The table shows that the overall incidence of impotence was 23% (19/83) among the diabetics and 20% (10/50) among the non-diabetics. No difference in libido emerged between the groups. Of the impotent men, 10 diabetics and four non-diabetics rarely or never experienced sexual desire whereas nine diabetics and six non-diabetics often did. All impotent subjects aged under 50 had "normal" libido, except for one diabetic aged 45.

Incidence of impotence in diabetic and non-diabetic hospital outpatients

Age (years)	Diabetics		Non-diabetics	
	No of subjects	No impotent	No of subjects	No impotent
15-20	9	0	1	0
21-30	4	1	11	1
31-40	9	1	8	0
41-50	24	3	12	4
51-60	37	14	18	5
Total	83	19 (23%)	50	10 (20%)

Although 19 diabetics admitted to impotence, only three had sought help for this. One, aged 28, had had diabetes mellitus for 12 years with impotence of recent origin associated with severe marital problems. He and his wife subsequently separated and he later attempted suicide. The second, aged 47, had had marital problems during three marriages over 15 years and had received oral testosterone before becoming diabetic. The third, aged 56, had had diabetes for four years. He had been treated elsewhere with testosterone proprionate injections for two years. All 19 impotent diabetics were examined for clinical evidence of autonomic neuropathy. Only one, aged 31, had postural hypotension and a significant reduction in sinus arrhythmia on deep respiration.

Comment

Assessment of impotence is difficult to standardise, so that large discrepancies occur in the reported incidence in diabetics. Joslin regarded impotence as uncommon in diabetics, whereas Rubin and Babbott found that 55% of 198 diabetic men suffered from it.[2] Despite the subjective nature of the disorder and its diagnosis there has been no controlled investigation of the comparative incidence of impotence in diabetics and non-diabetics.

The present study is too small to yield definitive results, but it indicates that the incidence of impotence in diabetic men attending a hospital clinic may be little different from that in other men of the same age living in the same geographical area and attending other hospital outpatient clinics. Thus impotence in diabetics may not necessarily be a specific complication of the disease but may perhaps be related to the general condition of not feeling completely well or at least attending hospital. This would account for the difficulty in showing any relation between the incidence of impotence and the severity of diabetes,[2] diabetic angiopathy,[3] or autonomic neuropathy.[4] A study of 30 diabetics complaining of impotence showed that two-thirds had normal nocturnal erections.[5] The authors concluded that the impotence was psychological rather than organic and wondered why it appeared to be so common in diabetics. Our findings indicate that impotence is equally common in other hospital outpatients. By the loose criteria applied in most studies "impotence" might even be more common in the general (non-hospital) population of this country than in the population studied by Kinsey *et al* with stricter criteria in the USA in 1948, although a detailed investigation would be needed to confirm or refute this.

[1] Kinsey AL, Pomeroy WB, Martin CE. *Sexual behaviour in the human male*. Philadelphia: W B Saunders, 1948.

[2] Rubin A, Babbott D. Impotence and diabetes mellitus. *JAMA* 1958;**168**: 498-500.

[3] Schoffling K, Federlin K, Ditschuneit H, Pfeiffer EF. Disorders of functions in male diabetics. *Diabetes* 1963;**12**:519-27.

[4] Rundles RW. Diabetic neuropathy; general review with report of 125 cases. *Medicine* 1945;**24**:111-60.

[5] Hosking DJ, Bennett T, Hampton JR, Evans DF, Clark AJ, Robertson G. Diabetic impotence: studies of nocturnal erection during REM sleep. *Br Med J* 1979;ii:1394-6.

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Generalised allergy to porcine and bovine monocomponent insulins

Before the introduction of highly purified insulins allergy to insulin was relatively common; local reactions occurred in up to 56% of patients and systemic reactions were found in 0·1-0·2%.[1] Only one report has been published on a generalised allergic reaction to monocomponent insulin.[2]

Case report

A 48-year-old woman with diabetes was treated for 10 years with diet and oral hypoglycaemic agents—apart from two periods, totalling two months, when she was given Lente insulin because of concurrent illnesses. When admitted for surgery to a breast abscess, control of her diabetes was poor so she was treated with soluble insulin; she immediately developed a weal at the injection site and six hours later complained of generalised pruritus. Similar symptoms and signs, together with angioneurotic oedema, occurred with both Actrapid MC insulin (Novo) and monocomponent beef insulin (Wellcome).

The patient had no history of allergy, her eosinophil count was normal, and complement concentrations were normal before and after challenge with Actrapid MC insulin.

