two winters after her return she suffered from bronchitis; but, in 1879, she had little or none. In the summer of this year she underwent great anxiety and mental worry, owing to the dangerous, and ultimately fatal, illness of a near relation, and her own health began to suffer. She had irregular action of the bowels; she always spoke of bilious attacks. In July of that year, she went to Harrogate, and there appears to have been very much out of health, and to have lost ground, though under most careful and skilful treatment; the bowels were regulated, and her diet—a point in which she was apt to err—attended to.

She returned to London early in November, but I did not see her until the 20th of that month, and found her emaciated, and weaker than when I had last seen her. She was suffering from flatulent distension of the bowels, which were, however, acting daily or every other day. She expressed the greatest dislike to physic, especially if aperient, from which she said she suffered much lately. Some simple carminative, and a carefully regulated diet, were enjoined.

This state of things continued for some weeks, with no great change; the tympanites continuing to annoy her. Fomentations, carminatives, copious tepid enemata of soap, oil, and water, occasionally of sweet-oil alone, were given, and generally for a time acted fairly well, sometimes freely. The tongue was generally clear; the pulse good; there was no increase of temperature; the urine was copious, depositing lithates, but otherwise normal; the spirits were very good generally, and she very hopeful of recovery, saying that, but for the flatulence, she felt well. About a month before the end, the bowels began to be confined, and she had occasional fits of nausea, which were relieved by diluted hydrocyanic acid. At length, it became necessary to have recourse to aperients, as the enemata failed to produce any effect. The tympanites increased; she was carefully dieted, and solid food prohibited. The bowels now became obstinately constipated, and she passed nothing for some days; withal, it was remarkable how little she seemed affected by it; in some ways, she seemed better, and would get up, and even go out. The most careful examination could detect neither stricture in the rectum nor physical signs of any visceral disease.

It was obvious that, aperients and enemata having failed, they should not be repeated. She was brought under the influence of opium, when the tympanites was relieved, but there was no action of the bowels. The large intestine could be felt, though the abdominal wall distended with fecal matter. Enemata again had no results; the obstruction was too high to be reached with the hand introduced into the bowel.

The question of surgical operation now discussed, but it was not accepted.

Things went on much in the same way for a few days, when I was summoned and found her in great pain. Nothing had passed; the general condition was serious. An operation was proposed, and it was done without loss of time. The ascending colon was opened, a quantity of fecal matter escaped, and there was some relief. But symptoms of peritonitis set in rapidly, with collapse; and she sank in a few hours.

On examination of the body, it was found that the colon above the stricture, which was situated at the splenic flexure, was enormously distended with fecal matter, that a portion of the intestines had given way, and that peritonitis had resulted from the extravasation. The bowel otherwise and the viscera generally were healthy. There was no cancerous infiltration of any organ, and the stricture was peculiar, presenting the appearance as of a cord tied round the gut. The passage was practically obliterated; probably had been so for some days; it had been gradually contracting for weeks, and, when complete occlusion of the passage occurred, the urgent symptoms had set in.

Beyond an epitheliomatous growth, pendulous from the inner part of the thigh, there was no disease. No glands were enlarged, no extension of malignant disease had taken place in any direction, and microscopic examination failed to detect any other than fibrous tissue in the stricture itself.

The origin of stricture of the bowel of this character is very obscure. I may add that I saw another case about a year ago, of very much the same character, in a woman about 60, where the origin was equally obscure.

LIABILITY OF DENTISTS.—The liability of a dentist for the consequences of his own carelessness, or want of skill, has been decided in the affirmative in the United States courts of law. A gentleman named Thomas J. Kelly brought an action, in New York, against a dentist for injuries caused by a piece of tooth, which was extracted, but allowed by the operator to drop down the plaintiff's throat while he was under the influence of nitrous oxide. The piece slipped from the forceps, and for four weeks the plaintiff suffered great pain. Judgment was given for 500 dollars damages, and this has been upheld on appeal.

OBSERVATIONS

ON THE PHYSIOLOGICAL AND THERAPEUTIC ACTION OF THE ELEMENT ARSENIC IN THE FORM OF ARSENITE AND ARSENIATE.

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BOTH oxides of the element arsenic—viz., arsenious and arsenic acids—are employed in medicine, either as such, or in combination with some base or other. In the *British Pharmacopœia*, the liquor potassæ arsenitis, the liquor arsenicalis, and the liquor sodæ arsenicatis, are given in practically equal doses. The solution strength of the former two is one part of arsenious acid in 120 parts of water; of the latter is one part of anhydrous arseniate of soda in 120 parts of water. Calculated out from their formulæ, it will be seen that the arseniate preparation contains a little over half as much metallic arsenic as either of the two arsenious acid preparations. This is practically recognising the arseniate as the more active preparation.

The present experiments were made in order to test this point; the importance of which is apparent. Chemically considered, the arseniate is a stabler form of combination than the arsenite, the two salts standing to one another in the same relation as the phosphate and phosphite of the analogous element phosphorus, though the difference in degree of stability between these two latter combinations is much greater.

