the pathologist simplify matters considerably. If this plan cannot be followed, it devolves upon the pathologist to remove such organs and fluids in which the suspected poison may be detected, in such a manner as to avoid contamination, and to place them in possession of the chemist with the least possible delay and by as direct means as circumstances will allow. It should be remembered that every step from the performance of the autopsy to the reception of the material for examination by the chemist may be made the subject of a searching examination in court, namely, how the organs were removed, what instruments were used and what condition they were in, what receptacles the material was placed in for transportation, how these receptacles were treated before and after the material was placed in them, whether any preservative fluid was used, were they properly sealed, and what means were employed in transporting them to the chemist. These points may appear trivial, yet too much attention cannot be paid to the minutest detail. Carelessness in this regard may nullify the entire work of the chemist and cause a break in an otherwise intact chain of evidence. On the other hand, such carelessness may be the means of unknowingly convicting an innocent defendant. Glass jars that have been used for no other purpose, carefully cleaned with soap and water, rinsed with water, then with alcohol, closed preferably with a ground glass stopper or clean, well-fitting cork, should be used. Stomach or intestinal contents, urine, and blood should be placed in separate jars. When the determination of the amount of poison in individual viscera is of importance they should be placed in separate jars. The condition of preservation or decay of the material should be noted. If the material cannot be placed in the hands of the chemist directly, a sufficient amount of strong alcohol should be added to cover the organs in the jars, in order to check decomposition and prevent breakage by the gases of decomposition. If this is done a portion of the same alcohol that was used in the jars should be sent to the chemist also. Finally all jars should be separately tied with tape, properly sealed and labelled. By this means, although the material may have necessarily passed through several hands, provided it reaches the chemist with intact seal, the chain of evidence, so far as the examination of the organs, fluids, etc., is concerned, remains unbroken.

Cases of Poisoning.—The evidence already at hand before the performance of an autopsy may point to a given poison as the cause of death, either from the clinical history of the case, or from the discovery of the empty poison bottle or package, or of some of the poison suspected to have been given to or taken by the deceased. This evidence, although a valuable guide, cannot be entirely relied upon and should not bias the judgment of the pathologist. Cases occur in which an entirely different poison is found to have been the cause of death, and some in which the presumptive evidence appeared very strong have proved to be deaths from natural causes, or the alterations found have turned out to be merely post-mortem changes. Cases occur in which there is no suspicion whatever, and yet poisoning is proved by autopsy and chemical examination. Such are not merely cases of sudden death without any clinical history, but often enough cases that have been ill for some time and treated for disease by a physician, competent enough comparatively, who may have filed a death certificate giving the disease diagnosed by him as the cause of death. In fact, it is well known that the results of some poisons may so closely resemble disease clinically and even pathologically that mistakes can easily be made. The only safe way to avoid error is for the clinician to insist upon an autopsy before signing the death certificate, and for the pathologist to employ chemical and bacteriological aid. In the performance of autopsies, whether poisoning is suspected or the cause of death is unknown or doubtful, a complete examination of the body should be made; and the pathologist should be ever mindful of those conditions, although often resembling the results of diseases such as cholera, dysentery, nephritis, malignant jaundice, or acute yellow atrophy of the liver, which may nevertheless be the results of poisons such as arsenic, mercury, potassium chlorate, or phosphorus.

Some poisons produce no characteristic changes in the tissues of the body, so far as gross or even microscopical examination is concerned; their presence cannot be positively affirmed until chemical examination is made. The pathologist may, however, be able to state that no pathological condition of the organs due to disease or traumatism, and sufficient to cause death, has been found. When the organs are found in a normal condition it may be easy enough to reach this conclusion. When, however, pathological changes are present, it is often very difficult to estimate their importance in the causation of death. Although our knowledge of the morphology of disease, and of the bacteriology of many of the infectious diseases, is extensive, that of its chemistry is not nearly so well advanced. Pathological conditions are found which in the absence of any suspicious circumstances are assumed to be the cause of death; yet we meet with cases in which these conditions are present and may even be very pronounced, but nevertheless death is the result of violence or of some other intercurrent disease. When the circumstances of a death are suspicious, a chemical examination is usually called for. There are cases, however, of sudden death in which no suspicions are entertained at the time, but in which, though a careful autopsy is performed, a conscientious pathologist cannot satisfy himself of the cause of death, even with the aid of microscopical and bacteriological examinations. In such cases a chemical examination alone can affirm or exclude poisoning.

