substances, and thereby exerting its antiseptic action as needed.

Chloramine, being a highly reactive substance, should not be mixed with other antiseptics. Both alcohol and hydrogen peroxide are decomposed by it.

REFERENCES.

¹ Dakin, BRITISH MEDICAL JOURNAL, August 25th, 1915. ² Tuffier, BRITISH MEDICAL JOURNAL, November 27th, 1915, p. 790; also Carrel, Dakin, Daufresne, Dehelly and Dumas, *ibid.*, October 23rd, 1915, p. 609. ³ *Trans. Chem. Soc.*, 87, p. 153, 1905.

GASSING ACCIDENTS FROM THE FUMES OF
EXPLOSIVES.*

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CASES of gas poisoning account every year for a good many deaths in the mines of the Rand, and for a considerably larger number of cases of temporary disablement and loss of working time.

The irritant acid fumes, commonly known as nitrous fumes, consist of a mixture of oxides of nitrogen. They belong to the group of respiratory irritant poisons in which chlorine, the vapours of bromine and ammonia, sulphuretted hydrogen and sulphurous acid, are also included. All of these cause, when inhaled, a somewhat similar train of symptoms. Fatal poisoning by nitrous fumes has occurred when persons have inhaled the acid fumes from a vessel containing nitric acid, which has been accidentally broken. In mines, poisoning from this source only occurs after the use of explosives in cases where the detonation has been imperfect, and particularly where burning of explosives has taken place. Nitric oxide (NO) is first formed, but this gas is rapidly oxidized, and nitrous fumes consist chiefly of nitrogen peroxide¹ (NO₂). This again quickly reacts with water vapour to form nitrous acid. What is actually inhaled is mainly a mixture of nitrous and nitric acids. Nitrous fumes are the red irritant fumes with the smell of which most miners are familiar. The smell is that of fuming nitric acid. The gas is intensely poisonous, and of all mine gases it is the most treacherous. We do not know the downward limit of its toxicity. Exposure to as little as 0.05 per cent. of nitric oxide for half an hour was found by Haldane to cause death in mice after about twenty-four hours, with the typical sequence of symptoms, and the symptoms were the same whether nitric oxide or the fumes of burning dynamite were added to the air. It was found that with small percentages of nitrous fumes the haemoglobin was normal at the time of death. With higher percentages (and more rapid death) the blood was found to be more or less chocolate coloured, and to contain methaemoglobin. Under the latter circumstances nitrous fumes act like carbon monoxide by disabling the haemoglobin, and may in concentration produce rapid death. Where the exposure is to small amounts death is due solely to the lung inflammation and its immediate consequences.² It is with this latter form of poisoning that we are locally familiar. Half of the amount mentioned is the highest figure (0.026 per cent. reckoned as NO₂) found by Mann in mine air after blasting,³ but the presence of the gas in poisonous amounts is probably not so very uncommon in ordinary blasting practice. I shall give some evidence for this statement later. What is certain is that quite a brief exposure to small quantities of nitrous fumes is sufficient to produce serious and even fatal poisoning. Air which contains enough nitrous fumes to cause feelings of irritation in the nose or air passages is very dangerous.

Dr. D. Macaulay was the first to direct attention to the important part played by nitrous fumes poisoning in gassing accidents on the Rand, and to give an adequate account of its symptoms and treatment.⁴

SYMPTOMS.

The symptoms of poisoning by nitrous fumes are very characteristic. At the moment of exposure they are slight. A man who inhales nitrous fumes has a sense of

irritation in the nose and throat, and of constriction, and perhaps pain, in the chest. There is headache, smarting of the eyes, and there is coughing. The latter is a characteristic sign. But these immediate irritant effects may not be severe, and commonly pass off altogether in a short time. The man may feel quite well and may continue work. He leaves the mine, has his supper perhaps, and goes to his room. Then, in from perhaps four to eight hours afterwards (very rarely later), acute symptoms suddenly come on, and progress with alarming rapidity. In a typical severe case there is marked and increasing distress in breathing, with coughing, and often severe pain in the chest. The cough is at first dry, and auscultation may at this stage reveal no moist sounds. But this condition is speedily followed by the expectoration of a copious frothy, rather fluid, blood-stained spit. The lungs become waterlogged, and auscultation now reveals copious moist sounds. There is cyanosis and marked dyspnoea and distress, followed, unless the case is promptly treated, and very often in spite of all treatment, by collapse, unconsciousness, and death *within a few hours*. Dr. Macaulay states that "the temperature is at first normal or subnormal, but, if the gas goes on for several hours, a rise takes place." I well remember the first case of this condition which I saw, now more than ten years ago. A miner was taken ill suddenly in his room on one of the mines at 11.30 p.m. I saw him at 1 a.m. He was cyanosed and in much distress. His lungs were full of moist sounds and one base was dull. The wooden floor of his room was covered with a blood-stained fluid expectoration. He died at 5 a.m. I could not reconcile his condition with that of any form of acute pneumonia which I have seen, but it agreed at every point with the published descriptions of nitrous fumes poisoning. It is probable, I think, that a certain number of cases returned as "pneumonia" amongst underground workers are really cases of nitrous fumes poisoning.

