

is usually of short duration in the acute form of poisoning, although cases are recorded in which it has lasted fourteen and eighteen hours. It is frequently entirely absent, when large doses have been taken. This stage is marked by restlessness, great physical activity, loquacity, greatly increased imaginative power, frequently to the extent of hallucinations, always of a pleasing character, and by increased cardiac action. In adult males priapism sometimes occurs during this stage. Vomiting is also of occasional occurrence, and greatly improves the patient's chances of ultimate recovery.

The condition of excitation passes, sometimes rather suddenly, into an intermediate stage of diminished excitability. The patient becomes weary, incapable of physical exertion, dull, and drowsy. He complains of a sense of weight in the extremities and an irresistible desire for sleep, to which he finally yields if not kept awake. The sleep is at first seemingly normal, though profound. The pulse and respiration are normal. The patient may be roused and sometimes kept awake by shaking him, by the infliction of pain, or by loud talking. The face is pale, the lips are somewhat livid, the surface is covered with perspiration, and the pupils are contracted. During this period the patient frequently experiences a violent itching of the skin, which is sometimes followed by the appearance of an exanthem which may be papular, red, bluish, or almost colorless, or resembling those of urticaria or of scarlatina.

The condition of somnolence is of short duration, and passes quickly into the stage of narcosis. The patient can no longer be roused, even by the most violent means. He lies motionless and senseless, with eyelids closed or partly closed. The surface is bathed in profuse perspiration, which exhales the odor of opium in opium cases. The face is pale, the lips are blue, the lower jaw is dropped, and the muscles are completely relaxed. The pupils are insensible to light, and contracted to the size of pinheads until death occurs, when they dilate. A few cases are, however, recorded in which the pupils were said to be dilated. At first the superficial arteries, temporals, and carotids are seen to pulsate fully, strongly, and rapidly, while the respiration is slow and shallow (eighty pulsations in the minute to four respiratory movements have been observed). Later, the pulse becomes feeble, slow, irregular, and easily compressible. The respiration becomes slow, shallow, stertorous, and accompanied by mucous râles. Retention of urine occurs early in the history, and continues until death or recovery.

From this period, if the case do not yield to treatment, the poisoning usually proceeds rapidly to a fatal termination. The surface of the body and even the expired air become cold. The skin is cyanosed and covered with a cold clammy perspiration. The pulse becomes slower, more feeble, and gradually imperceptible. The respiration is more shallow and feeble, while the râles become more pronounced. Individual muscles, or groups of muscles, are agitated by short, clonic twitchings, and occasionally convulsions and tetanus occur. Later, the muscles become completely paralyzed, the respiratory movements are made at longer intervals and finally cease; the circulation continues after the cessation of respiration. Finally, the action of the heart is arrested and the patient dies quietly. Sometimes epistaxis and other hemorrhages occur toward the end; and in some instances death results from cerebral hemorrhage.

Should recovery follow after the stage of narcosis, the respiration gradually becomes more frequent and more natural, the pulse becomes first perceptible and then gradually passes toward the normal, while the condition of coma passes into one of deep sleep, which may continue for from twenty-four to thirty-six hours longer, although the patient can be roused.

In the great majority of cases in which recovery has progressed so far that the patient may be roused it will be complete. Nevertheless, occasional instances are recorded in which the victim has relapsed into a deeply comatose condition and has finally died.

In cases of recovery the patient, on awakening, is weary, giddy, and uncertain in his movements. He may also suffer for some hours from nausea and headache, and for a longer time from loss of appetite and derangement of digestion. In two cases Dr. Edes (*British Medical and Surgical Journal*, 1881, cv., 251) has observed the presence of casts in the urine; in one case, accompanied by albumin.

DURATION.—In cases of fatal poisoning by the opiates death usually follows in from twelve to eighteen hours after the poison has been taken. Of 48 fatal cases, death followed within twenty-four hours in 43; within eight hours in 39; within twelve hours in 26; and within nine hours in 20. The minimum duration of the poisoning was forty-five minutes; and the maximum, fifty-six hours.

