tion showed that the lung and other wounds were firmly closed, and death was attributed to "secondary shock." In another case the diaphragm, spleen, and right ventricle were injured, the lung being intact. Suture of the ventricle and of the diaphragm was done and primary union occurred.

For comparison 9 cases of penetrating wounds of the chest were treated conservatively. They were not operated upon, either because they came in after twelve hours or because they refused operation. Their average stay in hospital was $11\frac{1}{2}$ days, as against $16\frac{1}{2}$ days for the operated cases, excluding two cases in which outside factors influenced their stay. The comparison is not absolutely fair because only the less serious cases are included in the second group; the serious cases are admitted sooner and do not refuse operative treatment.

Summary and Conclusions

17 consecutive cases of penetrating stab wounds of the chest were treated by operation. There was 1 death.

The average stay in hospital of 14 of these was $16\frac{1}{2}$ days.

A series of 9 less serious similar stabs treated conservatively had an average of $11\frac{1}{2}$ days.

The general impression obtained is that operation is worth while. The cases are much less worrying post-operatively: aspirations and repeated pulse observations are not required. All that is necessary is for a nurse to walk down the ward and look at the bottles under the beds. The patients move about freely in their beds, and after the drainage tubes are removed they do not complain of any pain. There is much less pain than after a laparotomy, and we have not come across any of the post-operative complications that occur in the latter.

I would like to express my thanks to my chief, Mr. W. Lennox Gordon, F.R.C.S., for his courtesy in allowing me the use of his beds for treating these cases and for his kindly interest in the work.

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ATROPINE POISONING

DESCRIPTION OF AN UNUSUAL CASE

BY

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Cases of atropine poisoning either with unusual features or occurring in epidemic form were frequently reported in the literature up to 1921. Since then very few descriptions of this condition have appeared, either because it is occurring less often or because cases of unusual interest are fewer. The following case presents sufficient uncommon features to merit a brief report.

Case History

The patient, a previously healthy man aged 73, was admitted to the Central Middlesex County Hospital at 8.30 p.m. on February 4, 1940. The story was that at 11 o'clock the previous night he had reached up to a shelf for a bottle of cough mixture, and by mistake had taken down a 1-oz. bottle full of linimentum belladonnae. This he emptied of all but three drachms at a single draught. He noticed an unusual taste and a burning sensation in his mouth, and realized his mistake. He was not unduly perturbed by this and got into bed and went to sleep immediately. At 7.30 the next morning his wife was awakened by his convulsions in bed, and, becoming alarmed, she summoned a doctor, who sent the

patient into hospital at once. No vomiting had occurred and no treatment had been given.

On admission, ten hours after taking the liniment, the patient was unconscious, with flushed face, stertorous breathing, and slight excoriations on the lips. His tongue and mouth were dry and leathery. The pupils were fully dilated and without reaction to light. All his limbs showed increased resistance to passive movement, and frequent convulsive movements of legs and arms occurred equally on both sides. Occasionally there was bilateral twitching of the lower face. All the tendon reflexes were brisk and equal, and an extensor plantar response was obtained on the left side. The corneal reflex was present, but the conjunctival reflex was absent. The temperature was 99.6° and the respirations 26. The cardiovascular system showed a pulse of 120, irregular, with extrasystoles. The blood pressure was 135/90. Heart sounds were poorly heard. The bladder was distended and 35 oz. of urine was subsequently withdrawn.

Morphine, $\frac{1}{4}$ grain, was given immediately, with physostigmine, $\frac{1}{50}$ grain. The stomach was empty, but free lavage with potassium permanganate 1 in 5,000 was carried out and half a pint of black coffee was left behind. The convulsive movements continued and the patient's condition was unchanged an hour later, except that restlessness was, if anything, increasing. Lumbar puncture was performed at 11.30 a.m. and 30 c.cm. of clear fluid withdrawn, the pressure falling from 200 mm. of water to 70. Shortly after this it was possible to rouse the patient. He was resistive and disoriented, mistaking the hospital and his nurses for his home and family. There was no direct evidence of hallucinations.