The typical sequence of symptoms in nitrous fumes poisoning is therefore this:

1. *Initial symptoms* of irritation occurring at the moment of exposure and usually comparatively slight. Nitrous fumes never, in our experience of ordinary gassing accidents, produce partial or complete unconsciousness at the time of exposure, as does carbon monoxide, although they may do so if present in massive amounts, and cases of rapid death from the concentrated fumes of burning explosives may be partly due to this cause.

2. *A latent period of several hours' duration*, during which the patient may and commonly does feel quite well.

3. *The sudden onset after that interval of acute symptoms*, due to a rapidly progressive inflammatory oedema of the lungs. The appearance of this well marked symptom sequence is of great diagnostic significance.

The *post-mortem* signs are also definite. There is an intense injection of the trachea and bronchi, which is characteristic. The lungs are intensely oedematous, often enlarged and tense with oedema. A copious frothy, blood-stained fluid exudes from them on section, and may be seen to occupy the air passages. There may be patches of incomplete consolidation, subpleural haemorrhages may be observed, and blood-stained fluid in the pleural cavities or the pericardium. The right heart and the great thoracic and abdominal veins are engorged with dark, thick, sometimes almost tarry, blood. The abdominal veins especially are often very greatly distended.⁴

There is one other point. Miners from time to time complain that after slight exposure to blasting fumes—perhaps from blasting at some distance from their working place—they are attacked the same night with difficulty of breathing, and perhaps slight spitting of blood, which pass off in a few hours. The affected man thinks he has had an attack of "asthma" or "bronchitis." He has really had a slight attack of nitrous fumes poisoning. The most sedulous care must be taken that, if any casual blasting has to take place before the end of the shift, the water blast shall be invariably employed immediately after, and that the fumes are thoroughly dispersed. Precautions must be taken, also, in the general blasting at the end of the shift, in order to obviate the risk of inhalation of drifting fumes of this dangerous gas by men waiting on the stations before being "hauled" up the shaft.⁵

Finally, I may state that the intensely irritating character of nitrous fumes has suggested that the repeated

* Extracts from a paper on accidents from poisonous or asphyxiating gases in mines, dealing generally with the subject, and also with the organization of the use of rescue appliances in mines, read before the Witwatersrand Branch of the British Medical Association, and published in full in the *Medical Journal of South Africa*.

inhalation of small quantities of this gas may, by maintaining a catarrhal condition of the air passages and lungs, be a contributory factor in the development of miner's phthisis.

The accidents under review fall into two groups: A. Those due to exposure to the fumes of explosives after blasting—the ordinary "gassing" accidents. B. Those due to explosives being accidentally set on fire and burned.

A. ACCIDENTS CAUSED BY THE FUMES OF EXPLOSIVES AFTER BLASTING.

I found it desirable to subdivide this group into two classes:

1. Cases in which the Exposure was Immediately or Shortly After Blasting.

These accidents occurred when persons returned too soon to the working places after blasting, or where they were affected by the fumes of explosives arising from blasting taking place in other parts of the workings. I found that in such cases either carbon monoxide or nitrous fumes poisoning might result, but that the latter type was considerably more common.

Whether the one type of poisoning or the other occurs under these circumstances is due in part to the character of the explosion, since if definite partial burning of the explosive takes place the risk of nitrous fumes poisoning is especially great. But the result will depend also on the duration of exposure. Unless carbon monoxide is present in large amounts, it takes time to produce its effects. On the other hand, the records abundantly show that quite a brief exposure to even small quantities of nitrous fumes is sufficient to produce serious or fatal poisoning from this source.

For the same reason drifting fumes from blasting will typically cause nitrous fumes poisoning, and persons may be "gassed" in this way by the fumes from blasting which has taken place at some considerable distance. Sometimes one or more of a party working in close proximity may be affected, while others escape.

