Clein consider that the drug ought to be included in Schedule 1 of the Poisons Rules. To obtain drugs on Schedule 1 the poisons book must be signed, but a prescription is not necessary.

It is not intended here to discuss the details of personality of the patient except in so far as they throw light on the reason for addiction to this type of drug—that is, one obtainable without prescription. It appears that not only must the drug be readily available—as all these are to the entire population—but the patient must be aware of their existence and also be prepared to take the drugs in an experimental fashion. They will take as much as is required to alleviate the symptoms rather than pay heed to warnings on the box about it being dangerous to exceed the stated dose. It is thus people who have some medical or nursing knowledge, and whose knowledge gives them a feeling of confidence, who are likely to take to these drugs. The patients concerned are usually not now working, since they could then obtain barbiturates, but have been connected with the medical, dental, or nursing professions in the past. The high incidence of drug addiction of all kinds among doctors, dentists, and nurses has long been recognized, and this series of patients must be considered as part of the same problem, which is not made easier to solve when the drugs concerned are so readily procurable.

The W.H.O. Technical Report series No. 57 (1952) divides drugs into three groups: (1) morphine and allied groups, with a pharmacological action producing addiction in any person if more than a certain—often small amount—is taken; (2) a group whose pharmacological action never produces compulsive craving, yet is found desirable by some individuals—producing habituation which can be interrupted without significant disturbances; (3) an intermediate group, in which the psychological make-up of the patient is paramount, but the pharmacological action of the drug does play a significant part. The sedatives mentioned here are usually regarded as belonging to the habit-forming group, whereas they should almost certainly be included in the intermediate group. Even if addiction is produced in only a small number of patients, these become much more difficult to treat if they can get supplies of drugs so easily. Fraser and Grider (1953) point out that drug addiction is a "contagious disease" which may spread from an addict to a hitherto drug-free though possibly predisposed person. At least two of these patients started their addiction this way.

Conclusion

It appears, therefore, that all drugs which have a euphoriant or sedative action are likely to cause addiction in predisposed personalities, and the treatment of such patients is made more difficult when such drugs can be freely obtained without any prescription. It is therefore recommended that all such drugs be included in Schedule 4 of the Poisons Rules—that is, can be obtained only on prescription.

In the case of new drugs which have such an action and therefore in all probability will lead to addiction, these should be included in the Scheduled List of Poisons *ab initio*, instead of being sold across the chemist's counter without hindrance until cases of addiction have been described. Vogel *et al.* (1948) point out that ideally the prevention of drug addiction is brought about by developing a people so emotionally sound and well integrated that they will have no need for chemical aids to adaptive behaviour. The second and more realistic approach in our present society is to keep the addiction-prone and the ex-addict from narcotic drugs.

Summary

Attention is drawn to the possibility of addiction, using the W.H.O. criteria, to drugs which may be obtained without prescription.

Seven cases of addiction to such drugs are described.

It is suggested that all drugs which have euphoriant or sedative action may cause addiction and should be placed on the list of Schedule 4 Poisons. They could then be obtained only on prescription.

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CASE OF INFANTILE ULCERATIVE COLITIS

BY

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We record here a case of ulcerative colitis in an infant who is probably the youngest patient suffering from this disease yet reported. The child was barely 3 weeks old when the symptoms first presented.

Ulcerative colitis in infants runs a similar course to that in adults. The disease may present as an acute fulminating attack, as recurrent subacute attacks, or as an insidious progressive unrelenting disease which keeps the patient a constant invalid. However, in infancy the disease seems to be more severe, has a higher incidence of complications, and is less responsive to treatment—a view which accords with that of Bargen and Kennedy (1955).

The diagnosis of infantile ulcerative colitis may be difficult and should be kept in mind when an infant presents with symptoms of persistent diarrhoea, especially if the motions contain blood and mucus. Proctoscopy, sigmoidoscopy, and barium enema will confirm the diagnosis in nearly every case.