Other poisons do produce effects more or less characteristic by their direct local action, by their absorption, and by their excretion. The task in the performance of the autopsy is to determine whether the changes found may have been produced by a poison, and if possible by what poison. Of course, the positive proof in every case must be furnished by chemical examination. The work of the pathologist, however, is of importance, on the one hand to obviate useless chemical examinations, on the other to insist upon a chemical examination when necessary, and to select such organs and fluids of importance for examination. On this account it is important to know what changes are characteristic of poisoning, and what are characteristic of special poisons.

Certain physical characteristics of poisons may lead to the suspicion of their presence. A green color may indicate the presence of aceto-arsenite of copper; yellow, potassium chromate or iodine; blue, sulphate of copper; or certain dyes, as such used in corrosive sublimate tablets, or in the heads of matches, may furnish an indication. The odor characteristic of phosphorus, or of bitter almonds, of alcohol, of chloroform, of laudanum, or of carbolic acid may furnish an indication. The granular or crystalline appearance of the substance, its insolubility, may furnish a clew. The chemical reaction, whether acid or alkaline, is important, and the contents of stomach and intestine should always be tested in this regard.

In the greatest number of poisoning cases, the poison is introduced by way of the mouth; unusually, by rectal, vaginal, intra-uterine, or hypodermatic injection. Some poisons produce no effect upon the mucous membrane; others are irritants and cause effects varying in intensity from congestion and ecchymoses to complete corrosion with production of eschars. Those poisons which produce the corrosive effect upon the mucosa may act either by coagulating its albuminous constituents—as happens in the case of the mineral acids, oxalic acid and carbolic acid, and mercuric chloride—or by dissolving them and causing a swelling and softening of the mucosa—as is true of sodium, potassium, and ammonium hydrate and potassium cyanide. Concentrated sulphuric acid dissolves coagulated albumen; if dropped on a mucous membrane a spot is formed that is transparent in the centre and white at the periphery where the acid has been diluted by the fluid of the tissue and the albumen precipi-

tated. When the caustic alkalies have acted upon a mucous membrane, if neutralized or if the reaction is changed by addition of acid, the albumen is precipitated and a grayish eschar appears.

These effects may be further changed in appearance by the action of the poison upon the blood with which it comes in contact. If the poison separates hæmatin from hæmoglobin and dissolves it, the eschar or the tissue may be discolored brown or brownish black by imbibition, as with sulphuric acid, hydrochloric acid, oxalic acid, and the caustic alkalies. Carbolic acid and corrosive sublimate coagulate blood but do not cause a separation of hæmatin. Each, however, produces a change of color in the coagulum, namely, carbolic acid a bright brick red, and sublimate a grayish violet.

The effects may vary according to the amount of the poison, its concentration, the duration of its action, and the condition of fulness or emptiness of the stomach and intestines. The lips and skin of the face and neck may show corrosive action of the poison. The mucous margin may present grayish-white or brownish eschars. Crescentic streaks on either side of the upper lip, extending upward from the corners of the mouth, may be present when the poisonous fluid has been imbibed from a tumbler; there may be streaks from either corner of the mouth passing downward over the cheek or chin, and down the neck, when the poison has been spilled while drinking. Corrosion of the lips and skin may be absent when the poison has been swallowed from a bottle. The mucosa of the mouth may show swelling and eschars, but from short duration of contact these may not be well marked. The mucosa of the œsophagus may show little action from the short duration of contact and the relatively slight amount of poison that remains in contact.

