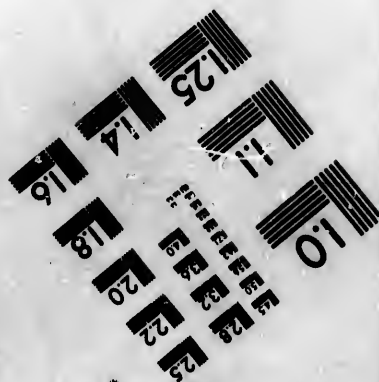
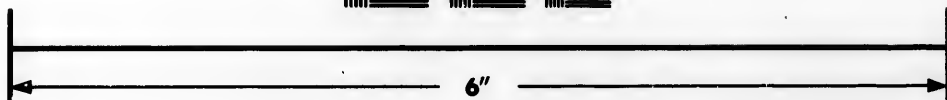


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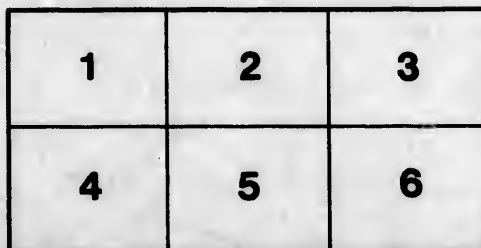
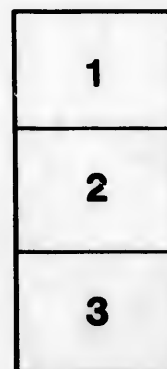
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# MUSHROOM POISONING

BY  
BEAUMONT SMALL.  
OTTAWA.

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# MUSHROOM POISONING.

BY  
BEAUMONT SMALL,  
OTTAWA.

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## MUSHROOM POISONING.

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THE subject of mushroom poisoning is of much importance to the practising physician, who may at any moment be called upon to care for such a case. In Europe, where fungi are articles of every-day diet, accidents are constantly occurring from the mistaken use of poisonous species. In America this form of food is not utilized to the same extent, and cases of poisoning are not so frequent; but very severe illnesses and deaths do occur from this cause, and an autumn rarely passes without some such case being referred to in our medical publications. In some instances it is due to the gathering of poisonous forms with the common mushroom. At other times it arises from the desire to gather species that are not so well known; and occasionally we find that the attractive appearance of the poisonous forms has tempted a child to gather and eat. In works upon this subject very many cases are reported, and the large percentage of deaths is noticeable. Ziemssen quotes the cases of a mother and daughter, both fatal; of a mother and child, both fatal; of five officers, all of whom died; of a family of seven, with three deaths. Blyth refers to the statistics of fifty-three cases of poisoning by *Amanita phalloides*, of which forty ended in death.

The earlier reports of cases are generally very imperfect and brief, and many of the more recent ones have the same deficiency. Others, however, are very carefully and minutely reported and are valuable contributions to the subject.

### Illustrative Cases.

The following are a few illustrative cases:

Two adult males ate, at 8:30 A.M., at breakfast, a quantity of mushrooms supposed to be *Amanita caesarea*, but in reality *A. muscaria*.

The first man ate about two dozen. At 8:45 he complained of feeling unwell, and at nine was found on his bed in a state of collapse. He complained of a sense of impending death, and there rapidly supervened blindness, trismus, difficulty in swallowing, unconsciousness, and convulsions. Death took place in the evening of the second day. The treatment consisted in the administration of emetics, and apomorphine and atropine hypodermically.

The second man ate about one dozen. After breakfast he rode to his office. At 9:30 he began to complain of diplopia and drowsiness. At ten o'clock he was found sitting in his chair, half stupid, with retraction of the head. He rapidly became unconscious and remained so for five hours, excepting upon two occasions, when he regained consciousness for a brief period. Cold sweats were a prominent symptom. There was no rise in temperature, no pain, nor any nausea. The treatment consisted in the hypodermic administration of apomorphine, which was of no effect. Strychnine and atropine were then given; of the latter, gr.  $\frac{1}{16}$  every two hours, about gr.  $\frac{1}{16}$  being given in the twenty-four hours. Castor oil and sweet oil were also given. The man recovered.<sup>1</sup>