Intradermal skin tests using 4 units each of Actrapid MC, Leo neutral, and soluble insulin produced weals of 5-6 cm diameter at all three sites, injections of 0·2 ml each of 0·1% methyl hydroxybenzoate and 0·3% cresol (both preservatives in Actrapid and Leo neutral insulins) caused weals of only 1 cm diameter. There was no reaction to an injection of normal saline.

Total IgE was measured by the paper radioimmunosorbent test (PRIST: Pharmacia) before and after challenge with Actrapid MC insulin and both values were raised at 132 U/ml and 165 U/ml respectively (normal values for non-atopic adults: 122 U/ml).

Specific anti-insulin IgE was detected in appreciable concentrations against soluble, Actrapid MC, and Leo neutral insulins using a radio allergosorbent test (RAST: Pharmacia) (table), and subsequently specific IgE antibody was demonstrated against monocomponent beef insulin by RAST and also by an indirect immunofluorescence technique using fluorescein labelled anti-IgE as described.[3]

IgG insulin antibodies were also present in high concentrations against highly purified beef and pork insulins but not against pro-insulin or other pancreatic hormones.

Specific IgE insulin antibodies (detected by RAST)

Insulin	0·05% human albumin (counts per minute)	Reported patient				Patient positive for insulin antibody	
		Before challenge		After challenge			
		Counts per minute	Ratio	Counts per minute	Ratio	Counts per minute	Ratio
Soluble	400	826	2·1	1064	2·7	225	0·6
Leo neutral	418	2253	5·4	1902	4·6	277	0·7
Actrapid MC	309	3916	12·7	3158	10·2	347	1·1

Counts per minute are expressed as mean of duplicate samples.
Ratio is cpm allergen/cpm 0·05% human albumin.
Patient positive for insulin antibody is a patient with insulin resistance and high concentrations of IgG antibodies but no evidence of allergy.

Comment

Allergic reactions to insulin are mediated by IgE antibodies, and while these have been found in many patients with allergy to standard insulins they were not demonstrated in the only reported case of allergy to monocomponent insulin.[2] Intermittent insulin treatment, as occurred in our case, is an important predisposing cause of insulin allergy, and Davidson *et al*[4] found that 30% of their patients with local reactions and 51% of those with systemic reactions had such a history. In addition they found that 10% of these patients also had insulin resistance; certainly our patient's IgG antibody concentrations were well in the range normally found in such patients.

Zinc has been incriminated as a cause of allergy,[5] but, as with protein impurities in standard insulins, it is thought to cause only local reactions while systemic allergy is due to the insulin molecule itself. Another difference from our patient is that allergy due to zinc is cured by changing to zinc-free insulin. Hormonal contaminants in insulin preparations may stimulate antibody formation but these have not been linked with allergy and, in any case, were not found in appreciable concentrations in this patient.

Insulin allergy is normally managed by changing to a less antigenic

insulin, but if satisfactory control cannot be achieved in our patient, with her present diet and large doses of oral hypoglycaemic agents, the only course would be to attempt desensitisation to insulin.

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[1] Goldstein HH. Allergy and diabetes. In: *Joslin's diabetes mellitus*. 11th edn. Philadelphia: Lea and Febiger, 1971.
[2] Leslie D. Generalised allergic reaction to monocomponent insulin. *Br Med J* 1977;ii:736-7.
[3] Sewell H, Smith DI, Willox A, Barnes CA, Matthews JB. Demonstration of islet cell antibodies. *Lancet* 1980;i:102-3.
[4] Davidson JA, Galloway JA, Peterson BH, *et al.* The use of purified insulins in insulin allergy. Presented at the 34th Annual Meeting of the American Diabetes Association in Atlanta, Georgia (June 14 1974).
[5] Feinglos MN, Jegasothy BV. "Insulin" allergy due to zinc. *Lancet* 1979;i:122-4.

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Spontaneous pseudomembranous colitis

Pseudomembranous colitis is a well recognised complication of antibiotic treatment. In the absence of antibiotics it may occur after surgery and in patients with chronic debilitating illnesses.[1] We report the case of a young man with pseudomembranous colitis in whom no predisposing factors could be identified. We have been unable to find similar cases in English-language publications.

Case report

A previously healthy 22-year-old man presented with a four-week history of worsening abdominal pain and diarrhoea. On admission to hospital he had cramping lower abdominal pain with six watery, malodorous stools a day. The stools contained mucus and small amounts of blood. On examination he was afebrile, but had moderate generalised abdominal tenderness. Sigmoidoscopy showed oedematous, non-friable mucosa with adherent, discrete, grey-yellow plaques. Biopsy of the mucosa showed superficial inflammation and pseudomembrane. A barium enema showed mucosal plaques throughout the large bowel.

