TABLE I—*Survival Rates in Both Groups of Patients according to Quality of Remission after Combination Chemotherapy*

	MOPP		MOP		All Cases	
	No. of Patients	No. Alive	No. of Patients	No. Alive	No. of Patients	No. Alive %
Complete remission	39	31	18	15	57	46 (81)
Partial remission	8	4	17	9	25	13 (52)
No remission	2	1	6	0	8	1 (13)

Discussion

The quality of the remission was important because it is related to the eventual outcome. In all 90 patients effective control of the disease was obtained in 81% patients after complete remission, 52% after partial remission, and 13% after no remission (table I). It is therefore probable that the significant difference between the complete remission rates of each treatment group will be lower ultimate survival rates in the group treated with MOP. Confirmation of this point may be provided by re-evaluating the patients in this comparative study later.

TABLE II—*Probable Causes of Death in 13 Patients Treated with MOPP and 17 Treated with MOP*

	Hodgkin's Disease	Treatment Complications	Unrelated
MOPP	7	3 (Marrow depression)	3 (Cardiovascular, cerebrovascular, bronchial carcinoma)
MOP	14	2 (Partial response, patient refusal)	1 (Pulmonary tuberculosis with partial response)

It is sometimes difficult to ascertain the exact cause of death in patients who have been treated for Hodgkin's disease. Examination of the probable causes of death in the two groups (table II), however, suggest that inclusion of prednisone in the drug combination does not reduce bone marrow toxicity. Three deaths were associated with bone marrow depression and all were in the group treated by MOPP.

Unless other contrary evidence is forthcoming it seems to be unsafe to omit corticosteroids from the MOPP combination in the treatment of stage IV Hodgkin's disease.

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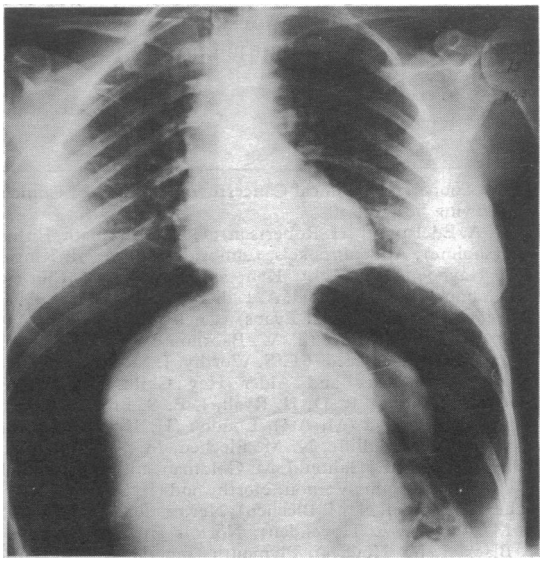
SHORT REPORTS

Case of Complete Gastric Rupture Complicating Resuscitation

A previously fit 30-year-old woman visited her dentist for treatment under intravenous anaesthesia. While supine 10 mg diazepam was given intravenously over 90 seconds, followed immediately by intravenous 2% methohexitone to enable a local anaesthetic to be injected. After 30 mg methohexitone had been given she began to cough and thick mucus was noted in her pharynx. The injection was stopped and the mucus aspirated but she became apnoeic and cyanosed. She was ventilated with pure oxygen from a Salisbury machine with a functioning reducing valve via a facemask and by squeezing a standard 2-l reservoir bag. The bag did not overfill and her chest was noted to inflate. After 90 seconds her cyanotic colour was replaced by pallor. In view of absent pulses and heart sounds external cardiac massage was begun. After four minutes her pulse returned and she regained consciousness.

She was transferred to hospital and her general condition was satisfactory but she complained of abdominal pain. The abdomen was grossly distended with surgical emphysema of the anterior wall. Shortly after admission a small haematemesis occurred. *X*-ray examination of the abdomen showed pneumoperitoneum but a limited Gastrografin study failed to show the site of the perforation (fig.). At laparotomy a 10-cm, full-thickness laceration of the stomach was found along the lesser curvature with numerous serosal tears running parallel. The rupture was repaired and recovery was uneventful.



X-ray appearances of the thorax and abdomen.