A boy, aged 12 years, ate, at 11:30 A.M., about one-third of the pileus of an *Amanita phalloides*. No ill effects were felt until 1 A.M., when he awoke complaining of thirst, which was followed by vomiting and purging. These symptoms continued all night, and less severely during the day. Castor oil and citrate of magnesia were given. The following morning he awakened with severe abdominal pain, and when seen by the physician he was feverish and suffering from palpitation, purging, vomiting, and extreme exhaustion. A mixture of chloroform, morphine, and cardamoms was given; also whiskey. The symptoms eased during the day, but the prostration continued. He slept the following night, but about 7 A.M. was seized with convulsions and died in half an hour, on the fourth day after eating the fungus. The post-mortem examination was made thirty-six hours after death. Rigidity was well marked, the face was cyanotic, and dark-brown fluid was issuing from the mouth and nostrils. Upon opening the abdomen, marks of recent peritonitis were seen as bands of lymph gluing the small intestines together. The stomach contained a small amount of dark fluid, the mucous membrane was inflamed, and the walls were softened. The whole of the small intestine was inflamed and there were numerous spots of gangrene. The liver was anæmic, the heart was empty, and the lungs were healthy.<sup>2</sup>

Mrs. N—, aged 55 years, ate a third of a raw mushroom, probably *A. phalloides*; about 10 A.M. No symptoms appeared until 7 P.M., after supper, when abdominal cramps commenced and steadily increased in severity. Vomiting began at 8 P.M., with marked prostration. The patient was found in a condition of collapse, with a temperature of 100.5° F.; pulse small, rapid, weak; respirations shallow; slight tympanites and abdominal tenderness; the pupils were contracted; there were cold perspirations, pallor, and an anxious, drawn expression, with mild delirium, intense retching, and profuse watery stools. Treatment: morphine sulphate gr.  $\frac{1}{4}$ , atropine nitrate gr.  $\frac{1}{16}$ , strychnine nitrate gr.  $\frac{1}{16}$ ; and hourly, fluid extract of belladonna gtt. i., fluid extract of nux vomica gtt. ss., bismuth subnitrate gr. ij.; after every emesis, sodium bicarbonate 3 ss. in half a glass of water; whiskey ad libitum, and hot applications. From 12 P.M. the symptoms gradually subsided. The treatment was continued for two days.<sup>3</sup>

Five persons, aged 13, 22, 29, 40, and 45 years, ate of what was

probably *Amanita verna*, prepared as a stew, at nine o'clock in the evening, five hours after the fungi had been gathered. No discomfort was experienced until towards morning, when all were attacked with pain in the stomach, nausea, and a sickening sensation at the epigastrium. Twelve hours after the mushrooms had been eaten all the symptoms had increased in severity, and in addition there were retching and vomiting. In thirty hours there was intense gastro-enteric irritation with relaxed bowels and distressing tenesmus. As these symptoms subsided they were followed by prostration, coldness of the surface, and a tendency to coma. In two of the patients who had eaten more than the others the symptoms advanced to coma and coma vigil, with the features shrunk, skin dusky, and pulse scarcely perceptible. One, aged thirteen years, died in fifty-six hours; another, aged twenty-two years, in sixty-three hours. The treatment consisted in the administration of bismuth and creosote, morphine, stimulants, heat to the surface, and atropine. The atropine was not begun until twenty-four hours had elapsed; it was given in repeated small doses until each patient had had respectively  $\frac{3}{16}$ ,  $\frac{1}{8}$ ,  $\frac{1}{16}$ ,  $\frac{1}{32}$ , and  $\frac{1}{64}$  of a grain. Temporary improvement was noticed after each dose of the antidote.

