Remarks

ON

POISONING BY FUNGI:

AMANITA PHALLOIDES.

By CHARLES B. PLOWRIGHT, M.D., F.R.C.S.ENG.,

Consulting Surgeon, West Norfolk and Lynn Hospital; formerly Professor of Comparative Anatomy and Physiology, Royal College of Surgeons of England.

A CERTAIN number of cases of poisoning by fungi occur every autumn in this country, although they are not so numerous with us as abroad. It is not long since that the tendency was to regard every fungus other than the common mushroom as poisonous. So far from this being the case, the majority of them are eatable, in the sense that they can be eaten without making the eater ill, when cooked in an appropriate manner. The really poisonous species can almost be counted on the fingers. But we may go even further than this, and say the vast majority of deaths in this country is caused by the consumption of one species, and one species alone. These deaths are caused by mistaking this particular species for the common mushroom which it to a certain extent resembles. This fungus also is the commonest cause of fatal poisoning on the Continent of Europe, where fungi are eaten as food and where all sorts and kinds are pressed into the service of keeping soul and body together, not as we eat mushrooms as luxuries. It so happens that our textbooks lead us to suppose that the poisonous qualities of all fungi are due to the alkaloid muscarin, the antidote for which is atropin. That certain English fungi do contain muscarin is perfectly true, but these are such as can hardly be mistaken for the common mushroom by the most careless. The species which causes so many deaths both here and abroad owes its lethal power not to an alkaloid but to a toxalbumin—phallin. This fungus, *Amanita phalloides*, presents certain very definite botanical characters by which it and its allies can easily be recognized.

During the last thirty years I have been able to trace it as the cause of the following four deaths in and near the town of King's Lynn.

CASE I.

R. H., of King's Lynn, a boy aged 12, ate at 11.30 a.m., on Saturday, September 27th, 1879, about a third of the pileus (top) of a specimen of *A. phalloides*, uncooked. He had no symptoms until 1 a.m. the following morning, September 28th (thirteen hours), when he had vomiting, diarrhoea, and great thirst. He was, however, able to eat his breakfast at 8 a.m. Diarrhoea and vomiting occurred again at noon (twenty-four hours). On the following day (September 29th) his symptoms were ameliorated; at 7 p.m. the late Dr. C. Glasier saw him. The above-mentioned symptoms continued and were accompanied by severe abdominal pain. On September 31st, at 7 a.m., he died, slightly convulsed—that is, on the fifth day (81½ hours).

Post-mortem examination showed peritonitis with the lymph; gastric mucous membrane vivid red; walls of the stomach softened, so as to be readily perforated by the finger; gangrenous spots on the mucous membrane of the intestine.¹

CASE II.

J. C. B., a little boy of 5, residing near King's Lynn, ate an unknown quantity of *A. phalloides* between 3 and 4 in the afternoon of August 29th, 1894. He had no symptoms till the middle of the night, ten to twelve hours after eating the fungus, when he had sickness and diarrhoea. Dr. P. de C. Potter saw him about 11 a.m. on August 30th; he had previously had an emetic of salt and water, which acted freely; afterwards a dose of castor oil was given him and milk. The most prominent symptoms besides diarrhoea and vomiting were severe abdominal pain and intense thirst, which continued until 8 p.m. on August 31st, when he became worse and died unconscious on September 1st at 7.30 a.m., sixty-eight hours (fourth day) after eating the fungus.

CASES III AND IV.

J. H. R., of King's Lynn, aged 32, on August 8th, 1905, gathered some *A. phalloides*, which he and his family, consisting of the wife, aged 22, a daughter Dora, aged 7, and a son Hughie, aged 1 year and 10 months, ate for their tea. During the preparation of the meal, the mother and the boy (Hughie) ate a certain quantity of the fungi raw. The total amount consumed by the whole family did not exceed ½ lb. About 6 a.m. Hughie awoke sick, his mother got up to attend to him and was sick too; Dora followed, and the father was taken ill last (eleven hours). The symptoms were the same in all—diarrhoea, vomiting, great thirst, perspiration, and abdominal pain. The latter was intense, of a crampy character, especially worse in the epigastrium, but extending over the whole abdomen. Dr. C. K. Bond, to whose kindness I am indebted for seeing the cases, at about 11 a.m. on August 9th found the whole family very ill with the above symptoms. The little boy Hughie became worse, and on August 10th was convulsed; he had slight distortion of the muscles of the face, slight nystagmus and widely-dilated pupils. He died at 8 p.m. on the third day (fifty-four hours). A *post-mortem* examination was made twenty hours after death. The body was well nourished, hypostatic discoloration was very marked, and there was a distinct icteric tinge. *Post-mortem* rigidity was absent. The stomach was empty. In places the mucous membrane was reddened; the rugae on the posterior and inferior surface were enlarged and red, and the mucous membrane markedly swollen. Little was seen on opening the small intestine, but the interior of the colon was slightly reddened; it contained some curdled milk. The mucous membrane was studded with pearly-white tubercles 2 to 4 mm. in size, consisting of the enlarged solitary glands. The liver was enlarged, very pale, and yellow in colour. The gall bladder was full. The kidneys were congested, the bladder three parts full; the spleen, not enlarged, was darker in colour than normal; the lungs were healthy; both ventricles of the heart were empty, and both auricles filled by soft clot. There was very little blood in the body generally.