Mixed poisoning must sometimes occur, but the number in which the evidence points to the probability of such a conjunction is surprisingly small. One or other type is almost invariably markedly predominant. I know of no case in which a man who had been rendered unconscious by carbon monoxide and had then recovered, developed later on signs of nitrous fumes poisoning. On the other hand, it is probable that carbon monoxide may co-operate in some cases of nitrous fumes poisoning in aggravating and prolonging the initial symptoms.

Another interesting point is this: It appears from the evidence that the danger from nitrous fumes exists only for a comparatively short time, perhaps an hour or so, after blasting. The longest interval observed between the occurrence of blasting and the occurrence of exposure, in this type of poisoning, was two hours, and this was exceptional. This circumstance, I would suggest, is probably due to the fact that nitrous fumes are very soluble in water, and that mine air is always highly charged with moisture, and also to the circumstance that these fumes are not often present in large quantity after blasting.

Let me now quote a few typical instances of accidents of this sort. These will suffice to indicate the circumstances in which these emergencies occur, and the general nature of the evidence on which I have relied. Some are drawn from the cases originally investigated, some were of more recent occurrence.*

CASE I.—Exposure Immediately After Blasting: Poisoning by Nitrous Fumes: Three Deaths.

The following interesting case was described by Dr. D. Macaulay. It was amongst the first in which the part played by nitrous fumes poisoning in gassing accidents was clearly recognized.

Two white men and three natives were working at the end of a drive, with a crosscut leading off it about a hundred feet from the face. The miners in the crosscut blasted at 3 p.m., before the party in the drive had left, and without giving them warning. The latter were imprisoned in the end of the drive for three-quarters of an hour. They felt that they were being gassed and turned on the compressed air. When the smoke had cleared they left the drive and went to the surface. The white men washed, changed, had their evening meal, and went

to their quarters. Acute symptoms of respiratory distress came on in both at 10.30 p.m., and when seen by Dr. Macaulay at 11 p.m. they were so ill that doubt was entertained by him whether it was possible to move them to the mine hospital a few hundred yards away. Both eventually recovered under energetic treatment. Nothing was reported at the time regarding the natives. They went as usual to the compound and did not report sick. Next morning one was found to be dead and another moribund. The third was very seriously ill, and he also eventually succumbed.

A post-mortem examination on these natives, made by Dr. Macaulay, at which I had the opportunity of being present, showed the typical signs of acute inflammatory oedema of the lungs, and great engorgement of the heart and venous system with dark blood.

CASE II.—Exposure Immediately After Blasting: Poisoning predominantly by Nitrous Fumes: One Death.

A white miner blasted the cut in the face of a drive. He did not use the water blast. He then returned with two natives to charge and blast the round, turning on the compressed air when doing so. As three holes of the round did not explode (there was therefore probably imperfect detonation and partial burning), he returned a second time to refire them. The natives stated that both times they returned "the smoke was too thick," and one failed to reach the face the second time. The white man did not say anything, but they saw that he was affected by the smoke. All left the mine together, apparently at 8 o'clock or a little after. The white miner felt ill on arriving at the surface, and waited for some hours in the change house. Afterwards he set out to walk home, then he telephoned for a cab. He walked from the cab into his house, reaching the latter after midnight. The medical officer of the mine was sent for to see him at 1.30 a.m., and found him "suffering from the effects of gassing usually ascribed to nitrous fumes." The man was too ill to be moved to hospital. He died at 2 p.m. the following day.

This case is an amazing example of a man throwing away his life, through sheer recklessness and contempt of the regulations which govern, or should govern, blasting operations.

In this case the prolonged character of the initial symptoms suggest a mixed poisoning, and the circumstances of the accident render this probable. The later symptoms were typical of nitrous fumes poisoning.

Post mortem the lungs were found to be tense ("ballooned") and oedematous.

The district surgeon, Dr. T. B. Gilchrist, states that the blood was somewhat chocolate coloured, but that after an hour's exposure to the air it became more red. No carboxyhaemoglobin was found in the blood.

2. Exposure Delayed until Several Hours After Blasting.

These cases occurred when the working places were entered on the following shift. They were caused by the stagnation of blasting fumes in close places owing to defective local ventilation, due either to the miner responsible having neglected to use the means of ventilation provided, or less commonly to the fact that the means of ventilation provided were inadequate. In every instance in this class carbon monoxide was the predominant cause of death, no doubt with carbon dioxide co-operating in some cases. Nitrous fumes poisoning never occurred under these conditions. It is only caused when the exposure occurs immediately or shortly after blasting, or after the actual burning of explosives.

