over an hour, and were associated with great emotional disturbance, shouting, and other evidence of cerebral irritability. On one occasion, while we were doing out-patients, this man was brought into hospital in a typical state of epilepsy; four attendants were necessary to hold him down, and he was very violent and twitching all over. He was given 10 c.cm. of evipan intravenously and his condition quieted at once into normal evipan sleep; on recovery, about an hour later, he was far less excited and almost normal.

This is the only case so far in which opportunity has occurred of using this drug in this way, but it would seem to indicate further possibilities for exploration. Doses of 5 to 10 c.cm. have also been used with success in cases of renal and gall-stone colic.

Conclusions

Sodium evipan in the majority of cases can be considered a safe anaesthetic. It should not, however, be administered single-handed, in view of the possibility of respiratory spasm, or of obstruction of the airway, which may occur while the doctor's attention is engaged elsewhere. Evipan can be repeated on several occasions without ill results; it is not advisable to follow the injection with chloroform; gas and oxygen being the ideal or, where thought necessary, ether.

Premedication with omnopon and scopolamine is a useful adjunct to anaesthesia, and, owing to the high therapeutic quotient of the evipan, and its rapid detoxication, does not carry with it the usual dangers so popularly ascribed to the clandestine association of morphine with the other barbiturates. As Moncrieff¹³ points out, however, the double depression produced by the combination of morphine and drugs of this type may cause respiratory failure, and we strongly deprecate preliminary sedation in patients who are bad anaesthetic risks. It would appear that the most valuable treatment in cases where respiratory depression has supervened would be the inhalation of a mixture containing oxygen and 7 per cent. carbon dioxide,¹³ though in a case above described this proved of no avail. Artificial respiration of itself is unlikely to be useful because carbon dioxide is washed out of the lungs; in combination with the above-mentioned inhalation, however, this difficulty is remedied. Although we have as yet had no experience with alpha-lobeline, we have not been impressed with the efficacy of stimulants. In those cases where we used coramine, it produced at most a temporary improvement, although given by the intravenous route. It is significant to note that, in the fatalities reported, the patients have been extremely ill, and most of them have died of respiratory failure.

Four practical points that seem to be evolved are as follows:

1. To avoid using the drug in cases of severe liver embarrassment, because detoxication cannot occur efficiently.

2. Where there is any question of depression of the respiratory centre—for example, in operations for cerebral tumour, or severe lung conditions—evipan would seem to be contraindicated.

3. In cases where evipan is contraindicated, but after careful weighing of risks it is thought to be the safest method of anaesthesia, it should be given alone without reinforcement by other drugs; the dosage should be cautious, and a preliminary injection of atropine should be administered with a view to counteracting the tendency to oedema of the lung.

4. Facilities should be at hand to administer oxygen and carbon dioxide mixtures ("carbogen") containing a minimal proportion of 7 per cent. CO₂ either by mask or, where necessary, via an intratracheal catheter in cases which are considered to be bad anaesthetic risks.

REFERENCES

- ¹ Weese, H.: *Deut. med. Woch.*, 1933, ii, 47.
- ² Kennedy, W. P., and Narayana, B.: *Quart. Journ. Exper. Phys.*, 1934, xxiv, 69.
- ³ Vide supra Ref. 1.
- ⁴ Vide supra Ref. 1.
- ⁵ Vide supra Ref. 2.
- ⁶ Abel, A. L., and Jarman, R.: *Lancet*, 1933, ii, 18.
- ⁷ Idem: *Ibid.*
- ⁸ *Lancet*, 1933, ii, 43.
- ⁹ Jarman, Ronald: *British Medical Journal*, 1934, i, 796.
- ¹⁰ Landau, E., and Wooley: *Ibid.*, 1934, i, 192.
- ¹¹ Peterkin, G. A. G.: *Ibid.*, 1934, i, 456.
- ¹² Slot, G., and McDade, R. S.: *Lancet*, 1933, ii, 1035.
- ¹³ Moncrieff, A.: *Proc. Roy. Soc. Med.*, 1934, xxvii, 5, 615.

