epidemica," of Thomas Sydenham, in 1669–76 ("Oper. Med.," Geneva, 1723, pp. 106 and 184), and of Torti ("Therap. Spec.," liv. iii., cap. ii., and liv. iv., cap. j.). Bontius ("De Medic. Indorum.," Lugd. Batav., 1642, p. 136), Delon ("Voyage aux Indes Orient.," Amsterdam, 1684), and Thevenot ("Voyage aux Indes Orientales," Paris, 1689, tom. iii.) observed and described epidemic cholera in India. In 1761, Donald Monro ("An Account of the Diseases in the British Military Hospital in Germany," London, 1764, p. 97) saw an epidemic of cholera in Westphalia; as did Agton Douglass and Bisset, in 1768, in the north of England and in Scotland. Harlem ("Die Indische Cholera"

1831, t. i., s. 144) quotes many dissertations upon the disease, but it is not until the last century that we have a clear account of the transportation of the disease from place to place. From 1817 it seemed to take on a new power of travelling, and owing to this spread the opportunities for its study have vastly increased. The new methods of intercourse and commerce were probably responsible for the appearance of the disease in Europe—not any new property which it developed.

A study of the history of the epidemics that have occurred outside of India will easily demonstrate the facts in regard to the ways by which the disease is transported from place to place.

History of Epidemics of Cholera and Their Lessons.—The dispute is active as to whether true cholera existed in India before its appearance outside of its limits in 1817, but the probabilities are all in favor of its having done so, epidemics of considerable proportions being reported in the eighteenth century; the especial point that seems to be changed in its nature being that it then seemed to take on the property of migration. Whether this was in reality a new property, or whether, as is much more probable, it was simply brought to the notice of Europeans by their being first attacked by it, is an unsettled question. There is no doubt, however, of the very great influence exerted upon its spread by the great pilgrimages to the various shrines of India; nor is there any doubt that the sole home of true cholera—the one place where it is present all the year in an endemic form—is the delta of the Ganges. There are also certain places in India, Indo-China, China and Japan in which it seems to be present nearly all the time, but it certainly is not endemic in Persia, on the borders of the Caspian Sea, nor in Mecca.

From this one place in which it is endemic, cholera has always been transported to Europe in the steps of the traveller and along the routes of commerce; neither wind, moisture, electricity, nor any of the forces of nature have taken any active part in the actual transportation of the disease, although unfavorable climatic and hygienic conditions, of course, may play a favoring part in the development of the disease, after the arrival of its cause.

Epidemics of Cholera.—Cholera has made five appearances in Europe—in 1830, in 1846, in 1865, in 1884, and in 1892. Each one of these appearances was a great epidemic.

There had been also, in 1823, in Astrachan, a small epidemic of cholera, important because it traced the route that the succeeding invasions would follow. Leaving Persia, where it prevailed in 1822, cholera invaded the southern provinces of Persia, forming the southern shore of the Caspian Sea. After some ravages, it became quiescent during the winter of 1822, to reappear in April, 1823, at Recht. From this city, following the western shore of the Caspian Sea, it crossed the Russian frontier in June, at the little town of Astara. From Astara it reached Lenkoran on the 29th of June. On the 11th of September it was seen at Bakou, and on the 22d at Astrachan, where it soon disappeared.

The First Epidemic.—The epidemic of 1830 had the same origin. Ghilan and Mazanderan, the two Persian provinces before invaded, were attacked in 1829. The disease was quiescent during the winter, but appeared in the spring in Ghilan and in the little port of Enselli, situated several hours' journey from Recht. As in 1822, the cholera followed the western border of the Caspian Sea, and showed itself about the middle of June, 1830, at Salian. Taking here two different directions, on the one side it appeared at Bakou, Kouba, and Derbent, and invaded Astrachan; on the other, following the whole valley of the Koura, it advanced toward Tiflis, passing by Elizabethpol, and spreading throughout the whole of the Caucasus. In this way it reached successively the neighboring regions of Astrachan, and advanced up the Volga. On the 4th of August it was at Saratow, thence extending into Russia, and reached the other European States.

This epidemic, by certain extremely interesting peculiarities, demonstrated from the very first the transportability of cholera.

The Second Epidemic.—In 1846, after having reached Salian by a course identical with the preceding, cholera was seen on November 8th in the city of Chemacka, a short distance from Salian. It was in Bakou and at Derbent in December. Forgotten during the winter, it appeared in April, 1847, in the districts of Derbent and of Kouba, and at Tamir-Khan-Choury. From thence it was transported by sick soldiers to the mineral waters of Kisliar. The disease was disseminated among the Calmucks scattered over the steppes near the Volga.

On July 15th it appeared at Astrachan, and advanced at the same time toward Tiflis. From Tiflis it reached Koutais, and was soon carried to Trebizond.

North of Tiflis, the cholera followed the great military road that crosses the Caucasus at the height of seven thousand feet, and toward the end of July it existed at Stavaropol, on the other slope. (It is to be observed that before reaching Tiflis, the cholera entered Persia by the great routes of travel that pass from Bakou, by Erivan, Natchichievan, Djoufa, Ordoubaz, and on toward Tauris. On the one side it attacked the region of the Black Sea, and invaded all its ports; on the other, it passed through Russia, Germany, France, and Italy.)

A striking thing about these epidemics, aside from the exact places that they attack, is their progress by successive stages—a form of advance that is always the same, and which is a trait common to all the epidemics of cholera that have followed the land route. This second epidemic persisted until 1855.

The Third Epidemic.—The great epidemic of 1865 was the first one appearing by the sea route. It demonstrated that the danger is not localized on the Caspian Sea, but that it is also present on the shore of the Red Sea. Its appearance by this route upset all the doctrines that had been held until that time, and the panic that it produced in Europe resulted in the first conference at Constantinople. It is interesting to follow in some of its phases the course of this epidemic, because its influence has been great. It started from Mecca, having been brought into that city by ships coming from India filled with pilgrims. Toward the end of April it broke out in Mecca and at Medina. The mortality increased very greatly during the three feast days at Arafat. More than thirty thousand of the pilgrims died of cholera, and the progress of the disease showed that *everywhere it accompanied these pilgrims*. Egypt, by reason of its proximity to Mecca, was the first country attacked.

From May 19th to June 10th ten steamers landed from twelve to fifteen thousand pilgrims at Suez. By false declarations from the captains they were passed at Suez, although the *Sydney*, an English steamer, had lost a number of cases during the voyage. The first steamer, landing May 19th at Suez, had thrown its dead into the sea. On the 21st, cases appeared at Suez, and among the number were the captain of the vessel and his wife.

June 2d, the first case appeared at Alexandria, and in two months cholera had four thousand victims in Alexandria, and in Egypt, in less than three months, it produced the death of more than sixty thousand individuals The foreign population emigrated *en masse*, and carried with them throughout the entire world the germs of the disease. Europeans and Levantines, to the number of from thirty to thirty-five thousand, started for all the ports of the Mediterranean, and cholera developed at Constantinople, at Smyrna, at Beyrout, in Mesopotamia, and at Odessa on the Black Sea, and was carried to New York and Guadaloupe by steamers, *appearing in the port at the same time that the steamer landed its passengers*.

Its importation into New York was as follows: The *Atlanta*, an English ship, left London on October 10th with a cargo of merchandise and forty passengers. The sanitary condition of London was at that time excellent. Reaching Havre on the 11th, where it remained one day only, it embarked five hundred and sixty-four new passengers, mostly Swiss, who had all passed through Paris, where, with certain exceptions, they had remained at