B. ACCIDENTS DUE TO EXPLOSIVES BEING ACCIDENTALLY SET ON FIRE AND BURNED.

The number of observations which have been made on the composition of the fumes actually evolved when nitro-glycerine explosives are burned in air is not large. Recorded results vary considerably, and the actual result which occurs when accidents from this cause happen underground also probably varies between rather wide limits. Dr. James Moir considers that, if the supply of air locally available in places where explosives are burned is sufficient to allow of vigorous combustion, the amount of carbon monoxide generated may not be greater, and may be less, than when the explosive is detonated. On the other hand, if the supply of air is insufficient, as may happen in a confined space, the amount of carbon monoxide will be increased in direct proportion to the degree of deficiency of air, and that of carbon dioxide correspondingly diminished.

In either case, however, but especially in the latter circumstances, large quantities of nitrous fumes will be evolved, within limits, varying according to the particular conditions present, from 1 to 5 cub. ft. per lb. of explosive burned, blasting gelatine being taken as the type. The latter figure agrees with that of Cullen and Greig.⁸

* I have to thank Mr. R. N. Kotzé, Government mining engineer, for permission to publish these illustrative cases.

Dr. Moir has supplied me with the following figures, which represent the behaviour of blasting gelatine in the several conditions specified in the table:

Table showing Quantity in Cubic Feet of Gases produced by 1 lb. of Blasting Gelatine under Different Circumstances.

Conditions.	CO ₂	N ₂	CO	NO
A. Perfect explosion, without wrapper ...	6.2	2.9	—	—
B. "Practical" explosion, with wrapper ...	5.5	2.9	0.9	?
C. Burned without wrapper in excess of air ...	5.7	2.3	0.7	1.2
D. Burned in vacuo	—	—	6.0	5.0

When burning takes place in the dead end of a drive or similar place combustion might be expected to begin as in C, and to finish as in D, according to the available air. The calculations are for pressure and temperature at a depth of 1,500 feet.

These being the determining conditions, one might conclude that nitrous fumes poisoning would be the type of gassing most frequently met with when explosives are set on fire underground, but that carbon monoxide poisoning may also occur. Experience shows that this is actually so, but a rather closer description of what happens is perhaps desirable.

1. When large quantities of explosives, say from 50 to 100 lb., are set on fire, their ignition is frequently followed by their explosion, and this fact introduces an important complication. I have therefore subdivided this group of accidents into two classes, according as explosion did or did not follow ignition. The former class includes one appalling disaster which happened on the Rand some years ago. Happily no accident of similar magnitude has occurred since.

CASE III.—Ignition of 100 lb. of Blasting Gelatine, followed by Explosion: Sixty-one Deaths.

In this case a miner, apparently, so far as the evidence goes, through sheer carelessness, set fire to the explosives in his explosives box at a shaft station. Three other boxes were near by, the four containing perhaps 100 lb. of gelatine, and the whole quantity was involved in the violent explosion which almost immediately followed. The station on which it occurred was completely wrecked, and much damage was caused for a considerable distance up and down the shaft. A large number of natives and some white men were in or near the shaft at the time waiting to ascend to the surface. Three whites and many natives were killed outright or succumbed before the rescue parties could reach them, perhaps an hour later. Many of the bodies were severely burnt; some had sustained other gross physical injuries. In those out of this number who survived the immediate effects of the concussion, shock, and burning caused by the explosion, carbon monoxide poisoning was probably the main cause of rapid death. Other whites and natives died later on from the after-effects of fumes, shock, and burning, but, in the absence of fuller information, one cannot push the analysis further.

I have therefore included the whole number under the heading "Indefinite." The eventual toll of deaths was six whites and fifty-five natives. Many members of the rescue parties were temporarily overcome by the fumes, but fortunately none died.

In a smaller accident of the same character perhaps 30 lb. of gelatine was set on fire and exploded, and involved in the explosion a similar quantity of gelignite 20 ft. away. A 6-gallon tin of paraffin was also ignited. Five coolies died later from the burning and battering of the explosion. They were not "gassed," as the fumes did not reach them. But a white miner who encountered the fumes 1,200 ft. away died the same night from acute oedema of the lungs.

2. If we eliminate accidents complicated in this manner, the preponderance of nitrous fumes poisoning in accidents due to burning of explosives becomes at once apparent. Out of fifteen such accidents, carbon monoxide (or mixed) poisoning was the cause of fifteen deaths in one (Case IV), and of one death in a second accident, although in this case other individuals were affected by nitrous fumes. In the other thirteen all the deaths were due to nitrous fumes poisoning.