INJECTION TREATMENT OF COMPLETE RECTAL PROLAPSE

WITH REPORT OF TWO CASES

BY

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The treatment of ordinary haemorrhoids by submucous injection of carbolic acid in almond oil has become a well-recognized and ordinary procedure, and since I introduced this particular technique into England in 1928¹ it has been very extensively adopted. I notice, however, that many writers upon the subject still emphasize the importance of a very careful selection of cases, and many of them have said that cases of haemorrhoids with marked prolapse requiring digital reposition are unsuitable for any treatment short of operation, except as a palliative. I have always dissented from this view, and have treated a very large number of such cases by submucous injection with gratifying success.

I describe below some cases of *complete prolapse of the whole rectal wall* which I have treated by means of injection, and I urge that a conscientious trial of this method should be made by other rectal surgeons before submitting patients to one of the ordinary operations for rectal prolapse. All operations for rectal prolapse are severe, and all are very liable to failure. It follows, therefore, that if there is a less dangerous and less drastic method of treating patients suffering from this distressing condition it is worthy of trial, even if the relief it gives is only temporary. One of the greatest advantages of treatment by injection is that it is an *ambulatory* method—that is, it does not necessitate any confinement to bed.

Of operations for rectal prolapse one of the most generally employed is that of Lockhart-Mummery. In describing his operation² Lockhart-Mummery writes:

"*Treatment After Operation.*—The wound is dressed twice daily and fresh dressings applied, but the packing is not removed for a week. At the end of this time an anaesthetic is given and the packing removed, and a fresh lot of gauze introduced. . . . The wound should not be allowed to heal under three weeks; in fact, the more slowly it heals the better the result. The bowels are kept confined until the seventh day after operation, when they are relieved by an enema before the removal of the packing; after this they are opened daily with an enema, a slipper bed-pan being used. The patient is not allowed out of bed for six weeks, and not allowed to sit up for an action of the bowels for two months at least. It is most important, in view of the large area of cellular tissue opened up, that there should be no sepsis at the time of the operation, but sepsis later is almost certain to occur after the removal of the packing, and is of no consequence" [sic]. "The slower the wound heals, and the more fibrous tissue involved, the better the result. Out of a total of thirty-two cases there have been only five failures" [that is, a recurrence rate of over 15 per cent.], "and two of these were permanently cured by repeating the operation."

Most people will not look forward with pleasure to such a programme as this, and, if one can tell a patient honestly that there is any hope of avoiding such a prospect by means of injection, I doubt whether one is to be found who would not cheerfully accept the risk of recurrence in order to do so—more especially if he is told that there is at least some risk of recurrence even after the most skilfully performed operation.

Of the other methods of operation for prolapse—by means of linear cauterization of the rectal wall, Whitehead's operation, sigmoidopexy and excision of a portion of the bowel with end-to-end anastomosis—all have the same grave objections from the patient's point of view. All involve long confinement to bed, all have a very definite mortality rate, and all have quite a high percentage rate of recurrence and failure. In elderly patients most surgeons would hesitate to recommend such operations, because for them the risk is very real.

Case Records

Case 1.—In October, 1929, I saw a lady, aged 58, who had suffered from rectal trouble for eighteen years. It had commenced with the ordinary symptoms of haemorrhoids—bleeding after defaecation, followed shortly by prolapse, at first after defaecation only and then by prolapse on walking, exertion of any sort, and even on sneezing. Gradually a condition of complete incontinence of faeces had developed. When I saw her she was in a wretched condition; her clothes were always stained with faeces, mucus, and blood, there was considerable emaciation, and she was extremely depressed. Perhaps fortunately for her, there was considerable constipation. On examination, the sphincter was quite lax, and it hardly contracted when she was asked to grip the examining finger. The anus looked almost as if a Whitehead's operation had been performed upon it. On straining, about $3\frac{1}{2}$ in. of the whole bowel wall prolapsed completely. The speculum, when introduced, practically "dropped in." She had some extremely large internal haemorrhoids, extending for some two inches up the rectum. The mucous membrane was ulcerated and inflamed from friction against her clothing.