On the action of arsenious acid, there has been much work done; on the action of arsenic acid, but little. It is known generally that, qualitatively, arsenic acid acts similarly to arsenious acid; but, beyond this very general statement, little definite is discoverable. Of late, a series of papers have appeared (*Archiv für experimentelle Pathologie und Pharmakologie*) on the subject of the mode of action of the element arsenic in its oxygen compounds. In the first paper (*Op. cit.*, Band xi, p. 213) the authors, Binz and Schulz, state, with reference to the comparative action of arsenious and arsenic acids, that some investigators have found that arsenic acid was slower in its action, whilst others have found it to be the more active preparation. They further state that, so far as their own task is concerned, which of these two statements is the true one is unimportant. It is clear that no such statement as this could be made in the face of very definite experimental results, giving superiority of action to either of the two oxides of arsenic.

Some work, however, had been already done on this subject. Thus, in a paper by Wöhler and Frerichs (*Annalen der Chemie und Pharmacie*, Band lxxv, 1848), "On the Changes which Organic Bodies, in particular, suffer in their Passage into the Urine;" two experiments with arsenic acid are quoted—one on a rabbit, one on a dog. From the results obtained, the authors concluded that arsenic acid was less poisonous than arsenious acid.

They further stated, as amongst the differences between the two oxides, that arsenic acid showed less local irritant and caustic action; that its effects appeared more slowly; and they suggested that these were probably, in part, due to a reduction to arsenious acid occurring in the alimentary canal; they thought that the marked injection of the large intestine which was found *post mortem* in the cases observed spoke in favour of this reduction.

With reference to this relative position of arsenious and arsenic acids, it is very natural that the analogous element, phosphorus, should have been looked to, in order to see if any particular relation existed between the corresponding oxides of this element. Of these, viz., the phosphorous and phosphoric acids, the latter, when its acidity is neutralised by a suitable base, e.g., soda, is very little, if at all, irritant; phosphate of lime is, of course, a natural constituent of the organism, and, indeed, it is probably in the form of phosphate alone that the element phosphorus occurs in the tissues.

As to the action of phosphorous acid, but little work has been done; it is not employed in therapeutics, nor mentioned in text-books on toxicology. In the above-mentioned paper (*Op. cit.*, p. 347), however, Wöhler and Frerichs quote some experiments, three in number, with phosphorous acid; the doses used were from 0.5 gramme to 1 gramme

of the pure dried acid, administered in dilute solution. The animals experimented on—a pigeon, guinea-pig, and cat—in each case died, but the experiments were less satisfactory than they might have been, since in two of the three cases there is no doubt that the fluid may, in part, have entered the air-passages. Indeed, the value of this paper of Wöhler and Frerichs, both with reference to arsenic acid and to phosphorous acid, lies less in the experiments themselves, than in their statement of what they conceive to be the position of matters. In the end paragraph, treating of this subject, they say: "It is not without interest that the analogy between the elements, arsenic and phosphorus, is manifest also in the action of these elements on animals. The lower oxides of both the arsenious and phosphorous acids are most poisonous, whilst, of the higher oxides, arsenic acid acts relatively mildly, but phosphoric acid is harmless." In addition to this general view, must be remembered their suggestion that arsenic acid is reduced to arsenious acid, and then becomes active.*

Further evidence on the subject of arsenic acid is very scanty. According to Taylor (*Medical Jurisprudence*, vol. i, p. 276), Orfila states it to be more poisonous than arsenious acid, but gives no instance. Dr. Glover (*Loc. cit.*) introduced four grains of arsenic acid into the stomach of a rabbit; the animal died in four hours, with symptoms of irritant poisoning. Dr. Christison (*Treatise on Poisons*, p. 284, 1845, ed. iv.) cites the potash salt; stating, however, that he has read of but two cases of poisoning with it. He further mentions an account of the accidental poisoning of seven horses with this salt; the animals died with the symptoms and morbid appearances of well-marked inflammation of the alimentary canal.

More recently, in the *Journal of Physiology*,† the results of experiments with arseniate of soda and arsenious acid are given. These, so far as stated, agree with those obtained in the present series of experiments. According to them, the arseniate would be decidedly the weaker poison, but, in particular, they show forth the relative slowness of action of the arseniate as compared with arsenious acid; the object in view, however, being different from the present one, the experiments were not planned so as to furnish the data requisite for a precise comparison of the relative activities of the two oxides. This same objection applies to the experiments of Sklarek, referred to in the above-mentioned paper in the *Journal of Physiology*.

The evidence, then, so far as it goes, is to the effect of qualitative similarity of action of the two oxides, but, as to the quantitative relation, the evidence is indefinite on the whole.‡ The view of Wöhler and Frerichs as to the relative position of the two acids, though clearly enough stated, is scarcely supported by sufficient experimental work; still, as representing a scientific aspect of the question, it must be treated as a theory of the mode of action of the element arsenic in its oxygen compounds.

Against this view, comes the more recent one of Binz and Schulz. These authors represent the mode of action of these oxides of arsenic on the tissues as one, the essential part in which is played by the element oxygen. Throughout, an analogy is drawn with the process of burning; thus, they liken the action to that of ozone, and they hold that the fatty degeneration of the cells, and the diminution of glycogen, which both obtain in cases of poisoning by arsenious acid, are the results of such burning.