Cases are recorded in which the symptoms of narcotism have disappeared and the patient has died at a greater interval of time than the above maximum. In such cases, although the death may have been accelerated by the action of the poison, the fatal result is immediately due to other causes. Thus, in a case quoted by Taylor, a patient suffering from disease of the heart took, in four hours, two hundred drops of laudanum, and was bled to the extent of thirty ounces. On the sixth day he was sufficiently recovered to undertake a journey, and died on the eleventh day. In this case the cause of death was failure of cardiac action, aggravated at least as much by loss of blood as by opium.

In cases of death in which the patient has suffered from a disease whose symptoms resemble those of opium poisoning, and has also received a large dose of an opiate, the question of duration may become one of considerable medico-legal importance. In a case which came under the author's observation a physician had given a child of four months gtt. x. of the Tinct. opii deodorata by mistake for Tinct. opii camphorata. The patient suffered from well-marked symptoms of opium poisoning, but under immediate treatment improved, and in forty-eight hours had apparently recovered. Death followed, however, in ninety hours from acute hydrocephalus.

Obviously, the same causes which influence the rapidity of action of the poison, and those which modify the effects of unusually large or small doses, will also influence the duration of the case, whether it terminate in death or in recovery.

LETHAL DOSE.—As the action of the opiates is much modified by conditions, such as age, habit, state of health, form of administration, and idiosyncrasy, it is impossible to fix a lethal dose applicable to all conditions. It may be said, however, that 0.065 gm. or gr. i., of morphin, or 0.4 gm. or gr. vi. of opium, would cause symptoms of poisoning in an adult not habituated to the drug, and possibly would cause death. Several instances of death from 0.2 to 0.26 gm. (gr. iij.-iv.) of morphin (or laudanum equivalent) are reported. In other cases death has followed after doses as small as 0.032 (gr. ss.), but in these other cases were also operative or the report is unsatisfactory (see Witthaus and Becker, "Medical Jurisprudence," iv., 730).

On the other hand, numerous cases are on record of persons, not addicted to the opium habit, who have recovered from very large doses. The largest quantity of morphin certainly thus recovered from was 3.89 gm. (gr. lx.) of the acetate (Wood, *Boston Med. and Surg. Journal*, 1876, 82). Although the relative immunity in these cases of very large dose may be ascribed in some degree to an idiosyncrasy of the patients, their escape has been probably more largely due either to non-absorption of the poison or to rejection of the major portion by vomiting.

Infants and children are peculiarly susceptible to the poisonous action of opiates, even in very minute doses. Cases of death from small doses of laudanum are cited as follows: Gtt. iv. (equivalent to gr. $\frac{1}{4}$, 0.011 gm., of opium) in a child of nine months; the same quantity in a child of five weeks; gtt. v. (= gr. $\frac{1}{2}$, 0.013 gm.) per rectum in a child of eighteen months; gtt. viij., during eighteen hours, in four doses (= gr. $\frac{1}{2}$, 0.022 gm.),

in a child of six weeks; gtt. iij. (= gr. $\frac{1}{3}$, 0.008 gm.) in an infant of two weeks; μ iss (= gr. $\frac{1}{16}$, 0.0065 gm.) in an infant of three days; gtt. ij. (= gr. $\frac{1}{8}$, 0.0054 gm.) in an infant of five days; the same quantity in another of four days; μ i. (= gr. $\frac{1}{32}$, 0.005 gm.) in an infant of seven days; and gtt. i. (= gr. $\frac{1}{32}$, 0.0026 gm.) in an infant of six days. A dose of gr. iv. pulv. ipecac. et opii (= gr. $\frac{2}{3}$, 0.026 gm., of opium) has caused the death of a child of four and a half years. Huseman and Taylor refer to a case (Edwards) in which the amount that caused the death of a four-weeks-old child was as low as 0.006 gm. (= gr. $\frac{1}{16}$) of opium, taken in the form of the camphorated tincture.