The stomach usually shows the greatest amount of change. This may affect its entire surface, or be confined more especially to the region of the fundus, greater curvature, and posterior surface. Exceptionally, the corrosive action may be confined to the smaller curvature and anterior surface, the most probable explanation being the ingestion of poison upon a full stomach, which shortly thereafter has emptied its contents into the duodenum, in which case the duodenum and jejunum show the effects more markedly. The summits of the folds in the mucosa show more decided effect on account of greater exposure, the sulci being in part protected by contact of opposite surfaces. Thus the eschars in the stomach form longitudinal streaks separated sometimes by intervening mucosa not escharotic or less markedly so. The corrosive action varies in depth and may extend through the peritoneum, involving adjacent organs such as the spleen, colon, pancreas, and liver, without perforation of the stomach; or the stomach may have been perforated by the action of the poison, and its contents have escaped into the peritoneal cavity producing characteristic changes wherever the poison has come in contact. Autodigestion of the stomach with perforation may occur without the presence of corrosive poisons.

In the duodenum and jejunum the crests and superior surfaces of the valvulæ are especially exposed and show the greatest amount of corrosive action, usually more intense nearer the pylorus and becoming less severe further down. Exceptionally, the duodenum as well as the stomach may escape, and a coil of jejunum further along show severe corrosion. The ileum rarely shows the effect of direct local action, and the same may be said of the colon, except in those cases in which the poison has been introduced directly into the rectum. The caput coli and first portion of the ascending colon may occasionally show the effect of local action, probably from the longer duration of contact of poisons that have passed with greater rapidity through the small intestine. The colon and lower part of the ileum may show the effects of poison by excretion. This is a characteristic effect in bichloride of mercury poisoning, especially if a period of a week or two has elapsed after its ingestion.

The effects upon the tissues from the absorption of poisons is shown in the degenerative changes, parenchymatous or fatty, in the functional epithelial cells of the organs, as the stomach and liver; in the muscle fibre of the heart and sometimes of the voluntary muscles; and in the epithelium of the kidney, more especially of the cortex, when excretion of the poison has taken place.

Carbolic Acid Poisoning.—Eschars on the lips may be white, grayish, or, when drying has occurred, dark brown. The eschars on the cutaneous surfaces, if any, are usually brown, dry, and leathery. The mucosa of the tongue and mouth may be white or grayish white, or show no change. Pharynx and œsophagus usually show grayish-white eschars. There is generally more or less œdema of the aryepiglottic folds about the aditus laryngis and the loose submucous tissue over the arytenoids and anterior wall of the pharynx.

The eschars in the stomach are usually longitudinal, involving the crests of the folds, and of a white or grayish color, while the intervening mucosa, where not escharotic, will present a light red tint due to the action of carbolic acid on the blood. The entire wall has a dense leathery feel, and the stomach may be markedly contracted. The action of carbolic acid may extend to the peritoneal coat and even to the spleen and liver, the color usually being pink, or light red, upon a grayish-white base.

The distribution of effects varies according to the concentration and amount of the carbolic acid, and the condition of the stomach, whether empty or full when the acid was taken. The escharotic action may extend to a variable distance down the small intestine, the valvulæ of the duodenum and jejunum perhaps showing grayish-white eschars, while, further along, the mucosa may present a pink discoloration and marked swelling and softening. The other organs show but little change, in the great majority of cases, since death occurs within a few minutes. Passive hyperemia is usually present. When cases have survived the ingestion of a smaller amount for some hours, the characteristic phenol urine is found, with marked parenchymatous degeneration of the kidneys.