When the precise mode of action, as they state it, is examined into, it will be seen that the above is an incomplete representation; for the authors consider that arsenious acid, in contact with tissues holding oxygen, takes from them this oxygen, and becomes arsenic acid; that in this higher form of oxidation the element arsenic readily parts with its oxygen to the protoplasmic tissues, and becomes reduced again to the state of arsenious acid. This process is held to continue indefinitely, reduction to arsenious acid being followed by reassumption of oxygen and reformation of arsenic acid; the steps are then retraced.

The process, then, as described by Binz and Schulz, would lead one to infer—indeed, there is actual statement—that the same molecule of protoplasm can take up oxygen from arsenic acid, reducing it to arsenious acid, and then may itself suffer reduction by the arsenious acid formed. It is just this to-and-fro movement (*Hin- und Herschwingen*) which, according to the authors, is the essential cause of

the disintegration of the tissues obtaining. Thus, then, we should have as cause for the tissue change, which is an undoubted fact, a process consisting of two precisely opposite steps—a burning and an unburning. It is, however, quite clear that no two such steps could yield any effective force whatever; for what the one does the other undoes; the resultant equals zero. That this is a true representation of the theory propounded will be seen on reference to the original papers. (Compare in particular vol. xi, pp. 213, 214, and again vol. xiv., p. 249, of the *Archiv für Experimentelle Pathologie und Pharmacologie*.) But even though one should not insist on this oscillation of the oxygen between arsenious acid on the one hand, and the protoplasmic molecule on the other, and should accept the view that arsenious acid reduces certain protoplasmic molecules, becoming itself thereby converted into the higher oxide, and then parts with its oxygen to certain other molecules, suffering thereby reduction to its former state, yet, even accepting this as their view of the process obtaining (a process to which, in no sense of the word, the term oscillation would be applicable), they would claim effect for each step in the process; i.e., reduction would effect disintegration of the tissues; so, also, would oxidation. Two opposite processes, burning and unburning, would be factors in the result obtaining.

Binz and Schulz instance, as analogous in kind, the action which occurs when nitric oxide, free oxygen, and a body capable of further oxidation, e.g., sulphurous acid, occur together. Here, as is well known, the nitric oxide takes up oxygen and becomes nitrous acid, and then suffers reduction by the sulphurous acid back to the state of nitric oxide; this process will continue indefinitely, provided a constant supply of fresh "oxygen" and fresh "oxidisable body" be maintained. It is, however, clear that this process is a continuous one and in one direction; there is a constant flow of oxygen by way of the carrier to the "oxidisable body"; at no time is oxygen passed on and then taken back.

If, without further criticism of previous statements, this representation be accepted as applicable to the function of arsenious acid in the organism, the position claimed for this compound will be that of a carrier of oxygen, whilst the active element will be oxygen, and the active preparation will be arsenic acid. The assumption of such would be quite warranted; for hæmoglobin appears to hold exactly such a position, as oxygen-carrier, in the organism. To such process the term "burning" would be strictly applicable. However, whether the last-mentioned theory, or the oscillation modification, be true, it is certain that, according to either theory, the two oxides of arsenic must have practically equal toxic value.

The method adopted by Binz and Schulz consisted in the admixture of arsenious acid and of arsenic acid, separately, with animal tissues just removed from the body, care being taken to secure intimate contact, whilst the steps of the process were effected as rapidly as possible, so that the metallic oxide might come into contact with tissues as near the living state as could be. The admixtures were then placed in an incubator. The further stages consisted in the separation by dialysis, and in the detection of the higher oxide where the lower had been used, of the lower oxide where the higher had been taken.

By such means, Binz and Schulz state that they were enabled to demonstrate that living protoplasm is capable of oxidising arsenious acid to the state of arsenic acid, and of reducing arsenic acid to the state of arsenious acid. But, even though this twofold action of oxidation and of reduction be granted, experiments of this nature yet yield no data as to the relative influence of these bodies on the organism. Indeed, with the knowledge that arsenious acid and arsenites are reducing agents, whilst arsenic acid itself is reducible, as, for instance, by sulphurous acid and by hydric sulphide, it would not be surprising to find that, in living tissues, where the processes of oxidation and of reduction are constantly going on, these oxides of arsenic should also take part in these processes. On chemical grounds, the settling of this question is extremely difficult; for qualitative negative results as to the oxidation of arsenious acid by living protoplasm will count as little against the positive results of Binz and Schulz; they will be put down to technical errors; whilst the quantitative solution of the question, viz., the determination of the amount of arsenious acid oxidised, would be extremely difficult, if, indeed, trustworthy results could be counted on.

There scarcely remains any other way of settling the question than by the examination of the symptoms produced. If the theory of Binz and Schulz hold, then, on their own admission, the two oxides of arsenic should be equally poisonous. This must be regarded as a test proposition.