Even during this early period of life occasional instances of recovery from relatively large doses are met with. A case is reported by Dr. Corbet, in the *Lancet*, August 29th, 1857, p. 220, in which an infant of one day received μ xxx. (= 2.3 grains, 0.15 gm. of opium), yet recovered within ten hours. This case is of interest, as the age of the infant precludes the possibility of its having become accustomed to the drug, as was probably the case with a child of six months who recovered from a dose of a teaspoonful of laudanum (= 4.6 grains, 0.298 gm. of opium) (Simmonds), although treatment was delayed for an hour; and with another of nine months who recovered from a dose of two teaspoonfuls (= 9.2 grains, 0.596 gm.) of the same tincture.

The dosing of infants and young children with officinal or proprietary preparations of opium by mothers and nurses is widely practised. One of the results of the practice is the large percentage of deaths from opiates among young children. A tabulation of 144 cases of opium poisoning, taken chiefly from English and American journals, gives this result:

	Total cases.	Children less than one year.	Children less than five years.	Children less than ten years.
Laudanum	79	24	25	26
Opium	18	9	10	11
Morphin	34	3	5	5
Patented opiates	13	10	12	12
Totals	144	46	52	54
Percentage of total		32	36	37.5

From which it appears that about one-third of the reported poisonings by the opiates occur in children less than one year old.

The poisonous action of the opiates is very greatly diminished by habit, probably more than that of any other poison. The amounts taken by adult opium-eaters, laudanum-drinkers, and morphin-injectors are sometimes enormous. Cases in which the consumption reaches 2 gm. (grs. xxx.) of opium, or one-half, one, or even two ounces of laudanum (= 0.5, 1, 2 gm.), in twenty-four hours are of by no means uncommon occurrence. Such cases sink into insignificance when compared with that of De Quincy, whose daily draught of laudanum at one time reached nearly nine fluidounces (= about 20 gm. of opium). Krüger-Hansen relates the case of a patient who consumed in one year over 300 gm. of opium, a daily average of over 0.8 gm. (about gr. xiiij.). Zeviani cites the case of a woman who, in thirty-three years, had taken over 100 kgm. of opium, equivalent to a daily average allowance of 8 gm. (= nearly gr. cxxv.); and as the dose is gradually increased by opium-eaters, the daily consumption in this case must have been much greater in the later years. Headland and Myers refer to instances in which gr. xvij. (= 1.16 gm.) of morphin were taken daily; and the author met with the case of a young man of twenty, of profligate habits, who had reached the same quantity, when he terminated his career with a large dose of potassium cyanid.

It is not to be inferred from these large amounts that an opium-eater can take an unlimited quantity of the drug without experiencing its poisonous action. It is

simply a question of quantity—a quantity necessarily varying in each case,—and instances are of frequent occurrence in which the opium-habitué has experienced the symptoms of acute poisoning, and has even died from the effects of an overdose.

The tolerance of opiates acquired by habit is not confined to adults; it is also produced in quite young children. A remarkable case in point was published by Dr. J. L. Little (*American Journal of Obstetrics*, 1878, xi.). A male infant, suffering from acute inflammation of the knee-joint, followed by an abscess, began at three weeks of age with small doses of paregoric, gradually increased to a teaspoonful. Subsequently Tinct. opii was substituted, and then Magendie's solution of morphin, in doses gradually increased, until, when nearly eight months of age, the child took in one day two fluidounces of Magendie's solution (equivalent to 2.07 gm., gr. xxxij.) of morphium sulfate.

TREATMENT.—The treatment in cases of acute opium poisoning should be directed, first, to removal of unabsorbed poison from the stomach; and, second, to prevention of death by coma and cessation of respiration, until the processes of elimination have removed that portion of the poison which has been absorbed.