I had the opportunity of seeing the mother on August 11th, that is, on the fourth day, seventy-two hours after eating the fungi. She was in bed, markedly jaundiced, and in great pain, rolling from side to side, and very restless. The pain was paroxysmal and cramp-like. She said that it began in the lower part of the abdomen, gradually increasing until it ended in the epigastrium. She was much too ill to examine minutely. During the night she aborted, about the third month. On the next day, August 12th, at 11 a.m., she was scarcely conscious; the pulse was 96, and she lay on her back moaning with her head retracted, restless, throwing her arms about. She had passed no urine; respiration was irregular, suggestive of the Cheyne-Stokes type. At 3 p.m. her pulse was 116, the temperature was 98.2°, the pupils were equal and normal in size. At 7 p.m. the pulse was 110, the temperature 97.6°. She died at 11 p.m. (fourth day).

The man had pain of the same character, much thirst, continued diarrhoea, and vomiting. His face was bathed with perspiration during the paroxysms of the pains, and had the pinched and retracted look which the French speak of as *la face vultueuse*. The symptoms gradually decreased, and he was convalescent on August 15th (eighth day).

Dora (the daughter) was removed to the Lynn Hospital under the care of Dr. J. R. Kingdon, by whose kindness I am allowed to publish the following notes: She was sick on the morning of August 12th (fourth day, 87 hours). The temperature was 99.6°. On August 13th she was free from pain except when the bowels acted; one action consisting of blood-stained mucus and undigested curds. The temperature was 99°, she passed 6 oz. urine, which did not contain albumin; the liver was enlarged, the tongue clean, and the pupils rather small. She complained greatly of thirst. On August 14th the bowels acted five times, the stools containing much blood and mucus. The temperature was 97°. She was given a mixture of chlorodyne and bismuth carbonate, and was fed on albumin water, broth, Benger's food and barley-water. On August 15th three loose motions, with traces of blood-stained mucus were passed. She still had slight tenesmus (eight days after eating the fungus). The face was still pale and pinched. On August 17th (tenth day) she was convalescent.

It is evident that *A. phalloides* is a highly poisonous fungus giving rise to a distinct group of symptoms resembling those of ptomaine poisoning. To it and to its varieties have been traced 18 deaths since 1900 in France alone.

Eighteen Fatal Cases of Poisoning by the Phalloides Group of Amanitae.

No.	Name of Reporter.	Date.	Sex.	Age.	First Symptom Appeared.	Death.	Treatment, etc.	Bulletin Société Mycologique de France.
1	Drs. V. and X. Gillot	1900, Sept. 17	F.	12	Hours. 11	Hours. 72	Emetics, purgatives, belladonna	1902, vol. xviii, p. 34.
2	"	"	F.	9	11	48	" " "	"
3	"	"	F.	3	11	28	" " "	"
4	"	1901, Sept. 30	F.	41	10	45	" " "	" " P. 41.
5	"	"	M.	79	11	63	" " "	"
6	"	"	F.	20	10	106	" " "	"
7	L. Rolland	1902, Oct. 20	M.	37	10	54	Atropine and morphine hypodermically	" " P. 418.
8	"	"	M.	32	10	53	" " "	"
9	"	"	M.	22	10	78	" " "	"
10	B. Souché	1903, Aug. 13	F.	50	4	48 (?)	" Atropine hypodermically	1904, vol. xx, p. 40.
11	"	"	M.	45	18	48	" " "	"
12	"	"	F.	11	18	96	" " "	"
13	"	"	F.	3	18	96	" " "	"
14	"	" Sept. 1	F.	10	10	104	" " "	" " P. 45.
15	"	"	F.	7	10	112	" " "	"
16	"	"	M.	4	10	105	" " "	"
17	Dr. X. Gillot	1904, Oct. 16	F.	34	9	48	" " "	1905, vol. xxi, p. 58.
18	V. Harlay	" Aug. 31	M.	23	17	72	" " "	" " P. 107.

The above table shows (1) the long period which usually elapses between the ingestion of the fungus and the appearance of the first symptom, averaging between ten and twelve hours; (2) the average period in which it proved fatal—72 hours; (3) the 6 cases treated by atropine proved fatal, as did the 3 others in which belladonna was given; (4) half the cases were under 20, and a third under 10 years of age.