What appears to happen is this: If the amount of explosives burned is large, and the fumes are met with in great concentration near the site of burning, a rapidly fatal massive poisoning results, which is certainly mixed, but in which carbon monoxide is probably the predominant factor. Or in these circumstances, and in the same acci-

dent, out of a number of persons in close proximity to each other some may be rapidly killed in the way described, while others die later with the typical signs of nitrous fumes poisoning. On the other hand, when the fumes have become dispersed, and therefore less concentrated, nitrous fumes poisoning will practically invariably follow. Each one of these results is well illustrated in Case V, cited below, and the first is clearly shown in Case IV.

The larger accidents of this group quite overshadow those due to poisoning from blasting fumes, since in the former large areas of the mine are quickly flooded with poisonous gases, and many deaths may result. Fortunately the greater number of accidents due to burning explosives are of a less tragic nature. These have occurred from smaller quantities of explosives being accidentally set on fire in the workings through carelessness or misadventure. The insatiable curiosity of natives to find out what really happens when gelatine burns has been the cause of not a few.

In such cases carbon monoxide poisoning may also occasionally result if the fumes are sufficiently concentrated and exposure sufficiently prolonged. Usually, however, when explosives are set on fire there is a hasty stampede to a place of safety. But nevertheless, let me repeat, a very brief exposure to the fumes of burning gelatine or gelignite, even although it be only a stick or half a stick, is quite enough to cause fatal poisoning by nitrous fumes. Nitrous fumes poisoning is therefore the typical and characteristic form of "gassing" under these circumstances. Several deaths have occurred from the inhalation of these fumes in lighting up with the old-fashioned "cheesa" stick, made of strips of blasting gelatine. The use of lighting sticks of this sort has as a consequence been prohibited for some time.

I shall quote three further cases of accidents of this group, each of which illustrates one or other of the points I have mentioned.

CASE IV.—Accidental Ignition of Gelatine Dynamite (15-20 lb.): Carbon Monoxide (or mixed) Poisoning: Fifteen Deaths.

A gang of thirty natives under a white miner were proceeding to their working place in a stope. The natives arrived first; some descended the stope; the others, fourteen in number, remained on the level above, in which, between them and the station, was the miner's explosives box. The miner followed, and had reached his box and passed it, when immediately an alarm was raised. Fifteen, perhaps twenty, pounds of gelatine dynamite contained in the box had become ignited, apparently, so far as the facts are obtainable, through the carelessness of the miner. The boys on the level rushed towards the station, right into the fumes, and all collapsed at once and dropped within a short distance of the box, where their bodies and that of the miner were found. The natives in the stope below the level escaped. Exposure was thus immediate, and apparently death almost equally so.

The blood from three of these cases was examined in the Government Laboratory. In each case it was cherry-red in colour and uncoagulated. It contained a high percentage of carbon monoxide, and the chemical evidence was stated to point to an "oxide of nitro-gen" as a contributory cause. Clearly here the proportion of these gases in the air at the moment, no doubt in conjunction with carbon dioxide also, was high enough to cause immediate unconsciousness and rapid death, and the case may be regarded as one of mixed poisoning, although carbon monoxide was probably the preponderant cause.

CASE V.—Burning of 50 lb. of Blasting Gelatine: Six Deaths from Carbon Monoxide (or mixed) Poisoning; Nineteen from Nitrous Fumes Poisoning; One from Pneumonia.

A white miner, S., was seen at 7.45 a.m., sitting beside his explosives box, which was at the dead end of a short crosscut communicating with a main level (14) and air-way. The explosives box was open, and he had a lamp and candle in dangerous proximity to it. One native witness stated that the candle was actually stuck on the corner of the open box.

A few minutes afterwards a flame was seen in the place. The miner ran out, and having told his own natives who were working near by to turn on the air into their working places, as there was gas about, he went away, and warned no one else. He afterwards denied all knowledge of how his explosives had gone on fire, but was eventually convicted before a magistrate and sharply sentenced.

Several witnesses say that they heard a blast, which may have been due to the explosion of part of the burning gelatine. There was certainly, however, no violent explosion. The fumes poured into the level and were carried by a strong ventilating current up the nearest stope, 70 ft. away, and so to the level above (13). There were natives and a white man (who afterwards died) working in this stope. This man at once ordered his natives out. Six were afterwards found dead or dying in the level above, having been overcome in the attempt to escape. Nineteen other natives died later on in the

mine hospital, and fifteen of these had been working in this same stope. The fumes spread for a considerable distance along Level 13, 600 ft. or 700 ft. in either direction, and ascended to Level 12 through several stopes in which many natives and some white men were working. Many of these were affected.

The medical record of these cases is of great interest. For it I am indebted to Dr. A. H. Watt, of the Simmer and Jack Hospital, who saw and treated them.