In the earlier stages of the poisoning, emetics are of value—zinc sulfate or ipecacuanha; or, if the patient be an obstinate suicide, apomorphin, hypodermically. On no account should tartar emetic, or any antimonial, be used as an emetic in this or any other form of poisoning. Stomach lavage is to be preferred to the exhibition of an emetic; particularly in the later stages, when, the patient having lost the power of swallowing, a hypodermic injection of apomorphin usually fails to provoke emesis. The siphon is to be preferred to an emetic, not only on account of its more certain and rapid action, but also because its use does not tend to increase the cerebral congestion as does the exhibition of the emetics. On the other hand, in some exceptional cases, in which opium in substance has been taken, an emetic may be necessary to remove masses too large to enter the pipe. In cases likely to lead to litigation, the material removed by the siphon should be preserved. The stomach having been emptied of its contents, the viscus is next to be well washed out, preferably with a solution (1 to 1,000) of potassium permanganate, about 500 c.c. of which are finally left in the stomach. This procedure is to be followed even when the poison has been taken hypodermically, as it is eliminated by the stomach.

If the case be seen before the stage of sopor has been established, it should be prevented, if possible, by keeping the patient in motion—walking him between two sufficiently robust assistants, preferably in the open air, if location and weather permit, but not in the direct sunlight. This "ambulatory treatment" has been beneficially prolonged in some cases for from six to eighteen hours. Under its influence sometimes the action of an emetic which has remained inert is brought about.

If the patient be already in a lethargic condition, he is to be roused without delay. This is best accomplished by cold affusions to the head, the body being kept warm and dry, flagellation to the palms and soles, or to the back with damp towels, or the use of the faradic current. When roused, the patient is to be kept awake as above.

Should the respiration have ceased or become very slow, it may frequently be stimulated by the application of the induced current, the positive pole being applied to the root of the neck over the point where the phrenic nerve crosses the scalenus anticus muscle, while the negative pole is carried laterally over the anterior attachments of the diaphragm. If the faradic current be not obtainable, or if it fail, artificial respiration is to be performed. To be of service this must be persisted in, in some cases, for many hours, and until normal respiration is again established. Dr. W. F. Cheatham has published (*North Carolina Medical Journal*, 1886, 20) a case in which this was the sole method of treatment. The respiration had ceased and the pulse was barely perceptible. Artificial respiration was applied. In thirty-seven minutes

the patient made an effort of respiration; in an hour and forty minutes the respiration was five per minute, though stertorous. In nine hours consciousness returned, and recovery followed.

If the case be at all prolonged, distention of the bladder and possibility of reabsorption are to be prevented by the use of the catheter. If this be done in a case in which there is the faintest possibility of litigation, the urine so removed should be carefully preserved.

Little can be said in favor of the different drugs that have been used as so-called physiological antidotes. Atropin, which is so frequently administered as an antidote to opium poisoning, unquestionably dilates the pupils, but has little, if any, effect upon the respiration. Cases are recorded in which, although atropin has been given until the pupils were widely dilated, the respiration has ceased, and the patient has subsequently recovered by means of artificial respiration (see paper of Dr. Cheatham, quoted above).

Tinctura belladonnae, strong infusion of coffee by the stomach or subcutaneously, extract of coffee, caffeine, brandy, digitalin, chloral hydrate, veratrum viride, and jaborandi have been used as antidotes. The last named, or pilocarpin, may be of value to increase the elimination, and thus lessen the duration of the poisoning.

POST-MORTEM APPEARANCES.—The autopsy reveals no lesions which are characteristic of opium poisoning, except, possibly, the odor of the drug. Obviously, if morphin have been the substance taken, or if other more powerfully odorous substances be present, this will not be observed. The surface of the body is livid. Rigor mortis is said to be of shorter duration than usual, although an autopsy is reported by Tardieu at which rigor mortis was well marked sixty-two hours after death. Putrefaction is said to be more rapid than usual. The blood is fluid and dark. The vessels of the brain and meninges are gorged with blood, and the cut surfaces of the brain substance present numerous dark red spots. The veins of the scalp are also filled with blood. Serous effusions are frequently met with between the membranes, more rarely in the ventricles. The lungs are usually congested. The stomach and other viscera are normal, so far as the action of the poison is concerned. The bladder is generally full of urine.

The congestion of the cerebral vessels and of the lungs are the most noteworthy appearances. Yet, as they may be absent in opium poisoning, and may be present when death has resulted from other causes, they are only of value as confirmatory evidence of the cause of death.