Sulphuric Acid Poisoning.—The eschars on the lips and skin are usually brown, leathery, and dry. The mucous membrane of the mouth and œsophagus presents grayish-white eschars. The stomach wall is thick and dense, the mucous membrane corroded, the eschars brown or black from the imbibition of dissolved hæmatin. Wherever the blood has been acted upon, whether extravasated or in the vessels, the coagulum is black, dry, and brittle. Either from the action of the acid or from casting off of necrotic portions of mucous membrane the surface may present an irregular nodular appearance. The fundus of the stomach may be perforated either during life or post mortem, and wherever the acid comes in contact with tissue a cloudy appearance is presented, due to coagulation of albumin. The mucosa of the small intestine may present a variegated appearance of grayish-white eschars, where the acid has caused a coagulation of the albumin of the tissue, with intervening dark brown or black areas, where ecchymoses have occurred or where a previous eschar has exfoliated laying bare the submucosa stained with hæmatin. The kidneys show parenchymatous degeneration or nephritis. In prolonged cases the necrotic mucosa of the stomach and intestine may be thrown off, showing a hemorrhagic, œdematous submucosa, with more or less imbibition of hæmatin.

Hydrochloric Acid Poisoning. — Hydrochloric acid produces no corrosive action on the skin. The eschars are grayish-white when simple coagulation of albumin has taken place, and dark brown or black when ecchymoses have occurred or when imbibition of dissolved hæmatin has taken place. The effect is very much like that of sulphuric acid, except that the drying of the eschars and of the blood clot is less pronounced, owing to the fact that hydrochloric acid has not so strong an affinity for water.

Nitric Acid Poisoning.—The eschars present a yellowish stain, due to the formation of xanthoproteic acid; otherwise they are not markedly different from those described above, except as regards the fact that nitric acid

does not separate and dissolve hæmatin. The brown or black discoloration of the eschars present with both sulphuric and hydrochloric acids does not occur.

Concentrated Acetic Acid.—A case of poisoning from this acid is on record; death was due to pneumonia, and a grayish-white corrosion of the mucous membrane of the mouth and respiratory passages was observed. A sponge saturated with concentrated acetic acid was held at the mouth and nose.

Oxalic Acid and Oxalate of Potassium.—In concentration they produce white or grayish corrosion of the mucous membrane of the pharynx and œsophagus. The mucosa of the stomach is swollen, injected, and escharotic, with imbibition of hæmatin; it is easily removed by washing. The escharotic action, however, is never as great as with sulphuric acid. White opacities (oxalate of lime) are found in the blood of the corroded portions, also in the uriniferous tubules. Perforation of the stomach may occur, with the resulting effect of a local peritonitis, due to the escape of the contents of the organ into the peritoneal cavity. These perforations, however, are in most instances a post-mortem occurrence. Wherever the poison acts upon the blood, black clots occur in which oxalate of lime crystals may be found.

Caustic Potash, Soda, and Ammonia.—The mucosa of the mouth may be swollen and red, the epithelium partly exfoliated, partly still adherent in whitish shreds. The œsophagus may have lost its epithelium, and may be swollen and hyperæmic, its lower portion being brownish and soft. The mucosa of the stomach is thrown into thick folds, markedly swollen and ecchymotic, with superficial losses of substance; it is dark brown, from imbibition of hæmatin. The submucous tissue is markedly œdematous. Croupous gastritis may follow the action of the caustic. Croupous bronchitis and circumscribed areas of bronchopneumonia may result from aspiration of the caustic soda or potash and may be the immediate cause of death. In other cases the cicatricial tissue following the exfoliation of the sloughs may finally, if not relieved surgically, cause death by stricture of the œsophagus.

