Influenza-related excess mortality in the Netherlands 1989/90

SIR,—The number of weekly influenza-like illnesses, as reported by general practitioners randomly distributed over the Netherlands, started to increase at the beginning of December, 1989, and reached a peak of 54 per 10 000 inhabitants around Christmas. This pattern resembles that experienced in 1988/89 and in earlier epidemics (fig 1). Unexpectedly the Netherlands Central Bureau of Statistics recorded a considerable increase in total mortality in December, 1989, and January, 1990.[1] The daily number of deaths rose from the expected mean of 370 (in the total population) to over 400 during the influenza period and reached 500 in the second week of January (fig 2). Mortality in these 2 months rose by 18%, the increase being most pronounced in people over 80 years old (26%). The excess deaths totalled 4100 (0·3 per 1000 population). In the winters of 1988/89 and 1987/88 no excess mortality was registered. An unexpected excess mortality of 0·5 per 1000 has also been reported in the UK,[2] where it was associated with influenza.

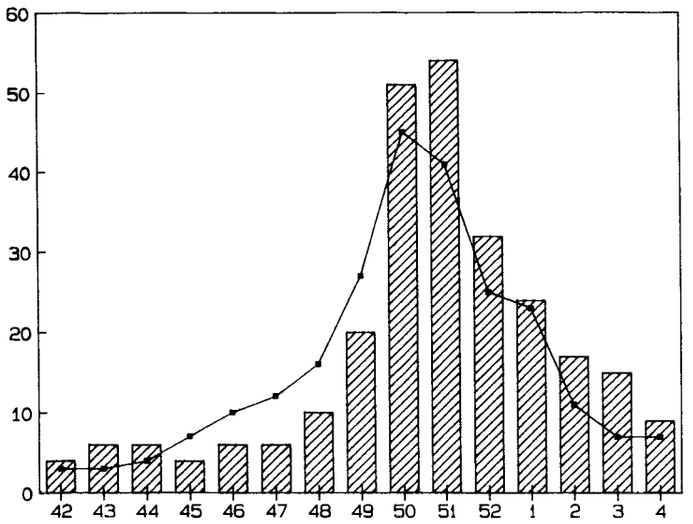

Fig 1—Influenza-like illness per 10 000 inhabitants per week in the Netherlands.

Line = 1988/89, bar = 1989/90 Horizontal axis shows winter week numbers 42 to 4

The predominant influenza strain throughout the 1989/80 epidemic was A/Shanghai/11/87(H3N2)-like. Haemagglutinin[3] and neuraminidase antigenic make-ups demonstrate that this strain is almost identical to the vaccine strain[4] and the epidemic strain of 1988/89. Sera of 100 inhabitants, selected at random, and collected before the epidemic of 1988/89 and another 100 found before the epidemic of 1989/90 were tested simultaneously against vaccine strain A/Shanghai/11/87(H3N2) and against two strains isolated during the two epidemics in the Netherlands. Protection was more prevalent before the latest epidemic than it was before the epidemic of 1988/89:

	Prevalence (%) of protective antibodies to:		
		Epidemic strain	
Year	*Vaccine strain*	*A/Ned/501/88*	*A/Ned/620/89*
1988	3	11	8
1989	10	25	20

Thus in 1989/90 moderate influenza rates were attended by unusually high mortality, an observation that cannot be explained by a major antigenic drift or by lack of antibody. We suggest a change in biological activity[5] as the cause.

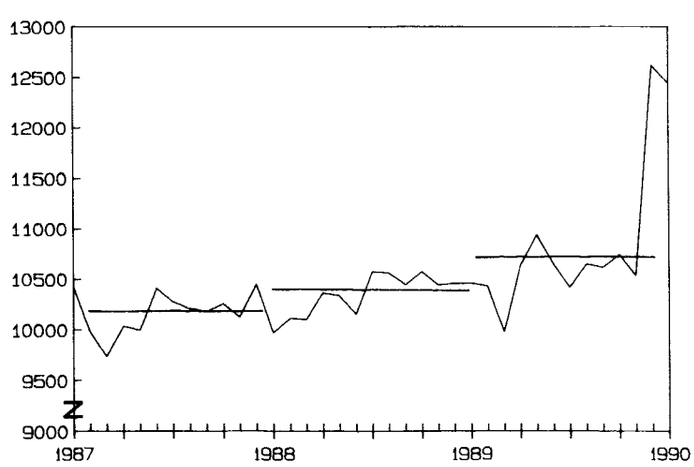

Fig 2—Deaths per month, seasonally adjusted, in the Netherlands.

Horizontal lines = annual averages

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Ventricular arrhythmia in secondary syphilis

SIR,—An upsurge in the incidence of congenital, primary, and secondary syphilis, the great imitator, has been reported lately.[1,2] In his 1934 treatise on syphilis, Stokes[3] noted that despite evidence of myocardial invasion by *Treponema pallidum* the "manifestations of this involvement are among the rarities of experience with early syphilis". We describe ventricular arrhythmias in a previously healthy young woman with secondary syphilis.

A 33-year-old woman with an unremarkable medical history presented with a febrile illness characterised initially by a truncal, non-pruritic, non-scaly, papular rash. After five days she had intermittent palpitations associated with shortness of breath and dizziness without chest pain or syncope. Although her examination and electrocardiogram (ECG) were unremarkable, a Holter monitor demonstrated non-sustained polymorphic ventricular tachycardia with 4–5 beat runs (heart rate 225 beats per minute), frequent couplets, and premature ventricular contractions (over 10 per minute), prompting her admission. She had no abnormalities other than the rash. Evaluation included normal blood counts, serum electrolytes, creatinine, liver function tests, ECG, and chest radiograph. Sedimentation rate was 60 mm/h, and rheumatoid factor, antinuclear antibodies, HIV-1, cytomegalovirus, Epstein-Barr virus, and anti-streptolysin O antibodies were negative. During the first 48 h she continued to have symptomatic episodes of non-sustained ventricular tachycardia. She had normal signal-averaged ECG, cardiac echocardiogram, exercise tolerance test, and stress regional myocardial perfusion study. Her ventricular tachycardia resolved spontaneously. When a sore throat and axillary and cervical adenopathy developed and the rash spread to her limbs, palms, and soles, VDRL (titre 16) and FTA (positive) tests were done. Skin biopsy showed dermal perivascular lymphoplasmacytic infiltrates with endothelial prominence typical of secondary syphilis, but spirochaetes were not identified by Steiner's stain. She was treated for secondary syphilis with 2·4 MU of intramuscular benzathine penicillin with resolution of her symptoms. Her husband also had a reactive VDRL and FTA.