Observations on some recent cases of poisoning : read before the Newcastle and Gateshead Pathological Society, March 13, 1856 / by George Robinson.

OBSERVATIONS

ON SOME

Recent Cases of Poisoning,

READ BEFORE THE

NEWCASTLE AND GATESHEAD PATHOLOGICAL SOCIETY,

[redacted] 1856,

By GEORGE ROBINSON, M.D.

Gateshead:
PRINTED AT THE OBSERVER STEAM PRESS, No. 27, HIGH-STREET,
BY D. DUNGLINSON.

1856.

On some Recent Cases of Poisoning.

The subject of poisoning having recently attracted much attention in this country, and having been professionally present during the judicial investigation of two important cases, I am induced to think that a few brief comments upon them, may not be altogether uninteresting to the members of this Society. As my object, in thus reverting to the past, is also to endeavour to deduce from its examination some useful and beneficial suggestions for the future, I may be allowed, *in limine*, to advert to the very unsatisfactory position in which medical and chemical witnesses are placed during medico-legal inquiries of this nature; for, being compelled to appear on the side either of the prosecution or of the defence, they are consequently made to assume more or less of the appearance of partisans—a position derogatory to, and inconsistent with, the dignity and neutrality of science. In order, therefore, to give greater weight to scientific testimony, and secure its impartiality, it is most desirable that all evidence relating to the abstract principles of science, and their application to jurisprudential purposes, should be delivered from neutral ground, and not be exposed even to the suspicion of having been fettered or perverted, by association with zealous advocates or interested friends. I may also observe, that the remuneration at present allowed to medical and chemical witnesses, when summoned on behalf of the Crown, is absurdly inadequate, and calculated to impede, and often frustrate, the ends of justice. The cases to which I am about to refer are—1. That of Joseph Snaith Wooler, tried at the Durham Winter Assizes, 1855, on the charge of poisoning his wife, by the repeated administration of small doses of arsenic. 2. That of William Palmer, convicted of the murder of John Parsons Cook, by poisoning with strychnia; and further charged with the murder of his wife, by the repeated administration of antimony.

It will be most convenient to consider these cases separately, and I shall therefore proceed briefly to detail the facts, and indicate the difficulties connected with each. The first and most important inquiry related to the cause of death—there being, in all these cases, more or less primâ facie reason for attributing it to natural agencies. And a rigid application of the principles of pathology, by showing us that the same symptoms may result from a great variety of causes, and that the connection between morbid appearances, and the cessation of life, is not always invariable and direct, does but increase the difficulty in arriving at a positive result from the medical facts alone.

In Mrs. Wooler's case the history of the symptoms, and of the *post-mortem* appearances, was briefly as follows :—She was 46 years of age, had resided in India for some time, and had always been considered a delicate and ailing person. On the 8th of May, 1855, when she was first visited regularly by Dr. Jackson, her symptoms resembled those of influenza, consisting of "slight difficulty of breathing, slight redness about the eyelids, slight tickling cough, and a very rapid, quick pulse." Afterwards, "diarrhœa and vomiting" appeared; also "hiccough, great dryness of the throat, great difficulty of swallowing, and ulceration of the tongue." These symptoms, with occasional intervals of remission, continued until death. "The symptoms indicated irritation of the whole tract of the alimentary canal, from the mouth downwards." The skin, particularly of the face, was red and rough. On June 23d, four days before death, tingling of the hands and tonic spasms of the muscles of the arms appeared. Twenty minutes before death she was suffering from violent and painful spasms of a tetanic character. The examination of the body took place on the 29th of June, two days after death. The body showed some emaciation. Thoracic cavities contained no liquid. Lungs less crepitant than usual: the apices of each infiltrated with tubercular matter to a slight extent. In the left lung, a cavity resembling in size and shape a kidney bean, and lined with mucous membrane: the lower and posterior portions of each lung loaded with black blood. The mucous coat of the trachea showed unusual vascularity, as did the mucous coat of the bronchial tubes to a greater degree as the tubes divided. The bronchial tubes contained frothy mucus. Œsophagus: slight vascularity of the mucous coat of upper portion. Liver of a saffron yellow hue, enlarged and encroaching on cavity of the thorax; upper portion of right lobe adherent to peritoneum, and its substance much softened, breaking down easily under the finger; the whole liver was softer than natural, but much less so than the part described. The stomach externally healthy; its mucous membrane exhibited great vascularity, and at the greater end spots of extravasation were distinctly visible under a lens; it was also soft and friable. The mucous coat of the duodenum was also softened. That of the jejunum normal. The mesenteric glands were of a dark colour. The upper part of the ilium was healthy, but the lower third exhibited numerous patches of ulceration, varying in size from a shilling to a crown piece, some involving the whole circumference of the gut, which at these parts was contracted and empty; the ulcerations extended in parts to the peritoneal coat, which was softened and easily torn: the colon shewed increased vascularity, its ulcerations smaller and less numerous; the rectum also slightly ulcerated. Pancreas highly vascular; spleen and kidneys slightly congested; uterus healthy; heart normal, and its right cavity filled with black blood. The body was disinterred on the 4th of August, when a large portion of liver was still contained in it, showing that the organ must have been greatly enlarged: it was also then discovered to have undergone the morbid change produced by fatty degeneration.