The six natives found dead were exposed to the fumes in great concentration, being nearest to the original seat of burning. Death in their case was presumably due to mixed poisoning from carbon monoxide and nitrous fumes.

Fifteen other natives from the same stope and four from other working places died in the mine hospital later with symptoms of nitrous fumes poisoning. Eight of these were dead or moribund on admission. They had not been overcome by the fumes at the time of exposure, and had been withdrawn from the mine in the ordinary way. In fact, during the day and through the evening large numbers of natives were admitted to the mine hospital suffering from respiratory symptoms. Many of these had been working in the stopes between 13 and 12 levels, where the fumes had travelled a greater distance before reaching them. They were recognized as suffering from nitrous fumes poisoning, and in the bad cases free bleeding was carried out, followed by respiratory stimulants. Dr. J. B. Tough informs me that in six natives whom he found comatose with a thin froth issuing continually from their mouths and who were freely bled, the colour of the blood changed during the operation from a dark asphyxial colour to that of oxygenated blood. All six recovered. Excluding those dead or moribund on admission the number treated was sixty-seven. One native died ten days later from pneumonia.

Fourteen white men were also affected by the fumes. What happened to them may be best described in Dr. Watt's own words:⁶

"Thirteen of them" (who, under his orders, came to hospital and were detained there overnight) "had an emetic, and the next day were all out of danger. One man disobeyed orders, and went home without receiving an emetic. He was sent for, but he had gone out for a stroll after having partaken of a hearty luncheon. He was found four hours after the accident, and came to hospital under protest, stating that he felt quite well. In the evening he developed general oedema and congestion of his lungs, and died before morning." This was regrettable, as this particular man had been active in getting the boys out, and had inhaled the gas in so doing.

Unfortunately, the *post-mortem* evidence in this case is incomplete. Not all of the bodies were dissected, as this was unnecessary for the immediate purpose of the district surgeon. Dr. Moller informs me that those which were examined did not show any signs of carbon monoxide poisoning. The blood was dark and the lungs very oedematous.

Pure carelessness on the part of three white miners was responsible in these two instances, and in the shaft accident already mentioned, for over one hundred deaths. If the regulations regarding the handling of explosives had been observed not one of them would have occurred. The careless miner (we have met with him rather frequently in these records) is a standing danger, not only to himself but to every one near him. Recklessness such as this is not fair play. But these accidents suggest that it might be well to provide all those engaged in the actual handling of explosives with approved (and tested) electric lamps for use in so doing.

I should like to mention, in a word, another occurrence, in which 100 lb. of gelatine was accidentally set on fire by a white learner when capping fuses. He himself and three natives died—the natives certainly from nitrous fumes poisoning. But, although the fumes spread for considerable distances through the mine, no one else died, owing to the prompt and resourceful action of the mine officials. Being immediately warned of what had happened, they at once withdrew all the workmen from the portions of the mine affected to fresh air. This is obviously the only thing to do. Every one should at once get by the nearest way out of the air current bearing the fumes. Rescuers also should be careful to approach places where burning has occurred only from the windward side. The familiar wet handkerchief over the mouth should not be omitted. Several men have saved themselves, when cut off from safety by the fumes, by retreating into a dead end and turning the air on at the face. Sometimes, however, this has not sufficed to protect them from nitrous fumes.

When small quantities of explosives are set on fire, every one should leave the place at once until it has been thoroughly blown out and wetted down. Neglect to do so was the cause of the following accident, the last which I shall quote. In this instance a much smaller quantity of explosive was ignited, and nitrous fumes poisoning followed. Observe once more in this instance the almost

clockwork precision with which this poison does its work. Compare it in this respect with Case I.

CASE VI.—Accidental Ignition of 5 lb. of Gelatine: Nitrous Fumes Poisoning: Four Deaths.

About 1.20 p.m. on a Saturday a miner (G.) accidentally set fire to half a packet (about 5 lb.) of gelatine in a stope. Another miner (H.) and his natives were working at the top of the stope. The fumes ascended the stope to the level above, through part of which men and boys had to pass. G. withdrew his boys and immediately warned H., who left the stope with his natives, and waited with them not far away in the level above for a short time. He then insisted (about 2 p.m.) on returning to charge his holes, although warned not to do so, and took with him a native. He was followed by G. They finished their work and all left the mine together afterwards. H. said he did not feel sick, and both men took the tram home.

Five natives were affected by the fumes. All went to the compound, but about 7 p.m. two became very ill and were taken to the mine hospital. They were spitting blood. One died in a quarter of an hour, the other the following day.