Mode of Experimentation.—By the kindness of Professor Graham of University College, we were furnished with solutions of arsenious acid, arsenite of soda, and arseniate of soda, of definite strength. The

* Wöhler and Frerichs make reference to a paper by Weigel and Krug on the Different Effects of Pure and Impure Phosphoric Acid (*Casper's Wochenschrift für die gesammte Heilkunde*, 1844, No. 28, p. 455. The impurities here referred to are phosphorous and arsenic acids, both of which, according to the authors, render the comparatively inactive phosphoric acid very poisonous. The papers would be in point if the experiments were more conclusive, but the materials supplied are so scanty, that they are in themselves of but very little value.

† On the Action of Arseniate of Soda and Arsenious Acid on Frogs, *Journal of Physiology*, vol. i, No. 4, Drs. Ringer and Murrell.

‡ It must be mentioned that Garrod, in his *Materia Medica*, states that, as the result of numerous observations, he finds the metal arsenic, in the higher oxide as arseniate of soda, equally effectual therapeutically with the lower oxide, but milder in its action, and less likely to produce irritation of the mucous membranes.

ARSENIOUS ACID.

Date.	Weight of Frog in Grammes.	Dose and Solution Strength.	Death of Central Nervous System. (No voluntary movements, spontaneous or on stimulation. No reflex action unless stated.)	Death of Muscular Tissue. No Irritability of Muscles of:			REMARKS
				Anterior Limb.	Thigh.	Lower Leg.	
May 9	27	0.5 c.c. (1% As ₂ O ₃)	2 h. 15 m.	6 h. 30 m.	12 h.	12 h.	Desquamation began after 2 h. 45 m.; was marked at end of 6 h. 30 m.
" 11	20.7	0.1 c.c. (1% As ₂ O ₃)	7 h. 20 m.	9 h. 35 m.	9 h. 35 m.	9 h. 35 m.	The muscular irritability was not quite gone in thigh and lower leg at end of 9 h. 35 m. Desquamation slight after 5 h. 20 m.; more advanced after 9 h. 35 m., but not marked even after 24 h.
PART I.							
May 13	?	0.1 c.c. (0.5% As)	12 h. 15 m. (reflex action still present.)	26 h.	26 h.	26 h.	Desquamation well marked over trunk and slightly over limbs at end of 12 h. 15 m. The weight of frog was forgotten, but it was a small specimen, less than that of 11th.
" 15	18.5	0.1 c.c. (0.25% As)	—	—	—	—	Experiment carried over 48 h.; frog appeared quite normal; slight desquamation appeared.
" 17	18.3	0.2 c.c. (0.25% As)	11 h. 15 m.	11 h. 15 m.	11 h. 15 m.	11 h. 15 m. (faint reaction.)	Death of the central nervous system must have occurred some time before the 11 h. 15 m., on account of the state of the muscles. Desquamation present after 11 h. 15 m.; after 24 h., well marked everywhere.
" 19	18.9	Ditto	24 h.	24 h.	26 h.	26 h. (faint reaction.)	Voluntary movement, sensation, reflex action, were all well marked at end of 12 h.; next morning completely gone. Desquamation present after 10 h., whilst frog was fairly lively.
" 22	21.5	Ditto	24 h.	24 h.	24 h.	24 h.	Frog fairly lively in evening, but respiration not good; next morning dead. Desquamation well marked after 24 h.
" 24	16.5	Ditto	11 h. 40 m.	24 h.	24 h.	24 h.	Muscles reacted vigorously everywhere at end of 11 h. 40 m.; next morning reaction = 0. Desquamation fairly marked after 11 h. 40 m. over back, slightly over limbs; subsequently more marked.
" 26	22.9	Ditto	24 h.	24 h.	24 h.	24 h.	Respiration slow at end of 12 h., else frog fairly well; next morning rigor mortis strong everywhere.
" 30	21.6	Ditto	24 h.	24 h.	24 h.	24 h. (faintest reaction.)	At end of 12 h., frog helpless, though sensation and reflex action good.
PART II.							
June 1	21.7	0.3 c.c. (0.125% As)	36 h.	36 h.	36 h. (faint)	36 h. (faint)	After 33 h., frog moribund, scarcely respired, but heart still seen beating through abdominal walls. Desquamation fairly marked after 24 h. over back; after 36 h., desquamation everywhere.
" 7	14.6	Ditto	24 h.	24 h.	24 h.	24 h.	At end of 12 h., frog moribund. Respiration 0, but heart still feebly beating. Voluntary movement, sensation, reflex action still present. Next morning, frog dead; muscles also. Desquamation slight after 12 h.; well marked after 24 h.
" 9	23.8	Ditto	—	—	—	—	Frog still alive after 29 h. (the other two frogs being dead). Voluntary movements helpless; respiration irregular; desquamation slight. Frog killed.
" 14	15.7	0.2 c.c. (0.125% As)	27 h.	27 h. (faint)	27 h. (faint)	27 h. (more marked, but slight).	Desquamation general but slight after 27 h.
" 16	16.4	Ditto	39 h.	39 h.	39 h.	39 h.	Frog was breathing very badly after 24 h., else condition seemed fair. Desquamation well marked over back after 24 h.; general after 39 h.
" 19	16.7	Ditto	12 h.	12 h.	12 h. (faint)	12 h. (faint)	The reaction of the thigh and lower leg muscles was only on right side. Desquamation slight after 12 h. over back; general and more marked after 24 h.
PART III—LARGER DOSAGE.							
June 22	24.45	0.5 cc. (1% As ₂ O ₃)	1 h. 10 m.	9 h. 30 m.	9 h. 30 m.	9 h. 30 m. (still present)	Desquamation commenced at end of 1 h. 30 m.; marked over whole body after 9 h. 30 m.; after 28 h. still more marked, strips off in sheets.
" 26	19.6	0.66 c.c. (1% As ₂ O ₃)	0 h. 40 m.	5 h. 10 m.	5 h. 10 m.	8 h. 40 m. (very faint).	Desquamation well marked after 4 h. over back of trunk and thigh.
" 29	20.75	Ditto	0 h. 50 m.	5 h. 30 m.	5 h. 30 m.	9 h. 30 m.	Desquamation slight over back after 2 h. 30 m.; strongly all over after 5 h. 30 m.
July 31	23.4	Ditto	0 h. 50 m.	4 h.	4 h.	4 h. (faint)	Desquamation well marked all over after 4 h.
Aug. 23	19.5	Ditto	0 h. 45 m.	7 h. 15 m.	7 h. 15 m.	7 h. 15 m. (very faint).	Desquamation well marked after 3 h. 15 m., epidermis stripping in large flakes.
" 25	16.5	Ditto	0 h. 50 m.	5 h. 40 m.	5 h. 40 m.	5 h. 40 m. (distinct).	Desquamation general 3 h.; after 6 h. more advanced.
" 26	20	Ditto	0 h. 35 m.	(Still present after 3 h. 15 m.)	(Still present after 3 h. 15 m.)	(Still present after 3 h. 15 m.)	Desquamation began after 40 m. Muscular irritability was strong in thigh and calf after 3 h. 15 m.; moderate in the anterior limb of one side, not in the other.
" 28	22	Ditto	0 h. 50 m.	4 h. 45 m.	4 h. 45 m.	4 h. 45 m. (still present)	Slight desquamation after 60 m.; well marked everywhere after 4 h. 45 m.
" 29	24.1	Ditto	0 h. 50 m.	6 h. 10 m.	6 h. 10 m.	6 h. 10 m.	Desquamation present after 50 m. over whole back; marked everywhere after 6 h. 10 m.