ANALYSIS.—To detect the presence of morphin in the viscera after death, or in articles of food, it is necessary to separate that alkaloid in a condition of as near purity as possible. In cases of opium poisoning it is further necessary to search for meconic acid, and, if possible, for other of the opium alkaloids.

If the facts of the case do not point very distinctly to opium or morphin as the poisonous agent, the process of Dragendorff should be followed for the separation of the alkaloids (Dragendorff, "Ermittl. v. Giften," 4 Aufl., 1895, 149-153).

If the indications of opium or morphin poisoning be sufficiently direct, the following simplified method for the separation of morphin and of meconic acid may be followed. The substances, if solid, are finely hashed and extracted several times with water containing one per cent. of hydrochloric acid at the ordinary temperature (if the materials be alkaline, the proportion of acid is to be increased to such an extent that the liquid, when in contact with it, retains its acid reaction). The aqueous extracts are filtered and shaken with amyl alcohol three or four times, and oftener if necessary, until the amyl alcohol is no longer colored, and the alcoholic layers separated. If the substances under examination be liquid, they are to be rendered acid with hydrochloric acid, filtered, and the filtrate treated with amyl alcohol. The amyl solution now contains meconic acid, if present in the objects examined; and the watery solution, the alkaloids as chlorides. To separate meconic acid, the amyl-

alcohol solution is shaken with successive portions of water, which are separated, until the water is no longer colored. The alcohol is evaporated over the water-bath; the residue extracted with hot water; the solution filtered; the water evaporated over the water-bath; the residue hot; the water evaporated over the water-bath; the residue extracted with alcohol; the solution filtered, and the alcohol evaporated. The tests for meconic acid are finally applied to a portion of the last residue. During this treatment a small portion of the meconic acid is converted into comenic acid, which does not, however, interfere with the tests.

To separate morphin from the aqueous liquid above mentioned, the hydrochloric acid is neutralized completely with ammonia, and the liquid rendered distinctly acid with acetic acid, and evaporated over the water-bath to the consistency of a syrup. The residue is extracted with four or five volumes of ninety-per-cent. alcohol and filtered. The filtrate is freed from alcohol by distillation. The residue, diluted with a small quantity of water, if thick, is heated to 50° to 60° C., an equal volume of amyl alcohol* is added and then sufficient ammonium-hydroxid solution to render the solution distinctly alkaline. The mixture is next strongly shaken at intervals for half an hour, the amyl alcohol separated, and the extraction of the aqueous liquid with amyl alcohol repeated three times. The united amyl solutions are evaporated to dryness; the residue is extracted several times with warm (not hot) water slightly acidulated with sulfuric acid, and the solution filtered. Upon the acid filtrate is floated a mixture of ten parts absolute ether and one part (ninety-five-per-cent.) alcohol; ammonium-hydroxid solution is added to alkaline reaction, and the whole strongly agitated. The ether-alcohol layer is separated; the extraction of the, now alkaline, aqueous liquid is similarly repeated several times, and the ether-alcohol evaporated in a number of small watch glasses. To portions of the residue so obtained, either dry or dissolved in a few drops of water, as the nature of the test may require, and now sufficiently freed from coloring and other foreign substances, the tests for morphin are to be applied.

TESTS.—I. *Morphin*. 1. With the general reagents for the alkaloids, the morphium salts give reactions as follows, the fractions indicating the maximum of dilution in which the alkaloid is capable of reacting: With phosphomolybdic acid, yellowish, amorphous precipitate, $\frac{1}{1000}$; with iodine in potassium-iodid solution, red-brown, amorphous precipitate, $\frac{1}{1000}$; with potassium and bismuth iodid, amorphous precipitate, subsequently changing to silky needles, $\frac{1}{1000}$; with auric chlorid, lemon-yellow precipitate, becoming darker; with phosphotungstic acid, flocculent precipitate, $\frac{1}{1000}$; with potassium iodhydrargyrate, yellowish, amorphous precipitate, $\frac{1}{1000}$; with platinic chlorid, slowly, yellow, crystalline precipitate, $\frac{1}{100}$; with picric acid, bright yellow, amorphous precipitate, $\frac{1}{100}$; and with tannic acid, a faint cloudiness, becoming somewhat thicker on standing. For the above tests the solutions of the alkaloidal residue are to be made with very dilute sulfuric acid, and the reagents should be as nearly neutral as their natures will permit.