Morphine, $\frac{1}{4}$ grain, and physostigmine, $\frac{1}{50}$ grain, were again given at 12 noon, and the convulsive movements grew less severe. Perspiration now became profuse and he began to take fluids well. The face was still flushed, the mouth dry, the eyes sparkling, and the pupils dilated and fixed. His speech was ataxic and slurred, with rambling incoherent phrases, and he began to show seemingly purposeful movements of his hands and arms. He tried to get out of bed, and required more attention. His pulse was stronger, and the blood pressure 150/80, but rhythm was unchanged. He could be fed with fluids without much difficulty.

At 5 p.m., six hours after admission, he was still semi-comatose, with fixed pupils, flushed face, and profuse sweating; he vomited 3 oz. of bile-stained fluid. The reflexes were brisk, the plantar flexor, the limbs less spastic, and the convulsive movements had ceased. He was restless and voluble, and seemed to be hallucinated, picking at the bedclothes. Morphine, $\frac{1}{6}$ grain, was given. At 8 p.m. he was more rational, asking where he was: he said he was very thirsty and had burning sensations in his feet. He was sweating profusely all over, and stated that everything he looked at was blurred. He went to sleep at 11 p.m., still sweating.

Next day he was mildly excited and elated, voluble but quite rational. He was unable to give any history of the poisoning. He complained of thirst, burning of throat, unsteadiness of limbs, and headache. He still could not see distinctly. He took fluids well and had full control of his sphincters. The only abnormal physical signs were fixed dilated pupils and a persistently raised pulse rate of 120. On the third day after admission he gave a clear rational account of the accident and showed a mild euphoria, tending to laugh about the whole affair. He was quite normal the next day; his temperature and pulse had settled, but his pupils were still widely dilated and showed only the faintest reaction to light.

The patient was discharged fully recovered eleven days after admission, with pupils still larger than normal and reacting poorly to light: there were no other abnormal signs or symptoms. When seen again two months later he was in perfect health.

Discussion

Most of the reported cases of atropine poisoning in advanced age with recovery are characterized by small dosage, and illustrate the accepted teaching that susceptibility to atropine increases with age. Moorhead (1919) describes two cases of severe poisoning, one in a man aged 84 and the other in a woman aged 75, both caused

by the absorption of linimentum belladonnae through the unbroken skin after massage. Métivier (1935) also describes how a woman aged 74 developed severe symptoms of poisoning after the instillation of eye drops containing 1% atropine. She became excited, confused, and hallucinated, and the symptoms took some little time to wear off.

A fatal case is described by Taylor (1934) in which a previously healthy woman aged 66 died sixteen hours after taking only 1 drachm of linimentum belladonnae. However, in younger people recovery can occur after much larger doses. One of the largest doses is reported by Grattan (1881), who describes the case of a woman aged 43 who recovered after she had taken a wineglassful of the liniment. The dose was estimated at 18 drachms. The present case is unusual in that a man aged 73 recovered after taking 5 drachms of linimentum belladonnae (the equivalent of 1.2 grains of atropine alkaloid). The dosage was checked by his doctor and his wife: the bottle was full and unopened, there was no evidence of any having been spilt, and the patient had swallowed it at one gulp and was unable to rid himself of any of it. A further unusual feature is that ten hours elapsed before any treatment was begun. No previous record was found of a patient recovering after a large dose of atropine with such delay in treatment, and it must be rare for any patient to pay such little heed to accidental poisoning.

