fibrinogen was absent, and a test for fibrin degradation products was positive. Direct spectroscopy of the blood revealed large amounts of oxyhaemoglobin and some methaemoglobin. Schumm's test revealed moderate amounts of methaemalbumin.

Shortly after admission he passed large amounts of fresh blood (approximately 1 litre in all) per rectum, and despite treatment with plasma and intravenous heparin, the patient died. Post-mortem examination showed complete absence of clotting of the blood throughout the body with the exception of the jejunum. The aorta showed haemolytic staining of the intima. The jejunum was bluish in colour and oedematous. There was ulceration of the mucosa and the submucosal blood vessels were grossly dilated and filled with thrombus. Histology showed a poor inflammatory response with large numbers of Gram-positive cocci and Gram-positive bacilli. The remainder of the gastrointestinal tract was normal, and there was no evidence of pancreatitis. The liver was pale with a mottled appearance on its cut surface, and histology showed areas of focal necrosis with vast numbers of Gram-positive rods, thought to be *Cl. welchii*. Both adrenal glands were normal and no abnormality, apart from congestion, was found in the spleen or kidneys.

Because of delay between death and post-mortem examination, the diagnosis of clostridial septicaemia rests on histological and haematological features, as culture of the affected intestine would have been too late after death to provide any useful evidence. Enteritis necroticans, caused by infection with *Cl. welchii* type F,¹ is a condition in which acute regional gangrene of the small intestine occurs, and particularly affects the jejunum. It is highly likely that this man developed enteritis necroticans from contaminated food and this led to disseminated intravascular coagulation and haemorrhage.

I should like to thank Dr. M. G. Lewis and Dr. B. E. Gilliver, Westminster Hospital, for pathological and haematological studies respectively.

—I am, etc.,

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Compression Bandaging for Oedema

SIR,—Your leading article entitled "When should digitalis be stopped?" (20 June, p. 680) raises the importance of the control of oedema of the legs which occurs in about 8% of patients with cardiac failure. Swollen legs are a menace because they are heavy, clumsy, weak, easily fatigued, and cause falls by tripping.

The control of oedema by diuretics is not always simple and raises its own side-effects, but it can be considerably assisted by the application of a compression bandage to the legs, which while it is comfortable and is limiting the swelling is kept on unchanged for 4 to 13 weeks.

Such a dressing requires two readily available materials; first, an elasticized stockinette whose gentle compression will not tire as it reduces the swelling, and, second, a moist non-stretch bandage that ensures fixation and arrests further distension. The stockinette extends from 2 in. (5 cm.) below the tubercle of the tibia to the waist of the foot, and over it the medicated bandage is wound from the knee to the root of the toes.

These are covered by a snug crepe or adhesive strapping 3-4 in. wide (7.5-10 cm.), overlapping each turn by half the width. Some skill in bandaging is needed, but given care and interest it is easily acquired.

By these compression applications, breathless patients with failing hearts and elephant

legs have been kept ambulant and able to go to toilet almost until their death. Their comfort is enhanced, they are better companions, and their nursing requirements are reduced.—I am, etc.,

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Serum Lipids, Typing, Fibrinolysis, and Smoking

SIR,—Recent papers on fibrinolysis (14 February, p. 428, and 16 May, p. 421) and your leading articles on smoking (25 April, p. 190 and 9 May, p. 313) prompted us to point to a relation between the two and to a common relation with coronary heart disease.

Typing the serum lipoprotein patterns of 90 male volunteers, aged 17-75, according to the methods of Stone and Thorp¹ and Thorp,² we found that four subjects had a Fredrickson type I abnormality (two of whom were borderline type V), four others a type IV, and two a type V, corresponding respectively with chylomicronaemia (Bürger-Grütz disease), pre- β -lipoproteinaemia (a carbohydrate-induced hyperlipaemia), and a combination of both chylomicronaemia and pre- β -lipoproteinaemia (carbohydrate plus fat-induced hyperlipaemia).³

Fredrickson's types I and V being rare diseases, it seemed almost impossible that the cases found were true cases, and questions arose about the nature of the "chylomicrons" measured. If these were not true chylomicrons there would be no type I disorder and type V would thus be the

error 0.004). Light smoking has no effect in comparison with non-smoking. The smoking effect is distinct in the group with L particles below 10 mg./100 ml., but not in the group with a larger L fraction (Table). This might point to an inhibition of normal fibrinolysis by smoking, whereas a high L fraction might have its main cause in a defect in the fibrinolysis system. Physical activity also plays a role. The L fraction was lower in the active subjects of the group than in the inactive subjects. The same was true for their serum cholesterol, triglyceride, phospholipid, total lipid, β - and pre- β -lipoproteins, and free fatty acid levels; their fibrinolysis times were shorter.⁴

Type I and V hyperlipaemia being a rare, and a long fibrinolysis time a frequently encountered risk factor for a "coronary," the (simple) procedure of Stone and Thorp¹ may be very useful for a quick detection of insufficient fibrinolysis. Scoring must of course be adapted; arbitrarily it can be said that levels up to 5 mg./100 ml. might be a desirable normal, 10 mg./100 ml. and over a too high level, and fibrinolysis times (euglobulin clot lysis) should preferably be (much) less than 140

TABLE.—Fibrinolysis Times in Minutes Related to the Large Particle Fraction and Smoking (means and ranges). Averages of Duplicate Measurements in each of Two Fasting and Serum Samples taken with an Interval of Two Weeks.

Large Particle Fraction	Non-smokers and Light Smokers*		Medium and Heavy Smokers**	
	Minutes	Number of Subjects	Minutes	Number of Subjects
I. ≤ 5.0 mg./100 ml.	91 (43-163)	13	164 (44-303)	15
II. 5.5-9.5 mg./100 ml.	128 (50-215)	20	160 (75-361)	14
III. ≥ 10.0 mg./100 ml.	182 (116-240)	10	180 (108-304)	18

*Up to 10 cigarettes, 4 cigars, or 5 pipes a day. **More than 10 cigarettes, 4 cigars, or 5 pipes a day.

common type IV. According to Stone and Thorp¹ no true chylomicrons are present in normal fasting blood, and the large particles (L) scattering the light in the nephelometer are probably debris of blood platelets, fibrin threads, etc. Merskey *et al.*⁴ found the amount of fibrin split products to be about 0.8 mg./100 ml., which is a relatively large part of the L fraction we measured. It suggested a positive relation with a long fibrinolysis time, which indeed could be demonstrated (bilateral tail error, Kendall's test about 10^{-3}). It is not known what other substances can be present in the L fraction except for debris of fibrin(ogen), platelets, and cells. With a defective fibrinolysis—more frequently found in survivors of a coronary infarction than in a healthy group⁵—there might be a larger scale of (partial) fibrin breakdown products than in case of slow but normal lysis.

Fibrinolysis is definitely inhibited by medium and heavy smoking (bilateral tail error, Kendall's test 10^{-3}), and this is reflected in the L fraction (Wilcoxon's test, bilateral tail

minutes. However, better criteria should be established.

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