ARSENITE OF SODA.

May 9	27	0.5 c.c. (1% As)	1 h. 35 m.	6 h. 30 m.	12 h. (flicker)	12 h. (flicker)	Desquamation began at end of 2 h. 40 m. over back; well marked everywhere after 6 h. 30 m.
" 11	23.3	0.1 c.c. "	2 h. 30 m. (? faintest reflex action still persisted.)	7 h. 15 m.	9 h. 30 m.	9 h. 30 m. (flicker.)	Desquamation began after 5 h. 15 m., and became well marked all over.
PART I.							
May 13	?	0.1 c.c. (0.5% As)	8 h. 30 m.	12 h.	12 h. (faintest flicker.)	12 h. (flicker)	Desquamation began after 12 h. over back and back of head, and slightly over limbs.
" 15	18.5	0.1 c.c. (0.25% As)	—	—	—	—	Experiment carried over 48 h.; frog appeared quite normal at end; no desquamation.
" 17	19.7	0.2 c.c. (0.25% As)	11 h. 30 m.	11 h. 30 m. (feeble reaction.)	11 h. 30 m. (feeble reaction.)	11 h. 30 m. (feeble reaction.)	Frog appeared fairly normal after 4 h.; the feeble reaction of muscles persisting after 11 h. 30 m., was of course gone by next morning.
" 19	20.9	Ditto	26 h.	27 h.	27 h. (faint response.)	27 h.	Desquamation well marked over back, slightly over limbs after 12 h.; marked all over after 24 h. At end of 24 h., voluntary movements still present, but sprawling and helpless; whilst sensation and reflex action appeared, if anything, excessive.
" 22	20.5	Ditto	27 h. 30 m.	30 h. 30 m.	30 h. 30 m. (faint response.)	33 h. 30 m. (faint response.)	Desquamation began after 10 h., whilst frog was breathing well and appeared well.
" 24	16.8	Ditto	11 h. 30 m.	11 h. 30 m. (feeble reaction.)	11 h. 30 m. (feeble reaction.)	11 h. 30 m. (feeble reaction.)	Desquamation. At end of 22 h., the respirations were very shallow; voluntary movement, when excited, very sprawling. Sensation appeared, if anything, excessive.

ARSENITE OF SODA—continued.