Father, mother, and child, aged 5 years, ate twelve, eight, and five mushrooms respectively, supposed to be *Amanita muscaria*, *Amanita phalloides*, and *Amanita verna*. They were gathered during the morning and eaten at dinner. Nausea and headache were complained of during the afternoon. At six o'clock more were eaten. The same symptoms were noted, but in greater intensity. At midnight purging and vomiting became severe, but there was not much pain, excepting cramps in the legs. The vomiting at first consisted of food; then it became choleric, as also did the stools, and finally of a pink or purplish color. In twenty-four hours the patients became jaundiced and had a peculiar glazed appearance about the eyes. Muscular twitchings took place, especially in the face, arms, and hands. During the next twelve hours the depression became severe, the heart was irregular, the respirations were jerky, and the pupils were dilated. The urine became saffron-colored, and before death it was purplish. The perspiration and breath smelled of mushrooms. In forty-eight hours the mother and child died within half an hour of one another. The child had convulsions for about four hours, up to two hours before death. The mother was restless and unconscious about five hours before death. The father recovered in about twelve hours after the symptoms had commenced. Treatment consisted of morphine gr.  $\frac{1}{4}$ , atropine sulphate gr.  $\frac{1}{16}$ , repeated in two hours; whiskey, ammonia, and nitroglycerin.

A man, aged 52 years, ate of an omelet prepared from mushrooms, among which were *Amanita phalloides* and *Boletus luridus*. Four hours after eating he was found covered with a cold, clammy perspiration; the breathing was stertorous and of the Cheyne-Stokes type; the pulse was almost imperceptible—28 per minute. The pupils were dilated. Atropine was given; also ether, coffee, and rum; and heat was applied to the surface. The symptoms continued to grow worse until one litre of decinormal saline solution was injected.

what was

Improvement followed immediately; this treatment was continued, and in one hour the pulse was 60 per minute, the respiration became stronger, and the patient rallied from the state of collapse. No diarrhoea or vomiting was present at any time."

### The Poisonous Principles in Mushrooms.

The poisonous properties of fungi and the active principles that produce the poisonous effects have received much attention from many careful observers. For many years a principle common to all species was sought for, and various chemists described several poisons under as many titles. Thus we have had *muscarine*, *bulbosine*, *amanitin*, *agaricin*, *agarythine*, and many others. At present all are resolved into two poisons, *muscarine* and *phallin*, the former characteristic of *Amanita muscaria*, being an alkaloid; the latter characteristic of *Amanita phalloides*, and an entirely different poison.

In addition to the action of poisonous species, very many instances of poisonous effects have followed the use of species which are known to be free from any poisonous properties. Most of these were undoubtedly due to gastrointestinal irritation, more or less severe, in some cases leading to peritonitis. All, however, cannot be traced to this condition, and in some cases the poisoning has been popularly explained by the fact that the edible form was gathered from a manure heap or from some other decomposing organic matter, which produced a poisonous action. Another explanation is that the mushroom may have been kept too long, and that decomposition and putrefactive changes had begun which rendered it poisonous in character. In the light of modern bacterial science, this idea of a toxic principle being generated by the decomposing mushroom is of much importance. We know that many cadaveric poisons are produced in this way, and we also know that the mushroom has been termed a "vegetable beefsteak" on account of the very large percentage of nitrogenous matter in its composition. The flesh of the mushroom, in addition to a large percentage of water, about eighty per cent., is made up of a proteid or nitrogenous substance called *fungin*, which contains from 3.2 to 7.2 per cent. of nitrogen. It is to this fungin that the mushroom owes its highly nutritive properties.

*Muscarine*,  $C_8H_{15}NO_3$ , the most carefully studied and best known of the poisonous principles, is an alkaloid, first described by Schmiedeberg and Koppe,<sup>7</sup> whose investigations form the foundation for our present knowledge of this subject. Their work has been confirmed by others (I. L. Prevost<sup>8</sup>) and reaffirmed by Schmiedeberg.<sup>9</sup>

It is a colorless, syrupy fluid, tasteless and without odor. It is very soluble in water and alcohol, but insoluble in ether; very slightly

soluble in chloroform. It is alkaline in reaction and combines with acids to form salts. The sulphate and nitrate are prepared for commerce. They are very hygroscopic and soluble in water and alcohol.

The quantity of muscarine present in the fungus has not been determined. It has been estimated variously from one-fifth to one per cent. of the dried fungus. The percentage varies greatly according to the season of the year, the locality in which it grows, and many such conditions.