We owe most of our knowledge of phallin to Kobert.² He found it to be a toxalbumin, very similar to the toxalbumin ricin contained in the seeds but not in the oil of the castor-oil plant, and abrin in the scarlet and black beans of *Abrus precatorius*—jequirity.

The most commonly observed symptoms in a case of phallin poisoning are, vomiting not occurring for several hours (10 to 12) after eating the fungus, diarrhoea, pain in the abdomen, cramps of the stomach and diaphragm, intense thirst, cold sweats, collapse, sometimes headache, delirium, suppression, more or less complete, of urine, slight but distinct jaundice in the latter stages of severe cases, subnormal temperature, sometimes cramps in the limbs.

Post-mortem Signs.—Absence of post-mortem rigidity, marked hypostatic discoloration, inflammation of the gastro-intestinal mucous membrane, localized ecchymoses of the liver, alimentary canal, pleura and lungs, enlarged solitary glands, fatty degeneration of the liver, fluidity of the blood, hyperaemia of the meninges.

Treatment.—The orthodox rule of emptying the stomach and intestines of course applies only to those cases seen in the early stage; usually the patient has been vomiting and purging long before he is seen—of course a dose of castor oil may be required. To relieve the great suffering morphine is almost sure to be required. To decompose any phallin left in the stomach, permanganate of potash, as has been suggested, should be tried. The subcutaneous administration of normal saline solution (sodii chlor. 3j, aquae Oj) has been used with success.

Dr. Ramond treated five cases of poisoning by *A. phalloides* with hypodermic injections of atropin gr. $\frac{3}{50}$, morphine gr. $\frac{1}{4}$. Three of these died after fifty-four, fifty-eight, and seventy-eight hours' suffering. To the other two he gave injections of 300 c.cm. of artificial serum, and they recovered.³

Three specimens of *Amanita phalloides* Fr. in various stages of development. Section of a full-grown specimen showing the volva (poison cup). From Nature (reduced).

The recognition of *A. phalloides* and its immediate allies is not difficult. Although it sometimes resembles the common mushroom, *A. phalloides* is never anything else but white beneath the cap, where everybody knows the mushroom is pink, purplish-brown, or almost black. On the top *phalloides* is frequently nearly white, but one can almost always see traces of yellowish-green, especially about the margin. The stem of the mushroom is nearly cylindrical—that is, nearly as thick above as it is below—but the stem of *phalloides* is always bulbous, and springs out of a cup made by the upper part of the bulb, as shown in the figure, the so-called "poison-cup."

The cup-like base of the stem, the permanently white gills, its shining pale greenish or yellowish-white top, and the fact that it never grows far away from trees, especially oak trees, should enable any one to recognize this most virulent fungus. There is one point more to be remembered about *phalloides*, namely, that it will "peel" almost as well as the common mushroom.

REFERENCES.

- ¹ For a full account of this case see *Lancet*, vol. ii, 1879. ² Kobert, R., *St. Petersburger Medicin. Wochenschrift*, Nos. 51 and 52, 1891, where a full account of the symptoms and post-mortem appearances is given. ³ *Bulletin Soc. Mycol. de France*, 1902, vol. xvii, p. 420.

CLINICAL EFFECTS OF ETHER ANAESTHESIA ON RENAL ACTIVITY.

BY

HAROLD PRINGLE, M.D., F.R.C.S.I., R. CHARLES B. MAUNSELL, M.B., F.R.C.S.I.,

Chief Demonstrator, Physiological Laboratory, Trinity College, Dublin. Surgeon to Mercer's Hospital, Dublin.

AND

SETON PRINGLE, M.B., F.R.C.S.I.,

Surgeon to Mercer's Hospital, Dublin.

[From Mercer's Hospital and the Physiological Laboratory, Trinity College, Dublin.]

THE following investigation was undertaken with the object of determining clinically the influence of ether anaesthesia on the secretion of urine, both as regards its quantity and its nitrogenous content. The investigation arose from a discussion we had in Mercer's Hospital regarding the relative advantages of chloroform and ether as a routine anaesthetic for operations, more especially in cases with renal disease. The idea for the investigation was suggested to us by a paper read last session at the Dublin University Biological Association by Professor Thompson, giving the results of his experiments on dogs; the method and the periods of collecting the urine employed by us, however, differ from his.

The method we adopted was to take periods before, during, and after anaesthesia, as follows:

Period I. Twenty-four hours, ending at 9 a.m. on morning of operation.

Period II. From 9 a.m. on the morning of operation till immediately preceding the administration of the anaesthetic.

Period III. During the induction of anaesthesia, ending with the abolition of corneal reflex.

Period IV. First half-hour of complete anaesthesia.

Period V. Second " " "

Period VI. Third " " "

Period VII. Six hours following anaesthesia.

Period VIII. Twenty-four hours following Period VII.