There being thus a tertain amount of natural disease present, the question arose wheher in this case death, with its attendant symptoms and necroscopic appearances, might not be accounted for without having recourse to the suspicion of a criminal act. General irritability of the mucous membranes is often observed in invalids,

and particularly in those who have resided in warm climates. Tubercular disease of the lung is commonly accompanied by ulceration of the lower third of the ilium. Obstinate vomiting may be induced by disease of any of the abdominal viscera, and could therefore be referred to the diseased liver, which would also to a certain extent explain the ulcerations of the large intestine, and the consequent dysenteric complication. The only symptom, therefore, which could not be accounted for by natural causes, was the tetanic spasm, commencing in the muscles of the forearm, and proceeding to general convulsions of the same character. But even this was not an insuperable difficulty, for it appeared that on the 2d of June she had a box of pills containing among other substances small doses of strychnia; and on June 14th, a fortnight before death, twelve pills, each containing one grain of nux vomica, were ordered, and a few were taken. These pills were gilded, and being covered with gold leaf, would be protected for some time from the solvent influence of the gastric fluids. It was therefore just possible that from this cause, added to a peculiar idiosyncracy in the patient, the characteristic effect of nux vomica might manifest itself in a severe form at a considerable period after its reception into the stomach. Had the presence of arsenic in the body of the deceased not been proved, it would therefore have been reasonable to refer her death to natural disease, for it is impossible to limit the possible complications of symptoms produced by natural disease. But as arsenic was undoubtedly present in the body, having been found in the interior of the liver and in other viscera, the inference became irresistible that it had been administered during life, probably with a guilty intent, and had either produced or aggravated the symptoms presented. Thenceforth the investigation turned more upon general than scientific evidence; and in the absence of an apparent motive and of direct proof of administration, a verdict of not guilty was returned. To the medical practitioner and the toxicologist the case must always possess much interest, as elucidating several important principles, among which may be enumerated :—1. The necessity of analyzing the ingesta, as well as the egesta, in cases of suspected poisoning. 2. The danger of commencing an analysis without having first proved the purity of the tests employed; one very important link in the chain of evidence being in this case nullified by the impurity of the muriatic acid used. 3. The propriety of keeping the stomach and intestines and their contents distinct from the other viscera, when removing the parts for analysis. 4. The duty of the medical practitioner to communicate suspicions of attempted poisoning to the patient, or his friends, or if the latter be suspected, to a magistrate or the police, as being the only means of relieving himself of a painful responsibility.