Carbon Monoxide Poisoning.—The appearance of the cadaver, in poisoning by carbon monoxide, is very characteristic, more especially in the cases of poisoning from illuminating gas. The post-mortem spots are of a pink hue, and give the cadaver an almost life-like appearance. The blood is fluid and cherry red in color. The mucous and serous membranes are of a rosy tint, and the organs (especially those containing much blood) present a cherry red appearance. This is especially well marked where the blood is seen upon a white background, as in the brain. The color should be determined at once as soon as the blood or organs are exposed to the air, as after a longer exposure blood that does not contain carbon monoxide hæmoglobin may become light red in color, the reduced hæmoglobin changing to oxyhæmoglobin, the oxygen being absorbed from the atmosphere. The appearance of the blood in some cases of carbon monoxide poisoning, although carbon monoxide hæmoglobin is present, may present a dark color from excess of reduced hæmoglobin, from carbon dioxide absorption. This occurs more commonly in poisoning from coal gas, and from inhalation of smoke at conflagrations. The appearance of the blood may also vary when several hours have intervened between the cessation of inhalation of carbon monoxide and death. Since vomiting is a symptom of carbon monoxide poisoning, cases may die, in an atmosphere of carbon monoxide, from aspiration of vomit, the formation of carbon monoxide hæmoglobin being as yet insufficient in amount to cause death.

Carbon monoxide hæmoglobin is readily demonstrated by the spectroscope, producing two absorption bands near D and E like oxyhæmoglobin, but not reduced like the latter by addition of ammonium sulphide. In doubtful cases, therefore, a specimen of the blood should be saved for this examination. It has been found that carbon monoxide hæmoglobin can be demonstrated in the blood of extravasations and in muscle when its demonstration fails in the blood taken from the heart. Where cases have died in an atmosphere of carbon monoxide, or shortly after being removed therefrom, the blood resists decomposition for a considerable time, and the spectroscopic examination may be of value even after the lapse of two or three months. Such blood also keeps its bright red color. A note on the color and condition of preservation of blood that is taken for examination is of importance, since with decomposition (especially if ammonia is present in abundance) hæmatin is formed. Such blood is of dark color and becomes cloudy when mixed with water. The absorption bands are not clear, or there is only a shading in the green. On addition of ammonium sulphide two bands appear—*i.e.*, the spectrum of reduced hæmatin.

The differential diagnosis between illuminating-gas and coal-gas poisoning may not be easy to make. Cases of poisoning by illuminating gas present the most characteristic appearances post mortem and on spectroscopic analysis; those of poisoning by coal gas, from the larger percentage of carbon dioxide, present less characteristic appearances, and the spectroscope may show the bands at D and E, after the addition of ammonium sulphide, together with a more or less deep intervening band between them. In cases of death in conflagrations the effect of inhalation of smoke, as shown by the presence of black, sooty deposits upon the respiratory mucosa, is quite characteristic.

Besides spectroscopic analysis there are a number of chemical tests, very easy of application at the autopsy table, which prove of aid in doubtful cases. The addition of a drop or two of a ten-per-cent. sodium hydrate solution changes the color of other blood to a dirty brown or brownish green; carbon monoxide blood remains bright red. Solution of a copper salt changes the color of other blood to chocolate brown; carbon monoxide blood remains red. Tannin, ferrocyanide of potassium, and acetate of lead form a brown precipitate with other blood, a red one with carbon monoxide blood.

These tests, and also the spectroscopic test, may produce recognizable results in some cases in which death has occurred even sixty hours after exposure to carbon monoxide; in other cases, however, the reaction can barely be made out even when the interval amounts to only two hours.

A certain number of lesions which sometimes occur subsequently to carbon monoxide poisoning may aid in reaching a diagnosis where from the length of time between cessation of exposure and death the above tests fail. Croupous inflammation of the fauces has been noted where death followed seventeen hours after coal-gas inhalation. In some cases there are vaso-motor and trophic disturbances of the skin which predispose to necrosis from pressure. A case with dermatitis bullosa on both hands has been reported, death taking place five days after exposure to coal gas. In another case death occurred at the end of eight days. Symmetrical softening of the anterior part of the inner capsule and adjoining portion of the head of the caudate nucleus, also of the inner part of the lenticular nucleus, has occurred in a number of cases in which a day or more has intervened. Its occurrence has been explained by Kolisko on the ground of the peculiar course of the arterial branch (the long anterior perforating branch of the anterior cerebral) which supplies the part, its course being in the reverse direction to that of the artery from which it springs, so that with the decrease of pressure, which is the result of carbon monoxide poisoning, a diminution in the flow of blood or even stasis may occur.

