

sided abdominal pain followed by bloody diarrhoea which rapidly improved without specific treatment. The barium-enema examination showed changes at the typical site which progressed from narrowing with marginal thumb-printing, through sacculation, to complete normality (Boley *et al.*, 1963; Marston *et al.*, 1966; Lea Thomas, 1968; Williams *et al.*, 1969). The only unusual feature was the patient's age and sex. The condition usually occurs in the age group at risk from degenerative vascular disease, more common in men (Morson, 1968). In a series of 36 patients with a mean age of 62 (range 42-82) reported by Lea Thomas (1968) the younger patients were mainly women with cardiac or rheumatoid disease.

Though the morbidity is that of acute ischaemia (Marston *et al.*, 1966, 1969; Morson, 1968) and large and small arterial changes may sometimes be found, aortography usually shows nothing abnormal, possibly because the smaller arteries and veins cannot be seen by this technique (Lea Thomas, 1968).

In view of this patient's age and sex and the absence of other precipitating factors it is reasonable to discuss her use of the contraceptive pill. An increased risk of pulmonary embolism, deep-vein thrombosis, and cerebral and coronary thrombosis has been established in women taking oral contraceptives in the United Kingdom (Inman *et al.*, 1970), especially those containing larger amounts of oestrogens. Gangrene of the small bowel, superior mesenteric artery thrombosis (Brennan *et al.*, 1968), and also mesenteric venous throm-

bosis (Reed and Coon, 1963); Civetta and Kolodny, 1970) have been described.

Kilpatrick *et al.* (1968) discussed the cases of two patients aged 29 and 32 taking oral contraceptives who showed radiological evidence of transient ischaemic colitis. These patients together with the present case suggest an aetiological relationship. We feel that ischaemic colitis should be considered in all young women who present with acute abdominal pain and blood-stained diarrhoea who are taking a contraceptive pill.

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Bullous Lesions in Nitrazepam Overdosage

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The appearance of bullae on the skin in unconscious states has been well attested. Much of the work has referred to cases of barbiturate poisoning. In the case described below there was strong evidence to suggest that the patient had taken nitrazepam (Mogadon) and none to suggest that she had taken barbiturates.

Case Report

A woman aged 24 was admitted to hospital deeply unconscious. There were tense bullae, some haemorrhagic and some on a reddened base, affecting the left side of the face, left hand and wrist, and left breast. There was no evidence on searching, checking with doctors who had seen her, or, later, questioning the patient that she had taken barbiturates, there was firm evidence that nitrazepam had been prescribed, and the patient stated that she had taken 100 tablets of this drug.

She recovered uneventfully after having been unconscious for about 36 hours, and the bullae healed to leave considerable scarring. There were psychiatric problems and she did not attend for follow-up. It was learnt a few months later from her general practitioner that she had scarring involving the carpometacarpal and metacarpophalangeal joints of her thumb and that she was having physiotherapy elsewhere, plastic surgery also having been considered.

Comment

Bullae in comatose patients have been noted for many years. Beveridge and Lawson (1965) reviewed the literature, described bullae in 19 out of 290 patients suffering from barbiturate overdose, and found no bullae in 69 patients suffering from overdosage of other drugs—phenothiazine derivatives

and non-barbiturate hypnotics. Beveridge (1970) reported that histologically necrosis of the epidermis, involving the sweat glands, was seen in some affected patients, prompting the idea that barbiturate might be excreted by these glands.

Gröschel *et al.* (1970) reported a case of barbiturate overdosage with bullae in which the blister fluid contained barbiturate. Mandy and Ackerman (1970), pointing out that bullae in carbon monoxide poisoning were described by Schmidt in 1865, surveyed reports of coma of other origin in which bullae had been described, including that produced by methadone, hydrocodone bitartrate, meprobamate, imipramine, and glutethimide. These authors themselves found bullae in 4% of 501 cases of barbiturate poisoning and in 40% of 300 patients dying from this cause. They stressed that bullae are not found in the absence of unconsciousness and suggested that trauma and hypoxia are aetiological factors.

It seems therefore that the presence of bullae in a comatose patient by no means indicates that barbiturates are the cause of the coma. In the collection of hypnotics which have been incriminated nitrazepam seems not to be recorded, and it is not chemically related to those which have been noted. That cases have occurred before, however, seems clear from the experience of R. Goulding (personal communication, 1970) at the Poisons Reference Service of Guy's Hospital.

Finally, as Freeman and Raza (1965) point out, bullae apparently identical can be seen in patients with coma of neurological origin in which there is no question of the ingestion of hypnotics; and they record two such cases.

It may be concluded that the bullae represent a reaction to pressure and hypoxia in the skin of the unconscious patient. Barbiturates have often been incriminated because they are a frequent cause of coma.

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