She absolutely declined to submit to operation, so with considerable hesitation I consented to see what I could do for her by means of injection. When she came for her second injection at the end of a week she reported that, though there was still slight prolapse, there was marked improvement, that she had much more control over defaecation, and that she had had no inconvenience from the treatment excepting slight pain for four hours after it. After the second injection she reported that there was no further prolapse at all, in spite of some exercise, but she still had some loss of control if the motions were liquid. She had five injections at weekly intervals, and after that she reported that she felt better than she had done for years, and there had been no prolapse from after the second injection, although there was still slight incontinence for liquid motions. Her constipation was being regulated by means of Kaylene-Ol, and she had been told to endeavour to adjust the dose so as to keep the motions formed. In this way she was able to get about in comfort, but she was not able always to control flatus. I found that all the piles were quite hard and small, the rectal wall was holding up perfectly, but the sphincter was still very lax.

She returned in September, 1930—eleven months later—reporting that up to the previous May and June she had been perfectly comfortable. Since then there had been a return of the lack of control of flatus, and, to a less extent, of liquid faeces, and more recently there had been slight prolapse on defaecation. Her weight was steady and satisfactory. I found no definite haemorrhoids, but the rectal mucosa was again lax and voluminous. She had four more injections, after which she said that she was "extraordinarily better," and that there was no prolapse or leakage even during an attack of diarrhoea. She came again in May, 1931, complaining of slight return of incontinence of flatus and liquid faeces, and I found that there was still no trace of piles, but that the anterior rectal wall prolapsed slightly on straining, and the sphincter remained very lax. She had one injection then and another on her next visit to London a

month later. I urged her to undergo a plastic operation to repair the sphincter and to narrow the anus, but she would not consent to do so.

Her next appearance was exactly a year later, when she said that she had remained well again for six months and that after that the prolapse had reappeared and had been gradually increasing; she had been a little incontinent again for the past two months. I again found a complete absence of haemorrhoids, but the mucous membrane was again loose and voluminous. I gave her three further injections. After that she remained well until March, 1933, again reporting a slight relapse after six months of complete comfort. She had three further injections. I had a satisfactory report from her in June of that year.

I fear that she is likely to have further relapses, but she is prepared to face these and to have further injections, rather than even to undergo a somewhat unimportant plastic operation to narrow the anus. Throughout the whole series of injections the only trouble and complications that she experienced were occasional pain for four to six hours after injection, and on two or three occasions some pyrexia for twenty-four hours after treatment.

Case 2.—An unmarried woman, aged 75, had had rectal trouble for seven or eight years—gradual loss of control over the bowel, considerable mucous discharge, very occasional bleeding and prolapse, the onset of which coincided with the appearance of the incontinence. She had been previously diagnosed as "colitis and prolapse," and advised that she was too old for any radical operation. Colonic irrigation produced some temporary benefit, but never real relief, either to the prolapse or to the incontinence.

On April 20th, 1933, I found the sphincter extremely lax, but fairly active. There were some moderate-sized internal haemorrhoids, and a huge prolapse of the whole bowel wall when she strained. The mucous membrane was very lax for the lower six inches. I injected this all round as high up as it extended, and also injected her haemorrhoids. At her next visit she reported that she was "greatly relieved." There was no prolapse after walking, but still a little after defaecation; the mucous discharge had ceased, and the incontinence was less. After her third treatment the prolapse had entirely ceased, even after defaecation, but there was still slight lack of control. At her fifth visit she reported herself quite comfortable, and stated she had spent three hours at a flower show without discomfort or fatigue. After that she went to stay in Scotland for three weeks, but I saw her again in June and gave her two more injections. She had no return of the prolapse, her bowels were acting less erratically, and she had less incontinence. I saw her twice again in August, and there was no return of the prolapse, but still incontinence of liquid faeces. On November 8th the patient wrote to me from South Africa that she was keeping perfectly well. She added: "The control is very much better. I have recovered from a pretty severe cold and cough, but now coughing does not trouble me at all."