In acute disease extensive *x*-ray changes may be apparent in less than two weeks (Hodgson and Kennedy, 1955). In their review they follow Weber and Bargen's (1940) classification and divide their patients as follows. Type 1:—*X*-ray findings positive, the lesion beginning in the rectum and progressing proximally; proctoscopic changes present. This group was the commonest in their series. Type 2:—Proctoscopic manifestations negative or equivocal; *x*-ray evidence of disease present. Type 3:—Unusual proctoscopical appearance with peculiar mucosal pattern and ulcers in the rectum and sigmoid; radiological examination reveals evidence of disease on the right side.

In nearly 95% of cases the disease involves the rectum and most of the colon. A preliminary bacterial examination of the stool is always carried out to exclude a bacillary dysentery.

As regards the prognosis, the younger the child, the more acute the attack, the worse the outlook. Lyons (1956) maintains that the longer the child is able to survive the ravages of the disease the better are his chances. The highest mortality is in the first year of the disease. Long-standing ulcerative colitis is associated with an increased incidence of malnutrition, retardation in development, and advanced pathological changes in the bowel, such as stricture, polyposis, and neoplasm. Except for frank neoplastic changes, prognosis generally is based more on the child's clinical adjustment to the disease than on the gross and microscopical findings in the bowel.

Treatment

Medical measures, except for the complications, seem to afford some relief. Most patients respond with alleviation of their symptoms, yet they never experience complete remissions, nor do they have a normally formed stool (Meeker and Goff, 1956). This presents rather a problem to the clinician in that the child is not entirely well but never ill enough to require surgical intervention. With the advent of steroid therapy it was at first thought that the disease could be held in check, but, except for the very acute fulminating attack, this treatment has in the main proved disappointing.

Active supportive therapy has been found to give the best results. This includes small frequent blood transfusions and parenteral protein and vitamin therapy, and the maintenance of correct fluid, electrolytic, and nitrogen balance.

It is generally conceded that surgery is mandatory in acute toxic ulcerative colitis not responding to medical therapy, and in threatening sequelae: (*a*) perforation, (*b*) stricture, (*c*) arthritis and dermatitis, (*d*) severe growth failure, (*e*) malignant degeneration. In resorting to surgery, an ileostomy alone should be performed only as a temporary emergency measure.

Total colectomy, with or without resection of the rectum, is the operation of choice. Many surgeons are inclined to the more conservative ileorectal anastomosis in the first place, with observation of the rectum at regular intervals by sigmoidoscopy.

Case Report

A male infant aged 3 months, an only child, was admitted to the children's medical ward on December 7, 1955. He had been breast-fed for the first month and then artificially fed. His weight on admission was 11 lb. 4 oz. (5.1 kg.). At the age of 3 weeks the mother first noticed that there was slight pink staining of the napkin by the motion. She also volunteered the information that the child whimpered at each bowel action. The motions were as frequent as two- to three-hourly and were soft and greenish. The child was admitted to a fever hospital on three occasions for investigation of the diarrhoea, and each time was discharged home without a diagnosis having been made. No pathogenic organisms had been cultured from the stools.

The family history revealed nothing relevant. The mother, father, and infant live in one room in the home of the grandparents.

On examination the pulse was 130 and the temperature 99.4° F. (37.4° C.). The baby appeared pale, but lay quietly on the examination couch. There was loss of elasticity of the skin, and the fontanelles were slightly depressed. The respiratory and cardiovascular systems showed no abnormality. Per abdomen the spleen and liver were not palpable, and rectally the sole abnormality was red blood on the examining finger.

Laboratory investigations: Hb, 86%; serum electrolytes normal; direct film of stools showed no parasites, but pus and red cells were abundant, and on culture no pathogenic organisms were grown. Two further stool examinations gave similar findings.

The child's condition continued to deteriorate, the motions were frequent, watery, and blood-stained, and the temperature remained between 100 and 102° F. (37.8 and 38.9° C.). Supportive therapy included small blood transfusions and half-strength Hartmann's solution subcutaneously. After a week the child's weight had dropped 1½ lb. (680 g.).

Changes in feeds were made, but for the next 14 days the child's condition remained *in statu quo*, except that the diarrhoea was much less. A small transfusion of 150 ml. was given. The patient failed, however, to make progress, motions again becoming frequent and bloody. There was now a marked fall in serum electrolytes, especially of the serum sodium. This was rectified by subcutaneous N/6 saline and Hartmann's solution.