The preceding case shows the difficulty of determining, from an examination of the symptoms and *post-mortem* appearances alone, whether or not death was occasioned by poison, even though poison was undoubtedly present in all the viscera of the deceased. How much more difficult, therefore, must it be to demonstrate, from the same uncertain data, the fact of poisoning in a case where poison was not found in the body, and was not proved to have been administered during life? Yet, on analyzing the medical evidence which led to the conviction of William Palmer for the murder of John Parsons Cook by the administration of strychnia, it will be seen

that on one symptom alone, tetanic convulsions—a symptom which in Mrs. Wooler's case was considered indicative of poisoning, not by strychnia, but by arsenic—the whole case for the prosecution virtually rested.

The medical history of the case lay in a very narrow compas. Mr. Cook was 28 years of age; had led a life of excitement and dissipation on the turf; and had some months previously to his death been affected with syphilitic or pseudo-syphilitic symptoms. On November 14th, 1855, he was at Shrewsbury Races, and in the evening was taken ill with pain in the stomach and heat in the throat, symptoms relieved by drinking freely of warm water, and thus inducing vomiting. He went to Rugeley on the following day (15th), and on the 17th had a severe attack of vomiting. On the night of the 18th, about 12 o'clock, he felt ill for a a short time, the particulars not mentioned; but he said he was "just mad for two minutes." Some vomiting seems to have continued at intervals up to the time of death. On the 19th, about a quarter to 12 P.M., he screamed violently, beat the bedclothes with his hands and arms stretched out, complained of a sense of suffocation, and was violently convulsed, but perfectly sensible. On attempting to drink he vomited the liquid. On the following night (November 20), about the same time, he suddenly started up in bed, called out, and requested Mr. Jones, who slept in the same room, to rub the back of his neck. Mr. Jones, in doing so, observed that the muscles there were stiff and hard. Three or four minutes afterwards, having previously swallowed some pills, he threw himself back in the bed, and was violently convulsed. He then asked to be raised up or he should be suffocated. The convulsions lasted five or ten minutes: they affected all the muscles of the body, stiffening the limbs, and rendering the whole body so rigid that it was impossible to raise him. He then asked to be turned over on the side: he was quite sensible. The action of the heart then gradually ceased, and he died very quietly about ten or fifteen minutes after he first raised himself in bed and called for assistance. The head was bent back, the hands clenched, and the whole body arched backwards.

On Monday, November 26th, the first *post mortem* examination took place. The only unusual appearances were — 1. That the heart was contracted, and contained no blood. 2. There were numerous small yellowish-white spots, about the size of mustard seed, at the larger end of the stomach. 3. The blood was fluid. 4. The lungs were slightly congested and emphysematous. 5. There were some slight atheromatous deposits on the lining membrane of the aorta, 6. On examination of the body on the 25th and 26th of February, 1856, the muscles of the neck and trunk were relaxed, while those of the arms and legs still retained their rigidity. On the inner surface of the dura mater of the spinal cord, opposite the middle of the neck, there was a slight roughness of the surface, produced by the presence of a few irregularly-shaped gritty granules, of very minute size, embedded in the dura mater. They were found scattered over a space less than an inch square, and raised the surface in a degree barely perceptible by the finger or eye.

A careful analysis of the stomach and intestines and their contents was made by Dr. Taylor and Dr. Rees, and tests applied for the discovery of any of the poisons which could cause sudden death, "including prussic and oxalic acids, morphia, opium, strychnia, veratria, nicotina, and conia, as well as for mercury, antimony, and

