later he was completely well with no evidence of any residual parotid enlargement.

Bilateral parotid swelling in association with hypersalivation has been reported in a 35 year old man with schizophrenia who had been treated with clonazepam and clozapine.⁴ Other drugs associated with parotitis include the butazones, bretylium, asparaginase, methyl dopa, and iodide preparations.⁵ The manufacturers of terbinafine are aware that a 65 year old woman developed parotid swelling and facial oedema after 12 days of terbinafine 250 mg and that this resolved 7 days after stopping treatment (Sandoz, personal communication). By April 1997 the Committee on Safety of Medicines had received three reports of parotid swelling associated with terbinafine, against a background of more than 500 000 patients

having been prescribed the drug (personal communication). We suspect that our case is an example of this rare but potentially alarming side effect. In view of the widespread use of terbinafine, general practitioners and hospital clinicians alike should be aware of this possible cause of parotid swelling.

- 1 Balfour JA, Faulds D. Terbinafine. A review of its pharmacodynamic properties and therapeutic potential in superficial mycoses. *Drugs* 1992;43:259-84.
- 2 Beutler M, Hartmann K, Kulm M, Gartmann J. Taste disorders and terbinafine. *BMJ* 1993;307:26.
- 3 Lowe G, Green C, Jennings P. Hepatitis associated with terbinafine treatment. *BMJ* 1993;306:248.
- 4 Martin SD. Drug-induced parotid swelling. *Br J Hosp Med* 1993;50:426.
- 5 Davies DM. *Textbook of adverse drug reactions*. 3rd ed. Oxford: Oxford University Press, 1985.

Nortriptyline intoxication induced by terbinafine

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We present a case of nortriptyline intoxication secondary to terbinafine treatment.

A 74 year old man with depression was treated with nortriptyline for several months (other drug treatment was cisapride, hydrochlorothiazide, and ranitidine). Serum nortriptyline concentration was maintained at 200 ng/ml for more than 3 months as his depression had recurred when the dose was lowered.

He complained of increasing fatigue, vertigo, loss of energy, and loss of appetite. A few days later he injured himself by falling down a flight of stairs. Nortriptyline concentration was 366 ng/ml and 7 days later was 450 ng/ml. All the symptoms observed could be explained by the increased nortriptyline concentration (therapeutic range 75-150 ng/ml). Terbinafine treatment, started 14 days earlier for onychomycosis, was suspected to be the cause of the increase and was consequently stopped. The nortriptyline dose was lowered from 125 mg to 75 mg a day. Nortriptyline concentration declined from 450 ng/ml to 320 ng/ml 1 week later and to 200 ng/ml 2 weeks later, stabilising at 125 ng/ml after 3 weeks (figure).

A rechallenge test was performed with terbinafine 250 mg daily. Nortriptyline concentration increased to 150 ng/ml on day 4 and to 200 ng/ml on day 7. On day 8 the patient complained of increasing fatigue, vertigo, drowsiness, and a heavy feeling in the head. This suggests that the serum concentration had risen well above 200 ng/ml as the patient had never before experienced such effects at similar concentrations. Terbinafine treatment was stopped and nortriptyline treatment was avoided for 1 day. Nortriptyline concentration decreased to 180 ng/ml on day 12 and to 150 ng/ml on day 15. No

Serum nortriptyline concentrations during first exposure to terbinafine and during rechallenge test

increases in concentrations of γ -glutamyl transferase, aspartate aminotransferase, or alanine aminotransferase were observed. Therefore, we postulate that the increased nortriptyline serum concentration was due to a pharmacokinetic interaction. To our knowledge, this is the first report of a potentially dangerous interaction between nortriptyline and terbinafine.

Correction

Intranasal chlorhexidine resulting in anaphylactic circulatory arrest

Peter Moul, consultant physician at the Whittington Hospital, London, was inadvertently omitted as an author of this drug point (27 September, p 785). He referred the patient whose case is discussed to the National Hospital for Neurology and Neurosurgery, and it was his history taking that pointed to chlorhexidine as a possible causative agent of the severe reaction while the patient was in intensive care there.

One hundred years ago Quackery in Germany

The Kingdom of Saxony is described by a German medical journal as an Eldorado for quacks. The annual report of the Saxon National Medical College for 1896 states that in that year no fewer than 745 quacks, naked and not ashamed, flourished in that happy land. This number was greater by 42 than that given for the previous year. The total number of legally qualified medical practitioners in Saxony for the corresponding period was 1,761, so that approximately there was one quack to every two regular doctors. In certain districts, however, such as Zittau,

Kamenitz, Rochlitz, Annaberg and Glachau, the quacks outnumbered the doctors. Of the 745 quacks, 163 were women. Of the special "lines" of quackery the most numerous represented was "nature healing," which was professed by 220 persons; then came "sympathy," with 106 exponents, homoeopathy with 97, massage with 72, "magnetism" with 46, and tapeworm-curing with 19. One enterprising gentleman makes a speciality of illumination of the cavities of the body with the electric light. (*BMJ* 1898;i:1222)