Muscarine was formerly considered to be characteristic of the fly agaric and similar fungi, but in the progress of chemical and bacterial science it has been obtained from other and very different sources. It has been separated as a ptomain from decomposing fish and also from horseflesh undergoing the same change (Vaughan). It is also prepared synthetically from cholin by the oxidizing action of nitric acid. Cholin, neurin, and other ptomains to which it is allied are all of cadaveric origin, and nearly all are powerful poisons. The physiological action of muscarine bears a resemblance to that of pilocarpine, and also of calabar bean. It is primarily an excitant to the nerve centres, the period of excitement rapidly passing into one of depression. Upon the brain it is an intoxicant, causing dizziness, vertigo, confusion of ideas, delirium, disturbed vision, ataxia, and other like symptoms, which may end in convulsions, coma, paralysis, and death. Its depressing action is specially directed to the cardiac and respiratory centres, and its fatal effects are due to paralysis of these organs. The heart remains in a dilated state after death. The vasomotor centres are also depressed, causing a lowering of the blood pressure. It increases the secretion of the sudoriparous, lacrymal, salivary, and all other glands, probably with the exception of the kidney. The muscular system is also irritated and weakened, giving rise to muscular fatigue and cramps of the extremities and of the intestines. The pupils are contracted.

The action of atropine upon the heart is directly antagonistic to muscarine, and furnishes us with the physiological and most valuable antidote. Atropine paralyzes the inhibitory nerves of the heart and increases the rapidity of its action. The effect of muscarine is to produce a slowing and weakening of its action. The opposing effect is frequently demonstrated upon the exposed heart, which, when failing from the presence of muscarine, is at once aroused into action by a drop of atropine solution, or, if the atropine has been first applied, the toxic effect of muscarine is prevented.

No cases of poisoning by pure muscarine have been reported. All our knowledge is derived from experimental work upon animals and man. It has been found that 8 to 12 mgm. (gr.  $\frac{1}{8}$  to  $\frac{1}{4}$ ) will

cause the death of a cat in about eighteen minutes, and 3 to 4 mgm. (gr.  $\frac{1}{20}$  to  $\frac{1}{15}$ ) will have the same result in a few hours. Five milligrams (gr.  $\frac{1}{12}$ ) taken by an adult man causes in a few minutes profuse salivation and lacrymation, increased frequency of pulse, nausea, giddiness, confusion, determination of blood to the head, and perspiration. There is no vomiting or diarrhoea. Fatal doses in cats cause salivation, contraction of pupils, vomiting and purging, rapid breathing, and dyspnoea; as death approaches the respirations grow slower, the pupils become dilated, and convulsions usher in death.

The poisonous principle of *Amanita phalloides* has been frequently investigated, and the poison obtained has been variously named. *Bulbosin*, *amanitin*, *phalloidin*, and several others have been described, but no satisfactory result was reached until Kobert<sup>11</sup> published the results of his researches and named the poison *phallin*. Phallin is an entirely different poison from muscarine. It is a toxalbumin, a member of that group to which belongs the specific poison of cheese, meat, rattlesnake poison, as well as the toxic agent of diphtheria and other bacterial poisons.

The action of phallin is directed to the blood corpuscles, causing their destruction and setting free the hæmoglobin. It produces such changes in the plasma that the serum escapes from the vessels into the various tissues and cavities, and a condition resembling that produced by cholera is the result. Its action is slow, and the symptoms of poisoning do not appear for an interval of from three or four to twelve or fourteen hours. They begin as severe abdominal pain, prostration, vomiting, free watery evacuations, and symptoms of collapse. Irritation to the nervous system is shown by muscular cramps, tetanic in character, and convulsions. Consciousness usually remains unaffected. The pulse becomes weak and flabby. The kidneys secrete much less urine, and there are signs of albuminuria and hæmoglobinuria.

The presence of this second poison explains the symptoms of many cases of poisoning that were obscure when muscarine was considered the sole poisonous principle of mushrooms. At present *A. muscaria* and *A. pantheroides* have been studied as the source of muscarine only, and *A. phalloides* alone has furnished phallin, but it is not probable that either poison is limited to these particular species. There is much more reason to believe that each poison is widely distributed. Muscarine is certainly the chief poison of *A. muscaria*, but many of the symptoms that follow poisoning by this fungus can be explained only by the presence of phallin.