It is too soon after six months to claim this case as a "cure"—indeed, I rather anticipate that there will be a recurrence sooner or later. However, if there is any tendency to relapse, she will have a few more injections, and, I hope, another year's relief.

Discussion

In both of these cases there was quite definite prolapse, not merely of internal haemorrhoids, but of a considerable length of the whole rectum. Both had toneless sphincters with obliteration of the anal canal, and both of them complained chiefly of incontinence of faeces. I do not suppose that complete relief will be permanent in either case—indeed, the first of these patients has been coming annually since 1929.

The technique differs slightly from the method of injection of internal haemorrhoids with phenol in almond oil which I described in 1928, in that for the high injection in true rectal prolapse one aims at inserting the needle slightly deeper than the submucous tissue. It should be introduced deeply enough to reach the muscular coat. If this is done successfully the swelling produced by the oil in the submucous tissue does not appear to the same

extent, and the "striation sign" that enough has been injected at any particular spot does not occur. One is reduced to guess at the correct amount. I have usually injected 2 to 3 c.cm. at each puncture. The aim is to inject all round the rectal wall. After two or three treatments, if one feels the rectum from the vagina, the whole bowel feels hard, and the sensation closely resembles that produced by an annular carcinoma of the rectum when felt from the vagina. All this thickening disappears some three or four weeks after the completion of the treatment. This inflammatory process results in close adhesion of the mucous and submucous coats to each other and to the muscular coat. This in itself prevents the bowel wall from prolapsing, and, moreover, the submucous space being more or less obliterated, the main haemorrhoidal vessels which feed the piles are compressed and the haemorrhoids are cured in that way.

This method of treating true prolapse, like the submucous injection of haemorrhoids, was first carried out in the United States. Considering how unsatisfactory the treatment of this condition by operation has proved, I hope that others will give their patients the option at least of trying injections before resorting to more drastic operations, with their discomforts, dangers, and uncertainties.

REFERENCES

- ¹ *Lancet*, March 17th, 1928.
² *Medical Annual*, 1922, p. 369.

ADDISON'S DISEASE DUE TO MALIGNANT INVOLVEMENT OF THE SOLAR PLEXUS

BY

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It is generally stated in textbooks that one of the rarer causes of Addison's disease is the involvement of the solar plexus and semilunar ganglia by malignant growth. Rolleston¹ says:

"There are cases in which symptoms like those met with in Addison's disease are found in association with alteration of the semilunar ganglia or abdominal sympathetic, the suprarenals appearing healthy. Thus the solar plexus and semilunar ganglia have been surrounded by lymphadenomatous growth, whilst the suprarenals were found to be intact."

However, very few such cases have been recorded in the literature. Addison's tenth case was probably of this nature, but after the collection made by Lewin,² who found healthy suprarenal glands in 12 per cent. of cases reported in the literature up to 1892, I have not been able to find such a case. Rowntree and Snell,³ after reviewing the work of the Italian clinicians Pende and Varvaro,⁴ state: "One may infer that there is a possibility of functional suprarenal insufficiency depending on interference with secretory nerves to the gland; however, we have not observed such a case in our series." Neither was any such case reported by Conybeare and Millis.⁵

The following case, besides the fact that the symptoms were presumably due to involvement of the solar plexus, and not the capsule, by malignant growth, is also of interest as regards the darkening of the hair and the high blood sugar.