the other metallic poisons. The result of the most minute examination was that there was only a slight trace of antimony in the parts examined, and no trace of any other substance." Antimony was also found in the liver, spleen, and kidneys. A special examination was made for strychnia and morphia, but no traces of either were discovered. The following process was used in thus searching for strychnia. The stomach was well washed with cold distilled water, to remove every soluble substance from the surface. The pyloric half was reserved, together with one half of the washings. The cardiac half, with the rest of the washings, was mixed with alcohol of about 840° specific gravity, acidulated with diluted sulphuric acid, and warmed to about 170°, frequently stirred, and allowed to digest for 24 hours. The liquid filtered off through a wet filter, was of a pale brownish colour, acid reäction, and *not bitter to the taste.* About one-fourth of this liquid was neutralized by carbonate of potash, and gently evaporated to dryness. The dry extract was treated with warm rectified spirits until exhausted. This was filtered. It formed a pale coloured liquid. It was again tasted by Dr. Taylor and Dr. Rees, but there was *no perceptible bitterness.* A portion of this liquid was evaporated gently on a (watch ?) glass on sand (bath ?), and films of a fine crystalline substance were left on the glass. The colour tests for strychnia (sulphuric acid and bichromate of potash) were applied to these films, but without satisfactory results. To the conclusion, *from the analysis*, that antimony had been taken into the system, and was present in the viscera during life, there could of course be no objection. But the opinion or belief that the death of this unfornate man was caused by strychnia, must, I think, always be surrounded with grave difficulties. Chemical analysis, we have seen, yielded no support to that opinion : on the symptoms and *post mortem* appèarances alone it rested. Yet there is not one of the latter which can be shown to be pathognomonic of, or invariably connected with, poisoning by strychnia. The emptiness of the heart, as correctly stated by Dr. Christison, depended rather upon the *mode* of death, than the *cause* of death. The immediate cause of the rigor mortis is too little known, and its period of occurrence and duration after death is too inconstant, for any valid inference to be drawn from its peculiarity, in these respects, in an individual case, while the idea, that in poisoning by strychnia, the tonic spasm passes uninterruptedly into cadaveric rigidity, is clearly a fallacy. And so with the symptoms. There is not one which may not result from other causes than strychnia.

There being, therefore, an utter absence of direct proof of poisoning by strychnia, the medical facts, with which alone I have now to deal, will admit of three different interpretations, viz. :—

1. That the deceased was poisoned by strychnia.
2. That he was poisoned by some other alkaloid.
3. That death may have been the result of natural causes.

Each of these hypotheses may be briefly and separately examined.

1. In support of the view taken by the prosecution, the only important medical fact, was the sudden and rapidly fatal attack of tetanic convulsions, to which may here be added, the general evidence that the accused had strychnia in his possession, and the requisite opportunity for its administration.

Against this conclusion may be urged.

A. The non-detection of strychnia by chemical tests, though

expressly searched for, in the stomach and intestines, by skilful and experienced analysts.

B. The absence of any bitter taste in the acidulated solution of the stomach and its contents. This fact is, I consider, utterly inconsistent with the presence of strychnia there; and whatever objections may be made to the colour tests, though their delicacy and conclusiveness seem to be beyond reasonable doubt, I cannot believe in the possibility of even the residue of a poisonous dose of strychnia being present in the stomach, without imparting its bitterness to an acidulated aqueous, or alcoholic solvent. It is true that the bitter taste may result from other substances than strychnia, but the latter cannot be present in a liquid, even in the proportion of a 500,000th part, without communicating to the palate the impression of its intense and permanent bitterness. By this natural test alone, used in a similar manner to that adopted by Drs. Taylor and Rees in the above-described process, I have seen students readily discriminate between the presence and absence of minute quantities of strychnia in fresh and putrid animal substances, in the stomachs of animals, &c. I cannot, therefore, adopt the opinion advanced in a paper read at the recent meeting of the British Association, that the presence of albuminous substances prevents the detection of strychnia by its taste.

C. The improbability of the analogous, but milder, paroxysm of the preceding night having been occasioned by strychnia, since it lasted only half-an-hour, whereas the twitchings and spasms of a large dose of strychnia would have continued for several hours.

D. The possibility of tetanic convulsions resulting from other causes than strychnia.

E. The fact that neither in this case, nor in cases of undoubted poisoning by strychnia, did the symptoms *per se* suggest, even to experienced medical practitioners, the idea of that cause of death until it was shown to be possible or probable by general and moral evidence.