Hydrocyanic acid.—This acid, alone, produces merely injection and ecchymoses of the mucosa of the stomach, which may in part be explained by the condition of asphyxia, death occurring rapidly therefrom. Besides the odor of bitter almonds there is nothing characteristic.

Cyanide of Potassium.—The mucosa of the stomach over its entire surface or at the fundus, or especially upon the crests of the folds, is deep red in color, swollen and softened, and presents sometimes almost a translucent appearance; a thick mucus, which is tinged a light red

or brownish red from blood, covers the surface. The stomach contents are usually blood tinged and stringy. The reaction is strongly alkaline. The mucosa is soapy or slippery to the touch. The characteristic odor of bitter almonds is present in the stomach, and also in other organs, as the brain and lungs. The odor of ammonia may be distinguished either from its presence in the cyanide, or through the effects of decomposition in the stomach. The redness and swelling of the mucous membrane are due to injection and ecchymoses, the primary effect of irritation, and to the secondary action of solution of the albuminous constituents of the tissue and imbibition of the superficial layers with hæmatin due to the strong alkaline action. The secondary effect, therefore, may be absent or poorly marked when the dose is small or when its effects have been counteracted by the acid contents of the stomach. In such cases, unless a characteristic odor is present, the diagnosis can be made only by chemical examination.

The same effects may be apparent in the mucosa of the duodenum, pharynx, œsophagus, larynx, trachea and bronchi, especially if, during vomiting, some of the potassium cyanide has been aspirated. In some cases the crests of the folds in the mucous membrane of the stomach may present a grayish-white appearance.

The blood usually presents the condition found in asphyxia, *i.e.*, dark and fluid. The spectrum is identical with that of oxyhæmoglobin and is reduced by ammonium sulphide. Occasionally it is light red. This color may be due, according to Hoffmann, to the hyperalkalinity of the blood, which is easily and quickly produced by the ammonia contained in cyanide of potassium, more especially in old samples. Traces of ammonia cause the appearance of a light red color in blood solution, and clarify turbid solutions. Others ascribe the light red color to the formation of a compound of cyanogen with methæmoglobin or hæmatin. If to a dilute solution of normal blood ferricyanide of potassium be added, there is an immediate change in color from red to brown, and in the spectroscope a methæmoglobin band appears between C and D. A trace of hydrocyanic acid or of cyanide of potassium will change the solution to a red color, and in the spectroscope there will appear in the green a broad band which, after the addition of ammonium sulphide, changes to two bands.

Nitrobenzol Poisoning.—The mucosa of the stomach and small intestine is injected and ecchymotic. The odor of bitter almonds in the stomach, brain, and lungs is even more marked and persistent than in hydrocyanic acid poisoning. The blood and muscle are brownish in color. From the presence of brownish methæmoglobin in the uriniferous tubules, especially in the pyramids, a resemblance to chlorate of potassium poisoning is produced.

Arsenic.—Arsenious acid usually does not produce corrosive effects upon the mucosa. These effects, although they have been observed in several cases, are evidently of rare occurrence. This poison, however, does produce an intense gastro-enteritis. There is nothing characteristic about the external appearance of the cadaver, or about the condition of the mouth, pharynx, or œsophagus. The mucosa of the stomach is intensely congested throughout or in patches; it is œdematous, swollen, and sometimes ecchymotic. It is covered with blood-tinged mucus, and scattered over its surface may be found granules or crystals of arsenious acid. These are sometimes large enough to be felt or even to be seen. The small intestine is filled with thin fluid, almost watery, with flocculi—the characteristic rice-water contents. The mucosa is congested, markedly swollen, œdematous, and flaccid. In the lower portion of the small intestine and in the large intestine, the mucosa may be pale. There is marked parenchymatous or fatty degeneration of the glands of the stomach and intestine, of the epithelial cells of the kidney and liver, and of the heart muscle. In some cases the stomach may present few or no changes, but the changes in the intestine are far more constant.