A barium-enema examination showed that the descending and proximal sigmoid colons were extremely irritable, resulting in rapid return of the enema. Filling of the colon was obtained only as far as the hepatic flexure. There was persistent spasm of the wall of the descending colon, and the mucosa of the sigmoid and descending colon appeared thickened.

Sigmoidoscopy under anaesthesia revealed a rectum half filled with blood-stained fluid. The mucosa was inflamed, but no ulcers were seen. As a result of these findings laparotomy was decided upon, and, as the child had been prepared for such a contingency, operation was proceeded with (D. L. S.)

Through a lower left paramedian incision the abdomen was explored. The whole of the sigmoid colon was dilated and thickened, as in inflammatory lesions such as ulcerative colitis. Numerous small glands were present in the mesentery. The transverse and ascending colons were normal. The risks of a major resection were weighed with the findings. It was decided to close the abdomen and try the child on a course of cortisone.

Cortisone therapy was started 10 days post-operatively. The initial dose was 50 mg. a day. At this stage laboratory investigation revealed: Hb, 92%; serum potassium, 4.5 mEq/l.; serum sodium, 135 mEq/l.; serum chloride, 95 mEq/l.; CO_2 combining power, 58 vols.

During the next few weeks the child's condition remained very much the same. The diarrhoea seemed to improve and less blood was found in the stool. There was no gain in weight. The cortisone was then reduced to 37.5 mg. a day. Serum electrolytes remained unchanged.

For the next eight weeks the cortisone was gradually reduced and the child's condition remained very much the same; he, however, had gained 1 lb. (450 g.) in weight and there was no evidence of any oedema.

At the stage where the cortisone was being given in doses of 5 mg. a day the child suddenly developed a pyrexia of 105° F. (40.6° C.) and a pulse of 150. Examination did not reveal any definite cause for this, but the cortisone was discontinued, as some apprehension was felt about the possibility of a perforation of the diseased colon. The temperature continued for five more days and then gradually subsided.

Soon after discontinuing the cortisone the motions again became more frequent and continued to contain blood and mucus. Weight remained stationary, and abdominal examination showed a palpable irregular descending and sigmoid colon. Three months after the start of cortisone the child had not shown any real improvement.

Sigmoidoscopy at this time revealed a red and ulcerated rectum and operation was recommended. Preparation was made for a colon resection, and an intravenous infusion into the long saphenous vein was started on the day of operation.

Operation (D. L. S.).—The incision was made through the old scar. On entering the abdominal cavity thorough search

was made to establish the exact extent of the disease. It was obvious that the descending colon, sigmoid colon, and upper rectum were maximally affected. The rest of the colon appeared to be quite normal. There was no evidence that a small perforation had occurred in the past. The left third of the transverse colon was included in the resection, and this was carried down to the rectum to within 2 cm. above the peritoneal reflexion. An end-to-end anastomosis was performed, the mesenteric opening closed, and the abdomen sutured without drainage.

Micropathology.—There was intense subacute inflammation of the submucosa with areas of ulceration, and mucosal regeneration at the edges of the desquamated areas. Exudate consisted mainly of eosinophil and plasma-cell type. Lymph nodes were the seat of hypertrophy.

Post-operatively the child received 150 ml. of blood and repeated subcutaneous infusions. On the third day he passed two blood-stained motions and his general condition was very satisfactory. Laboratory investigations: Hb, 84%; serum electrolytes normal.

Progress.—Despite slight vomiting, small oral feeds—1 oz. (28 ml.) of milk and 1 oz. (28 ml.) of water—were persisted with; and on the fourth day there was no vomiting, and the feeds were increased to 4 oz. (114 ml.) of milk, 1 oz. (28 ml.) of water, 1 dr. (4 g.) of glucose, and one tablet of sodium citrate four-hourly. By the sixth day the child was on full normal feeds and was having four to six motions a day. No blood or mucus was present. On the twelfth day the wound was well healed, and four days later the child was allowed home. The number of stools has gradually decreased to two a day, with an occasional streak of blood but no mucus. He gained approximately 1 lb. (450 g.) a week for the first two months, and since then the gain has continued steadily. His weight was 29 lb. (13.2 kg.) at the age of 2 years.