### Pathological Anatomy.

The post-mortem appearances are not very definite. By some it is stated that rigor mortis is absent. Others have found it to exist, but to disappear early. There are evidences of gastrointestinal irritation, and portions of the fungus may be present. The various organs are congested, especially the kidneys. The abdominal and pleural cavity may contain fluid colored by the transuded hæmoglobin. The heart is dilated. In a series of autopsies, when death probably resulted from phallin poisoning, there were numerous small ecchymoses on the pleural surfaces, and also in the lungs, heart, kidneys, liver, and other organs. The blood, also, was found to be of a dark cherry-red color and fluid. The veins were full. Fatty degeneration of the liver has been found in some cases.

### Symptoms.

The symptoms that follow the use of poisonous fungi coincide with the results of the experimental work with these poisons. In the majority of cases the symptoms are distinctly those of one or the other poison, sometimes complicated by the irritation of undigested portions of the fungus. In poisoning by muscarine the diagnostic points are the early onset of symptoms, the signs of intoxication, and the functional weakness of the heart and lungs. In poisoning by phallin the symptoms are delayed; there is an absence of cerebral disturbance, and severe gastroenteric irritation, becoming choleraic in character, is the prominent symptom.

In muscarine poisoning the alkaloid is very stable and is excreted with the urine, which retains the intoxicating properties. This, it is reported, the inhabitants of certain districts of Siberia take advantage of in order to prolong their intoxication. The fly agaric is a common fungus in northern Asia, and furnishes the natives with a substitute for the alcohol, opium, and the narcotics of other countries.

The duration and termination of cases of mushroom poisoning vary greatly according to the quantity of poisonous material taken into the system. They are also influenced by the health and strength of the patient. Cases due to muscarine run a more rapid course than those due to phallin. The symptoms begin almost at once, and death may take place in five or six hours; more frequently a fatal termination does not take place until the second or third day. Convalescence is always slow, more particularly in severe cases. When phallin is the poison, the course is slower. Death may take place on the



second day, but four or five days is the more common period. In some instances the patient has lingered seven days. Of five officers poisoned by eating *A. bulbosa*, symptoms did not begin until eleven hours had elapsed; two died on the second day, two on the third, and one on the fifth day. In another case a child died on the second day, the mother on the fifth day. In still another instance, one victim died after forty-eight hours, one at the end of sixty hours, and the third on the seventh day.

### Treatment.

The treatment consists of measures to allay the gastrointestinal irritation and overcome the depression, and also the employment of special antidotes to counteract the poison. The stomach should be emptied as rapidly as possible for the purpose of removing all portions of the fungi that may remain undigested. Castor oil and enemata should be used with the same object in view; milk, barley water, and other demulcent drinks, bismuth, magnesia, and antacids are to be given for the double purpose of soothing the irritated mucous membrane and of retarding the absorption of the poison. Muscarine becomes very soluble in the acid fluids, and for this reason acids should not be given. Alcohol and ammonia may be required to stimulate the heart and respiration and lessen the general depression.

As special antidotes, tannin, charcoal, and permanganate of potassium have been recommended. The first two are of value in rendering insoluble the poisons, and the latter for the purpose of decomposing the alkaloid; their value, however, is as yet uncertain.

When the poisoning is due to muscarine, the only antidote to depend upon is atropine, which should be immediately administered in all cases of suspected poisoning from this source. Experiments have proved conclusively that when the inhibitory nerves of the heart are depressed by atropine the effects of muscarine are almost entirely counteracted, and when the heart and respiration are failing from the poison, the atropine helps to restore their tone and force. In the cases in which it has been employed, even when death has finally taken place, its favorable effects have been specially mentioned. It should be administered hypodermically, in gr.  $\frac{1}{100}$  to  $\frac{1}{50}$  doses, repeated hourly, according to the symptoms and its effect.

When *A. phalloides* has been taken and phallin is the poisonous principle, there is no drug that can be employed with the same feeling of hopefulness, as there is no known antidote to this poison. As soon as the poisoning is suspected the same general treatment is indicated for the purpose of removing any portion of the fungus



and preventing further absorption. Stimulants should be freely administered. Nitrite of amyl and nitroglycerin may prove of service to maintain the cutaneous circulation. When the severe symptoms supervene and collapse is threatening, intravenous injections of decinormal saline solution or its subcutaneous use will prove of great service, and upon this procedure will depend the greatest hope of success. Transfusion of blood has also been recommended.