The medical and chemical facts, therefore, seem to me scarcely to warrant, much less to necessitate, the conclusion that Mr. Cook was poisoned by strychnia. Nor can I regard the theories employed to account for its disappearance from the stomach as at all satisfactory, or consistent with what we know of the processes of absorption and excretion in the living body. The former, though often a rapid, is a gradual action; and its rapidity is to a great extent regulated by the solubility of the substance presented to the blood-vessels. Hence a difficultly soluble substance like strychnia would be but slowly removed from the stomach, though from its energetic nature the symptoms of its poisonous operation would rapidly follow the introduction even of a most minute quantity into the circulation. To maintain, therefore, that a minimum fatal dose of strychnia may be received into the stomach, and destroy life, without leaving in that organ even the imperceptible quantity required to impart a bitter taste to acidulated water or spirit, is, I think, irreconcileable with known physiological and chemical laws. And even as regards the possession of strychnia by the accused and its subsequent disappearance, it is possible that he obtained it for some other purpose, and destroyed it when suspicion fell upon him. This would be analogous to what occurred during the investigations into Mrs. Wooler's death, when a bottle marked "liquor arsenicalis" disappeared, having doubtless been destroyed to avert suspicion.

2. With reference to the second theory, as to the cause of Mr. Cook's death, it is only necessary to prove that tetanic convulsions may be produced by other poisons than strychnia. I shall content myself with naming those mentioned in Dr. Taylor's work on Poisons, and I find cases there recorded in which this symptom has followed the ingestion of water hemlock (*cicuta virosa*), water parsnip (*œnanthe crocata*), fool's parsley (*œthusa cynapium*), and laburnum (*cytisus laburnum*). It is true that stupor was generally present, along with convulsions, but in poisoning by strychnia the same circumstance is occasionally observed. The most poisonous alkaloids are, if not in the actual possession, within the reach of every medical man; and when it is remembered that even the 50th part of a grain of one of these has endangered the life of an individual, the facility of their administration in medicine may readily be conceived. None of them present such facilities of detection as strychnia, and the results of a chemical analysis would be very similar to those observed in the case now under consideration.

3. It only remains to consider the *possibility* of death having resulted from natural causes. Can any man assign a limit to the mysterious destructive powers generated within the body itself? And in a rapidly progressive and modern science like pathology, of what value is the negative experience of the oldest and wisest practitioner on such a point? Fifty years since, little or nothing was known of the true nature and causation of convulsive diseases; and are we quite sure that we may not be equally behind the next generation? But what we do know of convulsive diseases serves but to demonstrate the utter impossibility, in many instances, of connecting them with any adequate or immediate cause. Intestinal or dental irritation will suddenly convulse and destroy a strong and healthy child. The presence of a spicula of calcareous matter, or a few granules in the dura mater, will induce epilepsy in the adult. And tetanic convulsions are occasionally the immediate precursor of death in zymotic diseases, where the blood is infected with the poison of cholera, fever, or small pox. Looking, then, to the history of Mr. Cook's case, and to the existence in the dura mater of the spinal cord of a number of small gritty granules, was it not *possible* for the fatal paroxysm to have been an unusual and fatal form of spinal epilepsy? The source of irritation created by those granules was seated close to the spinal cord; it had, consequently, a greater tendency to induce disorder in the spinal than in the cerebral nervous system. And the possible existence of a form of epilepsy characterized by a sense of suffocation without unconsciousness, but followed by convulsions, is distinctly recognized by Dr. Marshall Hall in his "Essays on the Theory of Convulsive Diseases" (London, 1848). While, as regards the occasionally tetanic character of epileptic convulsions, I may be allowed to refer to a case which occurred in this very hospital (the Newcastle Infirmary), under the eye of the excellent house-surgeon and several students, during Palmer's trial, in which the opisthotmos was so complete that the patient rested wholly upon his head and heels.

I have ventured to reason upon the medical facts in this case somewhat freely and fully, because the trial has established a precedent which may hereafter endanger the lives of innocent persons. For, once let it be granted that symptoms alone will suffice to prove death from a particular poison, and charges of this kind may be made with frightful facility.