Just at the same time (8 p.m.) the mine medical officer was called to see H., and found him sitting on the side of his bed, with a pulse of 120, expectorating blood-stained sputum. There were signs resembling those of commencing pneumonia at one base. The medical officer made arrangements to send him to hospital the following morning. At 3 a.m. he was dead, thirteen hours after exposure to the fumes.

Two other natives were taken to the hospital at 3 a.m. One died the following day; the other recovered.

The *post-mortem* notes on these four cases were kindly supplied to me by Dr. Stanley Heberden, district surgeon, who certified all as having died from nitrous fumes poisoning. All four reports are couched in almost identical terms. A summary of two will suffice.

Native S. (the boy who died within six hours after exposure). No disease of internal organs. Air passages full of froth; mucous membrane congested; lungs congested and oedematous. Right side of heart engorged with blood. The blood was dark and asphyxial in character. There was no alteration in colour suggesting the presence of carbon monoxide.

R. H. The body of a well-nourished European male. No injuries. The mucous membrane of the air passages is congested and the tubes are full of froth. The lungs are congested and tense with oedema. The right side of the heart is engorged with blood. The blood is dark (asphyxial), and there is no alteration in colour suggesting the presence of carbon monoxide.

MEDICAL TREATMENT.

Regarding the medical treatment of nitrous fumes poisoning there has been a good deal of local experience.

In all cases in the early stage, first produce emesis as soon as possible (apomorphine hypodermically). Follow this by stimulant, ammonia, or (preferably) pituitrin. Then send the case straight to hospital.

Atropine may prove useful in the latent stage.

In the acute stage with developing oedema of the lungs, Macaulay⁴ recommends as the first step wet cupping of the chest, back and front. This invariably relieves the symptomatic distress. It should be followed locally by poulticing, or better by jacket mustard-water fomentation, applied very hot and changed every hour.

In all recognized cases of acute oedema free blood-letting should next be performed. This is a most valuable remedial measure. In one of Macaulay's cases, after venesection, the haemorrhagic sputum speedily ceased, and never returned, and the patient made an uninterrupted recovery. Dr. Tough's experience, already quoted, also clearly shows the value of this measure. This has been confirmed by other observers. The condition of the blood may render bleeding difficult, but it should be free (12 to 20 oz.). Venesection should not be delayed until the condition becomes grave. It should be undertaken immediately on the recognition of the nature of the case.

Follow venesection by saline infusion, and repeat this if necessary.

Administer oxygen intermittently to relieve and tide over the asphyxial condition.

Give ammonia in repeated doses.

Injections of pituitrin (1 c.cm.), repeated every four hours, should be tried. I have found this drug, used in this way, of great service in the treatment of acute pneumonia.

These are the lines of treatment which have been followed locally, and in a considerable number of cases with marked success, although when the condition of acute oedema is well established probably a majority of cases will die in spite of treatment.

I had written the above notes before I had an opportunity of seeing the very valuable report by Lieutenants Elliot Black,

Glenny, and McNee, R.A.M.C., on Cases of Poisoning by Noxious Gases used by the Enemy.⁷

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NOTE ON CASES OF PHLEBOTOMUS FEVER AT AN ISLAND IN THE EASTERN MEDITERRANEAN.

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EIGHT cases of phlebotomus fever occurred amongst the officers and men attached to the garrison under my charge. These were due to infection by *Phlebotomus papatasi*. Seven cases occurred between July 21st and August 1st, 1915, and one other case on August 9th.

Six of these cases occurred in one locality and in one house. The patients were either mess-servants, sentries, or officers (1) who were on duty at and slept in this house. One case was an officer's servant (Private R.M.L.I.). He came in from another camp, and slept only one night at the house. On the morning of the fourth day afterwards he became ill with the fever. All the other patients had numerous marks of bites. They resembled mosquito bites; but the sandfly bites silently; it does not give warning of its presence by any peculiar musical note, as does the mosquito. A very faint note may sometimes be heard if the fly passes close to one's ear. The sudden sharp sting, when the phlebotomus bites, is sufficient to wake one from sound sleep.

After the first case, on account of the type, sandfly infection was suspected; the suspicion became greater when the localized incidence at the house became marked. A search was made for phlebotomi in the house, *Culex* larvae (? dengue) being searched for at the same time in the wells, etc., as the marks on patients' wrists and ankles closely resembled mosquito bites. No *Culex* larvae were found, but phlebotomi were found in the house, and later inside the mosquito net of an officer, who had been severely bitten and developed the fever. These were caught and proved to be *Phlebotomus papatasi*. Although specimens had been seen at the Royal Naval College, Greenwich, during the tropical course, after entry, I was not certain that this was the genuine sandfly, but the diagnosis was confirmed by Colonel Garnier, R.A.M.C., Egyptian Medical Service, and they were later examined in company with Dr. A. De Lisle, attached to the French Army Medical Service.