2. Morphin dissolves in concentrated nitric acid with an orange-red color, which gradually changes to yellow. Addition of stannous chlorid solution does not change the color of the yellow solution to violet, as it does with the similar color obtained with brucin. Limit, 0.01 mgm.

3. Morphin dissolves in concentrated sulfuric acid, forming a colorless solution. If this solution be heated over the water-bath for an hour, and allowed to cool, or, preferably, if it be allowed to stand in a desiccator twenty-four hours, and then treated with a trace of nitric acid or a minute granule of saltpetre, a beautiful violet color is produced, which soon changes to purple-red, and then gradually fades. Limit, 0.001 mgm. (A. Husemann).

A further portion of the sulfuric-acid solution, if

* It is absolutely essential that the amyl alcohol used should be purified, shortly before use, by repeated redistillation, until a portion, on evaporation, yields no residue capable of reducing iodic acid.

treated, after warming and subsequent cooling as above, with a small fragment of potassium dichromate, assumes a mahogany-brown color (J. Otto).

4. A fragment of solid morphin moistened with a solution of ferric chlorid, as neutral as possible (best obtained by dissolving the chlorid obtained by the dry method in water), assumes a brilliant blue color.

For the success of this test it is essential that the morphin salt be as free from impurities as possible, that little or no free acid be present, and that but a small quantity of the reagent be used. The color gradually changes to green and brown (Robiquet). Limit, 0.1 mgm.

5. A fragment of morphin moistened with Fröhde's reagent (a freshly prepared and colorless solution of 5 mgm. sodium or ammonium molybdate in 1 c.c. sulfuric acid) colors the reagent violet in a short time. The color changes to blue, and then to dirty green, and, finally, to faint reddish. Addition of water discharges the color instantly. Limit, 0.005 mgm.

6. Dissolve a small quantity of iodic acid in a few drops of water, in a small test tube, and agitate with a few drops of chloroform; the latter must remain colorless. Add the solution to be tested, and again agitate. The chloroform, which settles to the bottom, has a violet color, in the presence of morphin, while the aqueous layer is yellowish. Now float upon the surface of the liquid dilute ammonium hydroxid, with as little mixing of the liquids as possible: a brown band is formed at the junction of the ammoniacal and aqueous liquids (Serullas, Duflos, Lefort). Limits: For the violet color of the chloroform, $\frac{1}{1000}$; for the dark band with ammonium hydrate, $\frac{1}{10000}$.

This reaction is also produced by reducing agents other than morphin.

7. Dissolve the solid in warm, concentrated hydrochloric acid containing a little concentrated sulfuric acid, and heat in an air oven at 110° to 120° C. In the presence of morphin a purple color is produced, still visible in the presence of the accompanying carbonized matter. After evaporation of the hydrochloric acid, a further quantity of the dilute acid is added, and the mixture neutralized with sodium bicarbonate in slight excess; a cherry red color is produced, which changes to a dirty-greenish hue as the point of neutrality is reached. On addition of a few drops of a dilute alcoholic solution of iodine, the color changes to green, and the pigmentary substance now dissolves in ether with a purple color (Pellagri).

The reaction is due to the formation of apomorphin, and is consequently also observed with codein.

Many other tests for morphin are in use; the above are, however, sufficient. No one of them is in itself characteristic.

II. *Narcotin*. The reactions of the alkaloids of opium other than morphin are at present of but little toxicological interest, as they are substances which are not commonly met with, and hence are unlikely to cause poisoning. For the purpose, however, of distinguishing between morphin and opium poisoning by analysis (a distinction which may be of medico-legal importance), the reactions of narcotin and of meconic acid (see below) are taken advantage of. Narcotin is chosen from among the other opium alkaloids for this purpose, partly because it is more abundant in opium, and partly because of the sharpness of its reaction with sulfuric acid.

If the Dragendorff method have been followed, narcotin should be searched for in the residue of evaporation of benzene from the alkaline solution.