I have made but very slight reference to the general and moral evidence against the accused, believing its consideration to fall more strictly within the province of the Court and the jury, than of the medical witness or commentator. The toxicologist may use it to guide him in his inquiry, to indicate the path of special research, but his opinion should be formed solely from scientific facts and reasoning, and not be, even, in the slightest degree, influenced by popular suspicion and prejudice. If the non-detection of strychnia by Dr. Taylor and Dr. Rees tended to embarrass the case of the prosecution, the honesty of purpose with which those gentlemen discharged their important duty was evidenced by their abstinence from any attempt to misrepresent the phenomena observed in their analysis, or to convert negative facts into positive indications. And it must be allowed that the proof of a criminal administration of antimony was nearly complete.

Before leaving this case, I may take the opportunity of recording one or two facts which fell under my observation, when experimenting with strychnia, viz. :—

1. That common sulphuric acid, which always contains some nitric acid, reddens pure strychnia. In using the colour tests, it is therefore necessary to employ very pure sulphuric acid.

2. Strychnia, if given in solution, may destroy life immediately, by its action on the heart, *e.g.* — a small quantity, not above one-fourth of a grain, dissolved in acidulated water, was injected from a syringe down the throat of a rabbit. The animal almost instantly passed into a state of violent opisthotmos, and died in less than a minute. On examination, the heart was found gorged with blood, the coronary veins greatly distended, and one, on the anterior surface of the heart, had given way, and caused a large spot of ecchymosis in its substance, and beneath the serous pericardium.

3. Strychnia dissolves very readily in bisulphuret of carbon, which might be used instead of chloroform or ether to separate it in analysis.

4. It appeared to me to be more readily separated from, and detected in, animal than vegetable substances; and would consequently be found more readily in the stomach of a carnivorous, than of a herbivorous animal. To this cause I am inclined to refer the frequent failure to demonstrate its presence in the stomachs of rabbits poisoned by it, as a large quantity of vegetable matter is generally present there, even for a considerable time after the animal has taken food.

As the remaining case did not form the subject of a criminal trial, I should not have adverted to it, but that in connection with it my attention was particularly directed to the question of poisoning by antimony, and experiments were performed which may throw some light on the action of this important class of medicines. Mrs. Palmer's symptoms resembled those of severe English cholera—great prostration, vomiting, diarrhœa—and to that disease her death was at the time ascribed by the two experienced and respectable gentlemen who saw her during her fatal illness. She had been in Liverpool in September, 1854, during which month about 800 deaths from cholera occurred there, and immediately on her return she was seized with the above-mentioned choleraic symptoms. It was not till a year afterwards, when other cases had excited suspicion, that her body was examined for poison, and antimony discovered in the stomach and rectum, and throughout

all the viscera. My experiments were directed :—

1. To determine the earliest period at which antimony can be discovered in the blood and viscera after the administration of a single dose of tartar emetic.

2. To show that the comparatively mild and innocuous preparations of antimony may saturate the system with that metal as completely, and almost as speedily, as tartar emetic or other poisonous salts of antimony.

The process generally adopted was that of charring the animal matter by sulphuric acid, treating the residue with distilled water, acidulated by a little hydrochloric acid, filtering, and then testing by sulphuretted hydrogen and Marsh's apparatus. In every instance the metallic deposits on the porcelain were proved to be antimony by dissolving them in nitro-muriatic acid, and testing the solution by sulphuretted hydrogen. Rabbits were employed in all the experiments.

A.—Exp. 1.—20 grains of tarar emetic were dissolved in half-an-ounce of water, and injected into the throat of a rabbit, and swallowed. The animal died at the end of 15 minutes. The liver and other viscera, tested by Reinsch's and Marsh's processes, did *not* yield antimony, though the stomach did largely. The only part of the latter which exhibited any appearance of congestion was at the entrance of the œsophagus.

Exp. 2.—The same dose was similarly administered. The animal lived 20 minutes. The liver, lungs, blood, and kidneys, were similarly tested, and each yielded slight traces of antimony.