Date.	Weight of Frog in Grammes.	Dose and Solution Strength.	Death of Central Nervous System. (No voluntary movements, spontaneous or on stimulation. No reflex action unless stated.)	Death of Muscular Tissue. No Irritability of Muscles of:			REMARKS.
				Anterior Limb.	Thigh.	Lower Leg.	
May 26	19.3	0.2 c.c. (0.25% As)	12 h. (v. remarks)	24 h.	24 h.	24 h.	At end of 12 h., respirations were very slow and shallow, voluntary movements sprawling, helpless. Sensation appeared, if anything, excessive. Frog probably near death, but not seen till next morning. Desquamation commencing after 12 h.; well marked over whole body after 24 h.
" 30	18.6	Ditto	12 h.	24 h.	24 h.	24 h.	Frog not quite dead at end of 12 h., but distinctly moribund. Respirations quite ceased, but voluntary movements still present, though helpless and sprawling; reflex action good, and sensation appears excessive. Desquamation slight after 12 h.; well marked all over after 24 h.
PART II.							
June 1	20.5	0.3 c.c. (0.125% As)	23 h.	23 h.	23 h.	23 h.	At end of 12 h., nothing special except irregularity and slowness of respiration. Desquamation, none after 12 h.; well marked after 24 h.
" 5	17.35	Ditto	24 h.	24 h.	24 h.	24 h.	Desquamation, none after 12 h.; well marked after 24 h.
" 7	12.9	Ditto	11 h. 30 m.	11 h. 30 m.	11 h. 30 m.	11 h. 30 m. (faintest response.)	Desquamation over back after 11 h. 30 m.; well marked everywhere after 24 h. Respiration very slow and irregular after 4 h.
" 9	16.9	Ditto	12 h.	12 h. (faint response.)	12 h. (faint response.)	12 h. (stronger but feeble.)	Frog moribund at end of 9 h.; slight desquamation after 12 h.; after 26 h. more marked.
" 14	16.8	0.2 c.c. (0.125% As)	52 h.	52 h.	52 h.	52 h.	After 48 h., respirations very slow, irregular, cease for long intervals; voluntary movement very feeble; sensation and reflex action appear fair. Desquamation slight after 32 h.; well marked after 48 h., and general.
" 16	16.1	Ditto	24 h.	38 h.	38 h.	38 h.	Frog not quite dead at end of 24 h., but moribund, still sensation and reflex action appear good. Muscles not tested till next morning, but probably irritability gone soon after 24 h. Desquamation well marked over back after 24 h., general after 38 h.
" 19	19.25	Ditto	—	—	—	—	Frog appeared normal after 72 h.

ARSENATE OF SODA.

May 9	27.5	0.5 c.c. (1% As)	12 h. (faint reflex action persisted)	24 h.	24 h.	24 h.	At end of 12 h., the muscular irritability was marked everywhere. Slight desquamation all over body after 24 h.
" 11	20.4	0.1 c.c. (1% As)	28 h.	28 h.	28 h.	28 h.	Frog appeared rather sprawling at end of 21 h., else movements, sensation, and reflex action appeared good. No note as to desquamation.
PART I.							
May 13	?	0.1 c.c. (0.5% As)	—	—	—	—	Weight of frog not noted, but smaller than on 11th.
" 15	18.5	Ditto	—	—	—	—	After 48 h., frogs appeared quite normal. Experiment ended. On May 19th, the experiment proceeded 72 h.; frog quite normal.
" 17	15.5	Ditto	—	—	—	—	
" 19	14.8	Ditto	—	—	—	—	
" 22	15.5	Ditto	—	—	—	—	
" 24	14.5	Ditto	—	—	—	—	
" 26	18.7	Ditto	—	—	—	—	
" 30	15	Ditto	—	—	—	—	
PART II.							
June 1	15	0.3 c.c. (0.25% As)	—	—	—	—	After 57 h., frog apparently still living; during 27 h. frog continued in a state of complete voluntary paralysis, prone, and not moving unless stimulated, but then kicking out hind limbs vigorously. Respirations during this time ceased, but heart still acting. This was condition at end of 57 h. Frog ultimately died. Desquamation present over back and fore limbs after 52 h.
" 5	17	Ditto	—	—	—	—	After 46 h., frog lively; no desquamation.
" 7	13.3	Ditto	—	—	—	—	After 40 h., frog lively; no desquamation. A very little of the injection escaped from the puncture in this case.
" 9	14.8	Ditto	29 h.	—	—	—	At end of 26 h., there was no respiration, but frog was still able to turn over from back. After death (29 h.) the muscles reacted well (no note subsequently). No desquamation.
" 14	15	0.2 c.c. (0.25% As)	—	—	—	—	After 31 h., frog appeared normal. No desquamation.
" 16	15.2	Ditto	64 h.	64 h.	64 h.	64 h.	At end of 63 h., frog still living, and voluntary movements fairly vigorous, though ill controlled; sensation and reflex action good. No desquamation.
" 19	18.75	Ditto	—	—	—	—	After 72 h., frog appeared normal. No desquamation.
PART IIIA.							
June 22	23.8	0.5 c.c. (1% As)	9 h. 40 m.	—	—	—	At time of death, viz., 9 h. 40 minutes from injection, the muscles reacted strongly everywhere. Desquamation general at end of 28 h., but only slight. No desquamation at time of death.
" 26	18.3	Ditto	8 h. 45 m.	21 h. 15 m.	21 h. 15 m. (fair reaction)	21 h. 15 m. (good response)	6 h. 15 m. subsequently, there was no reaction of muscles anywhere. At end of 21 h. 15 m., practically no desquamation.
" 29	23.7	Ditto	9 h. 30 m.	22 h. 30 m.	22 h. 30 m.	22 h. 30 m. (faint reaction)	Frog appeared moribund at end of 6 h. 45 m. The reaction in the lower leg-muscles after 22 h. 30 m. was faint but distinct. No desquamation after 9 h. 30 m.; slight after 22 h. 30 m.; more marked after 26 h.
July 31	25.7	Ditto	9 h. 20 m.	—	—	—	Respiration much affected at end of 4 h. Muscles reacted powerfully at end of 9 h. 20 m., and everywhere. No note subsequently as to muscles. Desquamation very slight after 9 h. 20 m.; after 31 h., much more advanced.
PART IIIB.							
Aug. 24	18.8	0.5 c.c. (5% As)	3 h. 20 m.	27 h.	27 h. (faint reaction)	27 h. (fair reaction)	After 7 h. 20 m., very slight desquamation over back; none elsewhere; at end of 27 h., well marked.
" 25	16.6	Ditto	2 h.	22 h.	22 h. (faint reaction)	22 h. (feeble reaction)	At end of 22 h., the reaction of thigh-muscles was only obtained by applying electrodes to bare muscle; the lower leg-muscles reacted more distinctly, and through skin. After 5 h. 45 m., ? any desquamation; after 22 h., desquamation very strongly marked, almost as strongly as for the arsenious acid frog.
" 26	18.75	Ditto	3 h. 20 m.	17 h.	17 h. (slight reaction)	17 h. (slight reaction)	Frog was very bad, ? moribund, after 1 h. 20 m. Moderate desquamation after 17 h.; after 41 h., desquamation was quite as advanced as in arsenious acid frog.
" 28	18.6	Ditto	5 h. 15 m.	19 h. 15 m.	24 h. 35 m.	24 h. 35 m. (faint response)	Respiration much affected after 1 h. 45 m., also movements sprawling; ? any desquamation after 5 h. 15 m.; after 20 h., desquamation well marked.
" 29	28.1	Ditto	6 h. 15 m.	15 h. 30 m.	17 h. 15 m. (distinct reaction)	17 h. 15 m. (distinct reaction)	After 2 h. 15 m., still breathing, but movements sluggish; ? any desquamation after 6 h. 15 m.; slight general desquamation after 15 h.; after 43 h., desquamation more marked.