1. Of the general reagents for the alkaloids, phosphomolybdic acid, potassium iodhydrargyrate, iodine in potassium iodid, and picric acid give precipitates in solutions of $\frac{1}{1000}$ to $\frac{1}{10000}$.

2. Moistened with concentrated sulfuric acid at the ordinary temperature, narcotin produces an intensely yellow solution, which, on gradual heating, changes to orange, then, beginning at the borders, blue-violet, and, when the heat has been raised to the point of volatilizing of the acid, dark red. The colors are presented

more slowly, but more purely, by dissolving the residue in dilute sulfuric acid and evaporating quite slowly (Couerbe, Husemann). Limits: $\frac{1}{1000}$, very evident; $\frac{1}{10000}$, faint carmine only.

3. Dissolve in concentrated sulfuric acid, let stand an hour, and add a trace of nitric acid; a red color, which for some time increases in intensity.

III. *Meconic Acid*. 1. Crystallizes in white, glistening prisms, either single and large, or small and arranged in bundles, which at 100° C. lose their water of crystallization and become opaque. If heat have been applied to the solution in the presence of acids, the shorter, prismatic crystals of comenic acid will be also observed.

2. Meconic acid, or a meconate in solution, gives white or yellowish precipitates with lead acetate, silver nitrate, mercurous nitrate, and mercuric nitrate.

3. The characteristic reaction of meconic acid is the formation of an intense red color when the acid or one of its salts is moistened with a solution of ferric chlorid (Sertürner). The color does not disappear either on warming or on the addition of hydrochloric acid, or of auric chlorid, or of mercuric chlorid.

Comenic acid gives the same reaction. It can only be present as a product of decomposition of meconic acid. Acetic and thiocyanic acids and their salts also give a red color with ferric chlorid. The former may be present as a normal food constituent, and the latter is present in the saliva in quantity sufficient to give the reaction without any preliminary purification. The red color, however, produced by acetic acid is discharged by heat or by the addition of hydrochloric acid, and that due to the thiocyanate disappears instantly on addition of auric chlorid or of mercuric chlorid solution.

FAILURE OF DETECTION.—As morphin is oxidized to oxydimorphin in the body, more or less completely according to the magnitude of the dose, it is usually eliminated in cases of poisoning as a mixture of oxydimorphin and morphin, both of which respond to the reactions given above. This elimination is principally by the alimentary canal and only in traces by the urine, whatever may have been the channel of introduction. Therefore the stomach and intestinal contents, or the product of stomach lavage, are the situations in which the poison will most probably be detected, and we may expect to find it in the urine only when very large doses have been taken. It has also been detected in the liver and kidneys in several instances, but very rarely in the brain. The detection of morphin is by no means certain, and carefully conducted analysis may fail to show its presence in the cadaver after undoubted poisoning by it, even when the stomach has not been washed out and vomiting has not occurred.

Although morphin is more subject to decomposition than strychnin, it still withstands the influence of putrefaction quite well. In a case cited by Woodman and Tidy it was detected four months after death; and Stas gives an account of a case in which he detected morphin in all the organs of a body after thirteen months of burial.

In cases of long burial, caution is required that ptomaines be not mistaken for morphin, as occurred in an Italian case, in which Selmi showed that what a careless analyst had taken for morphin was in reality a ptomaine (Selmi, "Sulle Ptomaine," 1878). Such a mistake is impossible, if the tests described above are carefully applied (see Witthaus and Becker, "Med. Jur.," iv., 760-769).

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OPTIC NERVE. See Eye.

OPTOMETRY—from $\delta\pi\tau$, root of $\delta\psi\sigma\mu\alpha\iota$, fut. of $\delta\psi\alpha\sigma$, to see, and $\mu\epsilon\tau\rho\omega\iota$, measure—signified, in its older use, the measurement of the range of vision (*die Gesichtswerte*). With the attainment of broader and more accurate knowledge of the physiology and pathology of vision, quantitative methods have been applied to the investigation of other visual functions, and we now recognize, as parts of one general subject, the measurement