Exp. 3. — Same dose. Animal lived 2½ hours. Liver yielded antimony, both with Marsh's test and with sulphuretted hydrogen.

Exp. 4.—2½ grains of tartar emetic were swallowed. It lived two hours and a half. The liver, tested by sulphuretted hydrogen, after carbonization by sulphuric acid, gave an abundant precipitate of orange yellow sulphuret of antimony. The heart, lungs, and kidneys, treated with tartaric acid, digested, filtered, evaporated, and the residue dissolved in nitro-muriatic acid, gave distinct traces of antimony.

Exp. 5.—A large, strong rabbit swallowed from 10 to 12 grains of tartar emetic. At the end of three hours and a half it was alive and strong. It was then killed by dividing the jugular vein and the carotid artery on one side, and an ounce and a half of blood were thus obtained. This blood yielded antimony after the usual process of charring and filtration, both by the direct addition of sulphuretted hydrogen, and by Marsh's apparatus. The liver, similarly treated, also yielded orange yellow sulphuret of antimony.

B.—Exp. 1.—A rabbit swallowed two pills containing together 10 grains of Pulvis Antimonialis (Ph. L.) Two days afterwards it was killed, not having presented any symptoms of poisoning. The liver was treated as usual, and tested for antimony, and its presence there demonstrated. The heart, lungs, and kidneys also exhibited traces of the metal. This experiment was repeated with the same result, in my absence, by my friends, Mr. Furness and Mr. Zenner, of this town.

Exp. 2.—A strong, large rabbit swallowed, in two pills, nearly 10 grains of the true James's powder. Fifty hours afterwards, not having been at all injured by the medicine, it was killed by opening the cervical vessels, and an ounce and a half of blood obtained. This yielded abundant evidence of the presence in it of antimony, as did the liver and other viscera.

The same experiment was repeated in my absence by the gentlemen before-mentioned, and with the same results. It was observed that the quantity of antimony absorbed into the system was greater after the administration of James's powder than when the pulvis antimonialis was used. The former, therefore, would seem to be more soluble in the animal fluids; and hence, perhaps, its greater efficacy in some cases.

By the subjoined analysis of the three preparations of antimony, it will be seen that the quantity of the metal contained is nearly equal in all.

James's Powder (*Maclagan*).

Antimonite of Lime	3·40
Teroxide of Antimony	2·89
Antimonious Acid	43·47
Triphosphate of Lime	50·24

Antimonial Powder (*Maclagan*).

Antimonite of Lime	0·8
Teroxide of Antimony	3·98
Antimonious Acid	50·09
Triphosphate of Lime	45·13

Tartar Emetic (*Phillips*).

Teroxide of Antimony	43·35
Potash, Tartaric Acid	49·25
Water	7·40

In conclusion I may remark, that in proportion to the general diffusion of chemical knowledge will attempts at the destruction of life by chemical agency increase in frequency. Nor is it certain that advances in chemical science, though they may render the discovery of poisons more easy and sure, will always facilitate the conviction of the guilty. In my opinion, the prevention of the crime by judicious legislation is a much more feasible and hopeful mode of protecting human life. Had a proper machinery for medico-legal investigation existed in this country, and a public prosecutor been in action, I believe that evidence sufficient to convict the murderer of Mrs. Wooler might have been obtained. And had there been a proper check on the purity and sale of medicinal substances, we should not have heard of the recent poisoning of a number of children by the innocent administration of arsenical sulphur. By a more careful registration of the causes of death, and, as an essential preliminary to that end, by placing the machinery of registration in the hands of medical men—by requiring a license to be taken out for the sale of poisonous drugs — by punishing the adulteration of medicinal substances—by the abolition of the Government patronage of poisonous nostrums — by the appointment of public prosecutors and sanitary officers throughout the country—much may be done to check the existing tendency to this horrible crime, and so protect the helpless and the unsuspecting from the villainous designs of the brutal, the selfish, and the debased.

GATESHEAD: Printed at the *Observer* Steam Press, by D. DUNGLINSON.