### Classification of Mushrooms.

The higher forms of fungi which grow so profusely throughout the whole world are classified into three groups, according to their mode of producing spores. These are—

*Hymenomycetæ*, in which the spores are external to the lining membrane, which membrane is on the under surface of the pileus or cap and folded as gills. This comprises all such as are commonly called mushrooms or toadstools.

*Discomycetæ*, in which the spores are also formed external to the membrane, but in depressions, or lacunæ, on the upper and outer surface, as in the ordinary morel.

*Gastromycetæ*, in which the spores are contained in a cavity formed by the membrane, as in the well-known puff-balls.

Of the many thousand species that are included in these three classes, only a few are known to contain an active poison, and nearly all these are closely allied and form a single genus of the hymenomycetæ. The most important of the many genera of hymenomycetæ are *Agaricus* and *Amanita*. The former contains the ordinary edible mushrooms, the latter the poisonous forms.

These genera are variously arranged by different botanists, and this want of uniformity has led to much confusion of names. Formerly all were included in the single genus *Agaricus*, and we will find the poisonous forms referred to as *Agaricus muscaria*, etc. It is now more general to regard the *Amanita* as a sub-genus, or as a separate genus, with its distinctive name.

### DISTINCTION BETWEEN EDIBLE AND POISONOUS MUSHROOMS.

The structural and botanical differences between the amanitas and agarics are very slight, and their resemblance is the cause of the many accidents that occur. Between the common mushroom and the fly agaric the difference is very marked, and one should never be mistaken for the other, but there are many other edible mushrooms that bear a close resemblance. The danger is also increased by the fact

that certain species of the amanitæ, as *A. cesarea*, are edible; and unless the collector is a skilled mycologist a mistake may easily occur. Many suggestions have been offered to facilitate the recognition of the poisonous and edible species, but none is sufficiently trustworthy to be depended upon without some knowledge of the distinctive characters of each. The most important sign is the presence of the *volva* which is formed on the poisonous species, but is absent from the others. When young, the growing fungus is enveloped in a membrane, which is ruptured as the plant expands, its traces remaining as a cup-shaped ring, or sheath, around the base of the stem, and as excrescences on the upper surface of the cap. This volva is often below the surface of the soil and is easily overlooked. In gathering the fungus, the stem is generally broken off, and the characteristic base with the volva is left behind. The color of the gills is also suggestive, those of the edible forms being pinkish, while those of the poisonous species, with few exceptions, are white. A disagreeable, noxious odor and sharp, acrid taste also indicate a dangerous species.

The points of difference between the common edible mushroom and the two poisonous forms are very concisely described by Dr. W. G. Farlow of Harvard University, as follows:

"(1) The common mushroom has a pileus which is not covered with wart-like scales; gills which are brownish-purple when mature; a nearly cylindrical stalk, which is not hollow with a ring near the middle, and without a bulbous base sheathed by a membrane or by scales.

"(2) The fly agaric has a pileus marked with prominent warts; gills always white; a stalk, with a large ring around the upper part, and hollow or cottony inside, but solid at the base, where it is bulbous and scaly.

"(3) The death cup has a pileus without distinct warts, gills which are always white, and a hollow stalk, with a large ring, and a prominent bulb at the base, whose upper margin is membranous or bag-like.

"(4) Other points of difference are the different places in which these species grow, and also the colors, which, although they vary in each case, are brilliant yellow or red in the fly agaric, white varying to pale olive in the deadly agaric, and white usually tinged with a little brown in the mushroom.

"(5) In the mushroom the pileus averages from three to four inches in breadth, and the stalk is generally shorter than the breadth of the pileus and comparatively stout. The pileus remains convex for a long time, and does not become quite flat-topped until old.

The substance is firm and solid. In the fly agaric the pileus, at first oval and convex, soon becomes flat and attains a breadth of six to eight inches and sometimes more. The stalk has a length equal to or slightly exceeding the breadth of the pileus, and is comparatively slenderer than in the common mushroom. The pileus of the deadly agaric is thinner than that of the mushroom, and, from being rather bell-shaped when young, becomes gradually flat-topped with the centre a little raised. In breadth it is intermediate. The stalk usually is longer than the breadth of the pileus, and the habit is slenderer than the other two species."