Stool cultures for salmonella, shigella, campylobacter, and yersinia were negative as were examinations for ova and parasites. Stools were cultured on CCFA agar (cycloserine, cefoxitin, fructose, and egg yolk) incubated anaerobically, and an ultraviolet fluorescent organism was isolated.[2] The organism was identified by gas chromatography and API-20A carbohydrate utilisation as *Clostridium difficile*. Unfortunately clostridial toxins were not looked for.

Vancomycin 250 mg orally every 8 hours led to complete resolution of the patient's symptoms within 48 hours. After four days of treatment sigmoidoscopy showed complete resolution of the pseudomembrane, with only moderate mucosal oedema. Stool cultures three and six weeks after treatment were negative for *C difficile*. Five months after treatment the patient remained asymptomatic.

Six months before the onset of symptoms the patient had taken an eight-day course of amoxicillin for a penile cellulitis. Several months before this he had been on daily tetracycline for acne. Since the course of amoxicillin he had taken no antibiotics.

Comment

Pseudomembranous colitis is a toxin-induced diarrhoeal disorder usually associated with antibiotic use, surgery, or chronic illness. Profuse watery, non-bloody diarrhoea, abdominal pain, fever, and leucocytosis are common. Grey, white, or yellow plaques on the rectal mucosa are usually seen at sigmoidoscopy. The lesions consist of a fibrinopurulent exudate streaming from the lamina propria. Toxin-producing *C difficile* is usually recovered in cases of pseudomembranous colitis but is rarely found in patients with other bowel disorders.[3] After stopping other antibiotics, recognised treatment consists of supportive therapy and oral vancomycin.

This case of pseudomembranous colitis was in a young man with no history of surgery, systemic illness, or recent antibiotic use. Major symptoms were abdominal pain and watery diarhoea with small amounts of blood. Before sigmoidoscopy pseudomembranous colitis had not been considered. Nevertheless, the diagnosis of pseudomembranous colitis was then established by the sigmoidoscopic appearance, rectal biopsy, and by barium enema. *C difficile* was cultured from the stool. The patient responded promptly to oral vancomycin.

We have reviewed published work and found no other cases of pseudomembranous colitis without predisposing factors. Malcolm[4] reported an elderly lady with pseudomembranous colitis four years after a colectomy for Crohn's disease. There were no other predisposing factors. *C difficile* was searched for but not isolated in his patient.

Our patient took antibiotics six months before the onset of his symptoms, but it is difficult to implicate these as the cause of his illness. We cannot be certain that he did not take any antibiotics, but his history was consistent at all times to all physicians. We have no reason to believe otherwise.

This case report indicates that *C difficile*-induced pseudomembranous colitis may occur in the absence of recent antibiotic use, surgery, or other overt systemic illness. It also underlines the importance of sigmoidoscopy in the evaluation of acute diarrhoea.

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[1] Bartlett JG, Gorbach SL. Pseudomembranous enterocolitis (antibiotic-related colitis). In: Stollerman GH, ed. *Advances in Internal Medicine*. Chicago: Yearbook Medical Publishers, 1977:455-76.
[2] George WL, Sutter VL, Citron D, Finegold SM. Selective and differential medium for isolation of Clostridium difficile. *J Clin Microbiol* 1979;**9**: 214-9.
[3] Larson HE, Price AB, Honour P, Borriello SP. Clostridium difficile and the aetiology of pseudomembranous colitis. *Lancet* 1978;i:1063-6.
[4] Malcolm AJ. Pseudomembranous colitis in a colostomy. *Br Med J* 1978;ii: 479.

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Fatal mumps myocarditis in an 8-month-old child

Myocarditis is a well-documented but rare complication of mumps. Although recovery is usually complete, death several months after the acute infection from heart failure due to the damaged myocardium has occasionally been described.[1 2] We report a fatal case of mumps myocarditis which presented as a "cot death" in a previously healthy baby.

Case report

An 8-month-old girl was found in her pram early one afternoon sweating profusely and with mottled blue patches on her face. She was taken to hospital by ambulance but was dead on arrival. She was being looked after by her grandmother at the time. There was no history of illness immediately before death or previously; she had been bottle fed and had been born after 39 weeks' gestation by spontaneous vertex delivery. After birth she had been initially floppy and irritable, but she was discharged from hospital when 1 week old and was well at follow-up examination six months later. At necropsy the heart was enlarged owing to gross dilatation of all the chambers, and there was a clear yellow pericardial effusion of about 5 ml. No congenital abnormalities of the heart or main vessels were found and, apart from congestion, the other organs appeared normal. Microscopy showed diffuse infiltration of the myocardium by lymphocytes and occasional polymorphonuclear leucocytes (figure) and a mild pericarditis. The parotid glands were not examined but the submandibular glands were examined as part of an