arsenious acid solution contained 1 per cent. of the oxide, As_2O_3 , the arsenite and arseniate of soda solutions contained each 1 per cent. of the element arsenic. From the formula As_2O_3 is easily calculated the quantity of the solution of arsenious acid which must be taken in order to contain as much of the element arsenic as given quantities of the arsenite and arseniate solutions. The strengths of all three would have been made equivalent to 1 per cent. of the element, but for the insolubility of arsenious anhydride.

Of these solutions, definite quantities, containing known and therefore comparable quantities of the element arsenic, were injected under the skin of the back of frogs. The common English frog (*Rana temporaria*) was taken; and care was observed to select, for the parallel series of experiments, specimens as nearly as possible of the same size and condition. Each experiment consisted in the injection of three frogs with the above arsenic preparations; for each frog, one of the arsenic compounds. The three injections were performed at the same hour, one after the other, as speedily as possible. Each frog was then placed under a separate bell-jar, and observed. The observations extended over two days, as a rule; sometimes over three; they were taken morning, noon, and evening. The experiments were performed during the months of May and June 1882; a few during August of the same year.

Thus, comparable quantities of the element arsenic, in three different states of combination, were administered to frogs chosen as nearly alike as possible. The results obtained are given in the form of tables (pp. 1138-39). The headings chosen for the columns will not need explanation, except perhaps that described as "Death of the Central Nervous System". The evidence of this death is, that the frog neither moves spontaneously nor answers to any stimulus by volitional movements. The absence of these latter in response to stimulation has been assumed to correspond with abolition of sensation. This absence of the more complex co-ordinated movements was at times to be noted whilst the less complex co-ordinated movements constituting reflex action still persisted; but, as a rule, reflex action outlasted but a very short time the abolition of the more complex movements. Hence, practically, "Death of the Central Nervous System" corresponded to:

1. Absence of spontaneous movement;
2. Absence of volitional movement on stimulation;
3. Absence of reflex action.

Though these three occurred so nearly together as practically to constitute but one group, the above is the order of their disappearance. It may be further mentioned that, together with the above noted, the automatic centres of respiration and circulation ceased to perform their functions. It is clear that, with a peripheral apparatus, nerves, and muscles, still efficient, the above indicates extinction of the functions of the central nervous apparatus of the brain and spinal cord.