The following prophylactic measures were taken:

1. The sleeping place of mess-servants and sentries was changed from the hall on the ground floor to the lobby on the next floor higher up.

2. A space of ground alongside the house, but not part of the premises, contained old rubble, refuse, and fallen masonry, and was thought to be the probable breeding place and habitat of the fly. This was cleared and covered with lime; quicklime was applied for in order to spray or wash down all these old walls and crevices, but was not immediately available.

After these measures were taken no fresh case occurred, but this was probably in part due to the fact that a slight change in the weather followed, and the temperature dropped below 70° in the evenings, which is unfavourable to the continued presence of phlebotomi. The weather during June and early July was probably too hot and dry for the larvae to hatch out.

As was gathered subsequently from articles published in the *BRITISH MEDICAL JOURNAL*, and seen by me in September, prophylactic measure No. 1 above was probably more effective in the circumstances than was hoped for at the time. In the place where the men previously slept they were exposed to the flies from the

rubble, etc., which was only a few yards away across a narrow by-way, and level with the open windows of the hall, so that the flies entered each evening, probably attracted first by the light inside. As the phlebotomus does not tend to ascend much in flight these flies probably did not reach the second lobby, where the men afterwards slept. Although they remained in some of the rooms during the day this lobby was too bright to suit them, and there was always a free draught there.

The case which occurred on August 9th, 1915, was that of a ship's steward (pensioner, aged 43). This case occurred at another building, an old castle. His office and store were in a part of the castle suitable for the fly, that is, inside one of the old stone turrets. This building was not used by the men.

The flies were of a dull pale yellow or sand colour. Two dots of black pigment at the eyes were conspicuous when the fly was caught. The body was curved on itself in hump-backed fashion. The wings were long and narrow, and with a magnifying glass were seen to have long fine hairs along their edges. The wings (at rest) were always carried pointed backwards, upwards, and outwards from the body, diverging from each other. When an attempt was made to capture a fly it did not fly forward, but eluded capture by seeming to jump off at a right angle, alighting again about six or eight inches away in a straight line to right or left.

GENERAL DESCRIPTION OF THE CASES.

Incubation.

From the case of the private (R.M.L.I.), and from consideration of the time of onset after first noticing the bites, this appears to have been about three days.

Symptoms.

The onset is sudden; the patient's face is flushed and eyes puffy and injected as if he had been drinking heavily. He complains of dizziness, severe headache, and pains, generally in the back and region of the hip-joints. He is "tired" and feels very ill. In two of the cases there was nausea and slight vomiting at the onset. The temperature was generally about 101° at the onset, but usually reached 103° or more within ten hours. On the second day it was usually about 101°. In three of the cases it eased to normal on the evening of the second day. In the others it did not reach normal until the evening of the third day or later. After becoming normal, in several cases a rise next day to 99° or 99.4° was noted, the temperature afterwards steadying at normal.

During the night of the first day and subsequently, pain in the back, sacral region, and joints was marked, and also pain across the eyes; the eyeballs are rather tender to pressure. The patient slept little and did not sweat. By the second day the tongue was covered with a white fur, except at the tip, and the patient had no appetite. One patient had slight diarrhoea, but the others were constipated. The pulse-rate was never more than 80 in any of the cases, even when fever was high. In one case there was slight sore throat, and in another slight stupor and delirium on the third night.

After Effects.

A general feeling of fatigue and lack of energy, mental and bodily, lasted for some time. All cases showed this to some extent, but in three it was much less than in the others; these three cases were "two-day" cases. The others remained unfit for duty for a week or longer. The pains and feeling of stiffness sometimes remained. Although the patients said they felt well, they walked about shakily, and as if shrunken inside their uniforms. Their voices were noticed to be weak and rather higher pitched than normally.

Treatment.

During the attack acetyl-salicylic acid was freely given. Quinine was given to the first two cases, but discontinued when it was seen that they were not due to malaria and that quinine did not appear very effective. During the after-period mist. Fowleri was given for about a week.

In his article on phlebotomus fever at Peshawar, published in the *BRITISH MEDICAL JOURNAL* of July 31st, 1915, Captain Houston, M.B., R.A.M.C., remarks on the tendency of the infection to "cling more particularly to certain bungalows." It is interesting to note that the infection met with above was almost exclusively experienced at one particular house. Also, the detailed corre-