Sigmoidoscopy six months after operation showed a red rectal mucosa but no evidence of ulceration; six months later it still showed a congested mucosa but no evidence of ulceration. There has been no blood or mucus in the stool for the past few months.

At the time of writing the patient was very well and able to take all types of food; no restriction has been made in his diet.

Summary

This report deals with a child who at the age of 3 weeks first showed symptoms of ulcerative colitis. Medical treatment proved of no avail. Laparotomy was performed three weeks after admission and a definitive diagnosis of ulcerative colitis involving descending colon, sigmoid colon, and proximal rectum was made. No further operative procedure was undertaken, and a course of cortisone was suggested as a preliminary measure, with the proviso that if this therapy was not effective resection would have to be undertaken.

Three months after cortisone therapy there was no marked improvement, and on the cessation of the cortisone the child's health began to deteriorate rapidly. A resection of the left third of the transverse colon, descending colon, and sigmoid colon, with an end-to-end anastomosis, was performed. The child made an uneventful recovery, and 16 months after operation had gained over 14 lb. (6.4 kg.) in weight and was having two motions a day without any blood or mucus.

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CARCINOMA OF THE THYROID FOLLOWING IRRADIATION

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There is now evidence from several sources that irradiation of the thyroid gland in infancy or childhood increases the incidence of cancer of the thyroid in later life. Duffy and Fitzgerald (1950) found that a high proportion of children and adolescents with thyroid carcinoma had been subjected to thymic irradiation in infancy or childhood, and suggested that irradiation might have played a part in the aetiology of these tumours. Simpson *et al.* (1955) reported the results of a survey of 1,400 of 1,722 children who had been treated with *x* rays for thymic enlargement in infancy, and found that the incidence of malignant disease, in particular leukaemia and thyroid carcinoma, was significantly higher in the irradiated children than in their untreated siblings or in the general population. Clark (1955) observed that in 15 cases of thyroid carcinoma in children of 15 years of age or younger there was a history of *x*-ray therapy for benign conditions of the head, neck, or thorax in infancy or childhood. Simpson and Hempelmann (1957) in a later report confirmed their original observations, and suggested that there might be a correlation between the type of irradiation given and the subsequent development of tumours.

In this country Kilpatrick *et al.* (1957) found that three out of eight patients who developed thyroid carcinoma before the age of 35 years had been given ionizing radiation to the neck in childhood. In a later review Wilson *et al.* (1958) report seven patients who developed thyroid carcinoma following irradiation of the neck region. In six of these patients irradiation was given during infancy or childhood. Only one patient developed carcinoma of the thyroid following irradiation of the gland in adult life: this patient, who was treated for thyrotoxicosis by *x*-irradiation at the age of 26, developed an anaplastic carcinoma of the thyroid 37 years later.

It is generally considered that the adult thyroid is not susceptible to radiation cancer. Such a belief has been fostered by an absence in the literature of cases of thyroid carcinoma which might have been attributable to previous irradiation. Negative evidence of an association between radiation and cancer may, however, be misleading. At New End Hospital between 1954 and 1956 three patients who developed carcinoma of the thyroid following irradiation of the neck have been seen: in two of these irradiation was given during adult life.

Case 1

A woman aged 36 was seen in 1955 and found to have a mass in the left lobe of the thyroid gland. In 1937, at the age of 18, she had been treated with *x* rays for thyrotoxicosis. Ten treatments had been given. There were no appreciable radiation changes in the skin or subcutaneous tissues of the neck. Though there was no obvious evidence of hypothyroidism the improvement in her general health when she was subsequently given thyroxine suggested that she had probably been mildly hypothyroid in the first instance. The tumour was removed surgically, and proved on section to be a follicular carcinoma. The interval between irradiation and the diagnosis of cancer was 18 years.

Case 2

A woman aged 49 was seen in 1954. She complained of a goitre which had first been noticed in 1942. In 1916, at the age