The appearance of yellowish streaks occasionally seen on the stomach mucosa are due to the formation of yellow sulphide of arsenic.

The colon may be covered with thick mucus containing desquamated epithelial cells and many lymphocytes. There may be a croupous colitis. The mesenteric lymph nodes may be swollen. There may be ecchymoses in the pericardium and pleura, but especially under the endocardium. The blood is usually poorly coagulated, and in the peripheral vessels may be thick and tarry, due to loss of water. In some cases there is slight jaundice, and ecchymoses may appear in the faucial mucous membrane and in the cellular tissue of the neck, but not in the muscle. When ecchymoses appear in the pleura and mediastinum, and fatty degeneration of the heart, liver, and kidney is present, the case may resemble phosphorus poisoning. The hemorrhagic spots in the mucosa of the stomach may become eroded by the gastric contents, and this doubtless explains why a corrosive action is ascribed to arsenic, which it most probably does not possess.

Arsenic is more rapidly eliminated than other metallic poisons, and it is conceivable that death may occur from arsenic poisoning and yet quite small amounts of arsenic be found. It is therefore of importance to preserve for examination in suspected cases not only the stomach and intestine and their contents separately, but also the heart, kidney, liver, bone, and muscle, since in some cases arsenic has been demonstrated in these organs, more especially in the liver and bone, when its demonstration has failed in the stomach and intestine or in their contents, by reason of its having been already eliminated. Much of the arsenic that has been taken internally may have been gotten rid of by vomiting and diarrhœa, common with arsenic poisoning. In cases in which examination is made after burial it is important not only to take portions of every organ and tissue of the body, inasmuch as it is well known that arsenic may diffuse itself through the tissues post mortem, but also to take samples of the objects surrounding the cadaver, including wood of the casket and surrounding earth. In addition to this it is also of importance to take another sample of earth from another part of the cemetery. All organs should be carefully weighed at the time of the autopsy, and if possible the entire organ should be given to the chemist. If this cannot be done, provided the weights of the organs are known, a basis for calculation of the amount of arsenic is furnished. It is both affirmed and denied that the cadaver after arsenic poisoning resists decomposition for a considerable length of time. Mummification has been described as a characteristic appearance. This may, however, be due to other conditions, such as burial in sandy soil, et cetera.

Aceto-Arsenite of Copper, Paris Green.—The appearance of this substance, its characteristic color, its insolubility, and the fact that it appears in the stomach in pasty masses, loosely adherent to the mucosa, which is swollen, œdematous, congested, and ecchymotic beneath the attached mass, renders the diagnosis of this form of poisoning quite easy. The small intestine shows the same appearance as in poisoning by arsenious acid. The Paris green may be covered by a brownish magma, the reduced iron given as an antidote.

Phosphorus.—Red phosphorus is not poisonous; the yellow variety is intensely so. Acute cases (death in from four to eight hours) may show but few pathological changes. The contents of the stomach and intestines may smell of phosphorus and may shine in the dark on being shaken; pieces of matches may be found. The gastric mucosa, heart muscle, and epithelial cells of liver and kidney may show cloudy swelling. The subacute cases (death after from three to seven days) commonly show characteristic changes. The stomach is not corroded, ecchymoses and hemorrhagic erosions are common; the gland cells, especially the adelomorphous cells, are in marked fatty degeneration, so that the ducts are marked by yellowish points (gastradenitis phosphorica). The contents may be dark brown from the presence of blood. Phosphorus may no longer be demonstrated chemically in the stomach and its contents, in the sub-