Discussion

At necropsy lacerations confined to the mucosa of the stomach are found in 10% of cases after unsuccessful resuscitation[1] by assisted ventilation without endotracheal intubation and in combination with external cardiac massage. Complete gastric rupture has been reported in two cases,[2 3] in both of which the patient subsequently died from unrelated causes. In all reports the lesion has been confined to the lesser curvature, possibly because it is less elastic and has fewer mucosal folds than the rest of the stomach.[4] Mucosal tears or complete rupture appears to occur because of gaseous gastric inflation coupled with external cardiac massage. Gastric rupture, however, has been reported after the use of a nasal oxygen catheter alone, which accidentally passed through the cricopharyngeus.[4 5]

In this case the cardiac arrest and gastric inflation need an explanation. Since the patient had received intravenous diazepam on one previous occasion and methohexitone on three previous occasions without ill effect three possibilities exist:

(1) Excessive pharyngeal secretions irritating the vocal chords may have initiated laryngeal spasm and respiratory arrest, the consequent hypoxia in turn provoking cardiac arrest. When pharyngeal insufflation via a facemask is used in such circumstances air is delivered under pressure to both the vocal cord and the cricopharyngeal sphincter. The reflex activity of the glottis is greater than that of the cricopharyngeus, and therefore the air will simply pass through the cricopharyngeal sphincter and dilate the stomach.

(2) Simple airway obstruction with hypoxia followed by cardiac arrest and attempts at forced ventilation would tend to distend the stomach. The chest was noted to inflate, however, and the patient's cyanosis and general condition improved, so that this explanation is unlikely.

(3) Pharyngeal suction may have induced a "vagal reflex." This, however, would not specifically predispose to gastric distension and is thus unlikely.

Whatever the cause, endotracheal intubation will clearly avert this complication of resuscitation and should be employed as soon as possible.

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Effect of Fibrinolytic Treatment in Malignant Atrophic Papulosis

Malignant atrophic papulosis (Köhlmeier-Degos) is a rare multisystem disease characterized by typical skin lesions and multiple gastrointestinal infarctions. The mortality from the disease is about 60% and it does not respond to either systemic steroid or immunosuppressive treatment.[1] The small blood vessels are conspicuously involved in the disease process and there is impairment of tissue fibrinolysis in the skin lesions.[2] The beneficial effects of phenformin and ethyloestrenol in treating other forms of cutaneous vasculitis[3] prompted us to try this treatment in a patient with malignant atrophic papulosis.

Case Report

A 42-year-old woman presented in 1973 with a pruritic papular eruption of two years' duration. Examination showed about 50 typical Degos papules (up to 0·5 cm diameter) at various stages of development scattered on the trunk and limbs (fig.). She had no gastrointestinal symptoms and her general condition was otherwise normal. Skin biopsy showed a central mucin-containing necrobiotic area in the dermis with endothelial swelling in small blood vessels. Fibrinolysis autography[4] of frozen sections of a Degos papule showed a wedge-shaped area of absent fibrinolysis in the centre, and direct immunofluorescence diffuse fibrin staining around small blood vessels. The euglobulin lysis time was two and a half hours (normal). All other haematological, biochemical, and radiological findings were normal. Throughout the early months of 1974 she continued to develop two or three new lesions weekly. In April 1974, phenformin 50 mg twice daily and ethyloestrenol 2 mg four times daily were begun. A month later the pruritus had stopped and no new lesions had appeared. She was last seen in May 1975, when all the remaining papules had become smaller with atrophic porcelain-white centres. The euglobulin lysis time had remained at two and a half hours.

Comment

We believe this to be the first case of malignant atrophic papulosis in which fibrinolytic treatment has been tried. The response to treatment in the past year has been gratifying in that the formation of new skin lesions has been suppressed. We are aware that this is an uncontrolled observation and that the natural history of the disease may not have been altered. We submit, however, that fibrinolytic treatment is simple and safe and is therefore worth considering in a condition with such a potentially high mortality for which there is at present no effective treatment.

Appearances of three Degos papules on the arm at various stages of development. Late lesion (arrowed) shows typical porcelain-white centre.

We should like to thank Dr. S. C. Gold for encouragement in making this report, and Dr. T. J. Ryan for the fibrinolysis autographs. The case was presented at the dermatology section meeting of the Royal Society of Medicine on 16 May 1974.

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Asymptomatic Urethral Gonorrhoea in Men

The presence of asymptomatic female carriers of gonorrhoea has been recognized for many years and is an important factor in the disease's persistence. The concept of asymptomatic infection in the male was described nearly 20 years ago.[1] Two recent studies at the Norfolk Venereal Disease Clinic, Virginia, showed that about 10% of men exposed to women with culture-proved gonorrhoea harboured organisms in their urethrae without showing signs or symptoms.[2] These men are potentially infectious and are more likely to develop complications,[3] and Hunter Handsfield *et al.*[4] have emphasized that they form a reservoir of asymptomatic carriers. We report here on 24 such patients.

Patients and Methods

Over seven months 24 cases of asymptomatic gonococcal urethritis were seen by one doctor (P.M.P.). None of the 24 patients had symptoms such as discomfort during micturition or urethral discharge. From a public health point of view what the patient perceives as symptoms of gonorrhoea or a sexually transmitted disease is more important than the finding of signs during examination by the physician.