The special characteristics of the amanitas are as follows: "*Pileus* (or cap) at first campanulate, then plane; fleshy towards the centre, attenuated at the margin; gills ventricose, narrow behind, free, numerous, at length denticulate, the imperfect ones few, of a determinate form according to the kind, and, with one exception (*A. caesareus*), white. *Stalk* generally enlarged at the base, frequently bulbous, solid, or stuffed with a cotton-like substance, which is at length absorbed; ring descending, imperfect, fugaceous; flesh white, unchanging" (Badham).

## POISONOUS MUSHROOMS.

The following species, growing in this country, comprise all that are known constantly to possess active toxic properties. There are others that are probably poisonous, but as yet they have not been properly authenticated, nor have any deaths been attributed to their use. Many of them often produce distressing symptoms, but they are uncertain and may often be eaten with perfect safety.

**AMANITA MUSCARIA** Grev. *Agaricus muscaria* L. Fly agaric. This is a large showy fungus, very common in some localities, growing in oak and pine woods from June to late in the autumn. The pileus is of an orange or yellow color, sometimes becoming brilliant red, in others it varies almost to a white; it is covered with warty excrescences, generally whitish. The under or spore-producing surface is white. The plant is very free from insects and flies and does not blacken when broken. In early growth the pileus is convex, but becomes flat and sometimes concave. The stem is bulbous, white, and springs from a volva. It is rough and covered with warty growths on shaving-like shreds. It should be readily distinguished from the edible species by its color and appearance, the color of its gills, and its place of growth, as it is never found in meadows, nor does the mushroom grow in woods. The fly agaric is of sturdy growth, ranging from four to sixteen inches in height. Mistakes are more

liable to arise in mistaking the fly agaric for some of the edible amanita, as *Amanita cesarea* and *Amanita rubescens*.

**AMANITA PHALLOIDES** (Pers.) Fr. Death cup. This species is not so large nor so brilliant as the *A. muscaria*. It grows from three to six or eight inches high, and is found in woods, but often extends into meadows and fields. It may be gathered in summer and is very common in the autumn months. The pileus is white or fawn-colored, sometimes becoming yellow or greenish and smooth, and the investing membrane, separating from the cap, remains as a deep cup-like volva, which has given it the name of death cup. The stem is white, or tinged in the darker forms, and smooth, excepting when the investing cap is closely adherent, when it has a bulbous appearance. It also is very free from insects and remains of a clear color. Its odor and taste, when fresh, are not noticeable, but after it has been gathered they become disagreeable. Its gills and spores are white.

This species is likely to be mistaken for the common edible mushroom, but it bears a much greater resemblance to *Lepiota naucina*, another fungus having white gills and spores, but quite free from any poisonous properties. Another source of danger is that in some immature specimens of *A. phalloides* the gills are of a faintly pink color.

**AMANITA VERA** Bull. The vernal or bulbous amanita is considered by many to be simply a variety of *A. phalloides*, and is one of the most important of the poisonous species on account of its resemblance to the common edible mushroom. It is smaller than *A. phalloides*, more delicately formed, and of a pure white color. It is found in the early summer months and is very common in many localities. In Europe it appears in the spring, from which it derives its specific name. Its distinctive character is the manner in which the sheath adheres to the stem, giving the stem a very bulbous appearance, and depriving it of its cup-shaped volva.

**AMANITA PANTHERINUS** Deb. is a common species with many varieties. It is of a yellowish-brown color, over which there are many markings of a darker hue, which give it a mottled appearance. The gills and flesh are white. The following instance of poisoning is quoted by Christison: "A boy, having eaten some of the fungus, became delirious and maniacal and gradually passed into a condition of trance. Recovery took place."

**BOLETUS LURIDUS** Schœff. This fungus is of a different class from the agarics and amanitas, as its spores are formed in lacunæ on the under surface of the pileus. In its shape it resembles the agarics. The upper surface varies from an amber to a brown color; the under surface and stem are of a bright red or ferruginous brown. The flesh is yellowish; when broken and exposed, it changes to a blue. The

pileus is from two to six inches broad. The stem is solid, bulbous, sometimes quite smooth, and more or less mottled. It grows in woods and thickets.

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