The treatment itself was along orthodox lines according to such authorities as Glaister (1931) and Taylor, with the addition of spinal drainage for the convulsive movements. This procedure seems not to have been previously reported in similar cases. Most benefit appeared to follow the administration of morphine in adequate dosage, although the Edinburgh committee quoted by Taylor concluded that physostigmine had in relation to atropine a wider range of antidotal action than morphine. The rational procedure seems to be a combination of the two—physostigmine as a physiological antidote and morphine as a cortical depressant. Pilocarpine, a time-honoured remedy in atropine poisoning, was not used in this case as there appears to be little evidence that it antagonizes the action of atropine on the brain (Cushny, 1924).

Further unusual features were noted in the symptomatology. Whereas the selective action of atropine on the central nervous system is well known, increased spasticity (resistance to passive movement) in the limbs with an extensor plantar response is not usually described. Presumably these signs are only indicative of the extent and severity of the poisoning. The profuse sweating is unusual, and may be taken as a good prognostic sign. It was probably greater than could be explained by the amount of physostigmine administered, and it is suggested that it represented a release phenomenon occurring as the action of the atropine wore off.

Summary

A case of accidental atropine poisoning in a man aged 73 is described and the unusual features briefly discussed.

Recovery occurred after 5 drachms of linimentum belladonnae (the equivalent of 1.2 grains of atropine alkaloid) had been taken and no treatment given until ten hours afterwards. Certain unusual symptoms are mentioned.

Treatment comprised morphine, physostigmine, and spinal drainage.

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PERFORATED PEPTIC ULCER

A REVIEW OF 175 PERSONAL CASES

BY

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"The perforation of a gastric or duodenal ulcer is one of the most serious and most overwhelming catastrophes that can befall a human being."—MOYNIHAN.

Although this well-worn subject is covered by a vast amount of literature the frequency of the condition and the vital necessity for early recognition and urgent operation justify periodical review and reconsideration. It is doubtful whether any case gives the surgeon as great a feeling of satisfaction as a perforated peptic ulcer successfully dealt with, and certainly no patient is more grateful. There still appears to be a doubt regarding the future prospects of such a case, and relatively recent statistics are conflicting. It is one of the purposes of this paper to discuss the after-histories of 89 cases in which the follow-up was successful.

The present series, consisting of 175 cases, is an entirely personal one, extending over the decade 1930–40. Of these, 7 occurred in women and 168 in male subjects.

TABLE I.—Showing Site of Ulcer

Duodenal ulcer	100
Gastric ulcer	71
Pyloric ring, 33; Pyloric antrum, 6; Anterior wall of body of stomach, 6; Posterior wall of body of stomach, 1; Lesser curvature, 11; Site not particularized, 14.	
Gastro-jejunal ulcer	3
Ulcer of duodenal stump (after gastrectomy some years previously)	1

In 7 cases the perforation was the second which had occurred. I carried out both operations in 2 of these.

Previous History

In reviewing this group of cases it was found that the majority had had some symptoms previous to the acute perforation. Often these symptoms consisted merely of a period of indigestion lasting for a few days or weeks, but many patients had a long-standing history. When such was the case the symptoms for the ten days or fortnight before the perforation were often said to be especially severe. A few patients stoutly adhered to the statement that there had been no previous indigestion whatsoever. In this connexion confusion has probably arisen, as many patients with perforated ulcer are, owing to pain, reluctant to speak, much less to relate details, of previous mild symptoms. For this reason most of the patients in the present series were interrogated regarding the previous history before discharge from hospital. With this precaution few were found who had not had some indigestion before the acute onset. While some cases perforated when a heavy weight was being lifted—a packing-case in one instance, thus raising a medico-legal problem—I was impressed by the fact that several perforations occurred while the patients were in bed and asleep, the severe pain awakening them.

TABLE II.—Total Length of History

No previous symptoms	15
History of several days	5
" " " weeks	10
" " " months	35
" " " years	53

Physical Signs and Diagnosis

The diagnosis is usually easy if it is remembered that board-like rigidity of the abdominal wall depends to a certain extent on the muscular development and usual tonus of that covering. In a female with a lax visceroprotic type of abdominal wall it is dangerous to expect to find a hard board-like rigidity, and it is valuable,