Sir W. Wilkes⁶ stated that only one or two cases were recorded where the hair had darkened in Addison's disease, and though it is stated³ that "the hair often becomes darker," this is not a symptom which I have found recorded in any individual case. The fasting blood sugar is usually low or well within normal limits^{7, 8}; not, as in our case, as high as 0.137 per cent.

Case Record

The patient, a man of 50, was first seen at the end of August, 1933. His family history was entirely uninteresting, and his own past history was uneventful. He only remembered having measles, and becoming very freckled as a child in the summer. During the war he served in France, and since then had been a caretaker.

History.—Some time in 1930 he began to get epigastric pain after his meals: this pain was relieved by alkalis, and did not seriously worry him, although it never completely disappeared. In April, 1933, he noticed that he was more sunburnt than usual, and at the same time his abdominal pains became more severe and different in character. He got steadily worse until the end of August. His face, hands, and forearms grew progressively darker in colour; his body as a whole did not become pigmented, save to a slight degree over his abdomen. The whites of his eyes and carunculae he noticed remained normal. His nipples and genitalia became a deep black. His hair and moustache, which had been turning grey, became much darker, "as if he were becoming younger." There also appeared small discrete dark spots over his trunk and arms. Periodically the pigment grew much more intense, and then lighter again, so that "his face was copper-coloured, or like a negro." These sudden increases in pigmentation were always associated with severe abdominal pains, lasting about half an hour, relieved by bringing up wind, and not related to food or exercise. He also became progressively weaker, so that by August he had to stay in bed. He quite lost his sense of taste and smell—chicken, fish, and bread all tasting the same—while everything appeared to have an earthy smell.

Progress.—In September he began to feel much better. He was becoming almost normal in colour, his abdominal pains were less, and he felt he could work. An x-ray, taken at this time, showed a small lesser-curve ulcer. He returned to work for a week, but had to stop, as he began to have severe pains and vomiting. The pain became much less, but the vomiting continued, generally preceded by nausea, but leaving him feeling quite well afterwards. It had no relation to his meals. Late in November he was admitted to hospital.

Examination.—He was well covered, in spite of having lost 2 st. in the last six months. His skin was of a uniform dark yellow-brown, with darker discrete spots. The palms of the hands were pale, with the creases dark brown. The nails were normal. Pigment began sharply at the wrist. His hair was a mixture of black and grey. His moustache and axillary hair were dark brown. He stated that originally his moustache had been grey. There was a dark line on the buccal mucosa opposite the crowns of the teeth. Pigmentation was most intense over the arms, face, and genitalia. A biopsy on a piece of his pigmented skin showed no free iron. His cardiovascular system was normal. His blood pressure was 144/98 mm. Hg on admission, falling to 96/57 mm. two weeks before his death. His blood count was 3,760,000 red cells, with a colour index of 0.5; 13,000 white cells, 87 per cent. polymorphs, 10 per cent. lymphocytes, 3 per cent. large hyalines. In the epigastrium a mass of indefinite outlines could be palpated: it was thought to be a gastric carcinoma. His gastric juice contained blood, and no free HCl until after an injection of histamine, when 0.168 per cent. of free HCl was present. The faeces contained large quantities of occult blood. The x-ray of his stomach suggested a neoplasm. An x-ray of the suprarenal areas was negative. Clinically and by x rays his chest was normal; his nervous system and urine were normal; the blood urea was 42 mg. per 100 c.cm., and the Wassermann reaction was negative. Fasting blood sugar was 0.137 per cent. While in hospital the patient's condition steadily deteriorated.

Asthenia was pronounced from the date of admission, and became so marked that he could scarcely move or speak. Vomiting was incessant. He died on December 30th, 1933.

Post-mortem Findings.—I am indebted to Dr. J. L. Edwards for the following report on the necropsy. The patient showed emaciation and extreme pigmentation of a brownish type, mainly of exposed parts. The adrenals appeared normal. There was no evidence of malignancy, tuberculosis, or atrophy, but a large mass of carcinomatous glands pressed directly on to the coeliac plexus. The brain appeared normal.