The subsequent death of this peripheral apparatus is given in the next column, headed "Death of Muscular Tissue". The nerves are left out, because their condition is rather less readily examined, and hence not so well adapted for comparative examination. The sciatic nerves were, however, tested in nearly every case; and it was established that, at the time of "death of the central nervous system", as above evidenced, the nerve-trunks still conducted well; and, further, that death of the nerve-trunk preceded, as a rule, by a considerable period, the death of the muscular tissue itself. Hence, then, the two test-tissues chosen are:

1. That of the central nervous system;
2. That of the muscles.

In the column headed "Remarks," is given the peculiar action on the skin in the shape of desquamation, which may vary from a slight separation of the epithelium to a condition in which this may be picked up or scraped off in large flakes. This condition has been fully described in the *Journal of Physiology*, vol. i. In this same column, anything noteworthy at the time is recorded.

[To be continued.]

SOUTH MEATH AND DROGHEDA MEDICAL ASSOCIATION.—The usual annual meeting of this society was held on November 24th, in the Board-room of the County Infirmary, kindly lent by Dr. MacDonnell. Several papers were read, and matters discussed of interest in connection with the profession; after which the members dined together at the Queen's Arms Hotel. The chair was occupied by Dr. Gartlan, senior member, who presided with his usual tact and ability. A number of toasts were given and happily responded to by the several members, many of whom also contributed much to a very enjoyable and intellectual evening by capital singing. Owing to pressing duties and the unfavourable weather, a number were debarred from coming, who had signified their intention to be present.

NOTE ON MESENTERIC CYSTS AND TUMOURS.

By T. SPENCER WELLS,

President of the Royal College of Surgeons; Surgeon to the Queen's Household.

UP to the date of the publication of my last work on *Ovarian and Uterine Tumours*, I had not met with any cases of mesenteric cysts or tumours. In the chapter on diagnosis, I had spoken of collections of fluid in the peritoneal cavity more or less completely encysted by adhesions—of hydatid cysts—of peritoneal cancer—and of fibro-plastic and fatty tumours of the peritoneum, of the omentum, and of the sub-peritoneal cellular tissue. But I had not then read Mr. Doran's interesting case of cyst of the great omentum, published in the *Obstetrical Transactions* (vol. 23, page 165); nor had I ever seen a solid tumour of the mesentery. During the past summer, however, I have seen one case closely resembling Mr. Doran's, with Dr. Duke of St. Leonard's, but which appeared to be rather mesenteric than omental. I did not attempt to remove the cyst, only removing the fluid contents. The lady died a few weeks afterwards; and, as no examination of the body was permitted, I cannot say more of the case. About the same time, I removed a large solid mesenteric tumour from a married lady, forty years of age, a patient of Mr. Arthur Jackson of Sheffield. She had been married eighteen years, but had no children, though she had a premature labour some months after marriage. Two or three early abortions followed, and no further pregnancy. She was in good health until about four years ago, when pain in the iliac regions and slight abdominal enlargement was noticed; but no tumour was discovered until early in 1881. After that, increase was very manifest, with some apparent diminution after each menstrual period. Various opinions were given as to the nature of the tumour—some believing it to be ovarian, others uterine. I frankly confessed my own inability to give a very positive opinion as to its nature or connections; but expressed a very confident belief that I could remove it without any unusual difficulty or danger. It was quite solid, central in the abdomen, freely movable, about the size of an adult head, and imparting transmitted rather than associated movements to a uterus somewhat enlarged. I removed the tumour on June 20th, 1882, at Sheffield. Mr. Shaw kept the patient in a state of perfect anaesthesia with bichloride of methylene, and I was ably assisted by Mr. Arthur Jackson, Mr. Favell, and Dr. Redpath. Phenolised spray and all the usual antiseptic precautions and dressings were carefully employed. The tumour was solid, and its origin was clearly in the cellular tissue, at the root of the mesentery proper, near the lumbar vertebrae. The ascending colon was closely connected with the tumour in front, and to the right. All its blood-supply was derived from the mesenteric vessels. Those which were divided were secured with carbolic silk, the ends of all the ligatures cut off short, and returned. The uterus and both ovaries were healthy. No drainage was employed, and the wound was closed exactly as after ovariectomy. There was some sickness during the first three days; but recovery may be said to have followed without fever. The highest temperature was on the third day, but was only 100° . The patient left her bed on July 12th, and I have heard of her lately as being in good health.

The tumour was sent to the Sheffield Pathological Society for examination and report. I have not yet received the report; but I hope it will be forwarded, as the removal of a solid mesenteric tumour may still be regarded as a surgical curiosity.

ON FREE REMOVAL OF MAMMARY CANCER, WITH EXTIRPATION OF THE AXILLARY GLANDS

AS A NECESSARY ACCOMPANIMENT.*

By W. MITCHELL BANKS, M.D., F.R.C.S.,

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I HAPPEN to live in a district where cancer is common. Liverpool is in an area which the Registrar-General's statistics show to be clearly of a malignant habit. The reason of this I cannot say; but it is a fact, and possibly thus it is that my attention has been for some time drawn to the subject. In 1877, I published a little article in the *Liverpool and Manchester Medical and Surgical Reports*, based upon a paper read before a meeting of the Lancashire and Cheshire Branch of the Association. In it I asserted that surgeons did not remove cancers of the breast. Five years later, before the whole Association, I reassert

* Read in the Section of Surgery at the Annual Meeting of the British Medical Association in Worcester, August 1882.