This observation raises the hypothesis of an intermediary compartment apparently corresponding to the interstitial sector. Thus, our kinetic study is in agreement with the work of Herfst on the interstitial origin of blister fluid (13).

Finally, with respect to Group III, the kinetic study demonstrates that orally administered zinc gluconate does reach the epidermis, but with a 72-hour delay, resulting in a rise in epidermal zinc. This factor points clearly to the problem of the exact role of zinc at the level of the epidermis and its mechanism in therapeutic action. Prospective studies in this area are already under way.

REFERENCES

Cholinergic Urticaria with Anaphylaxis Induced by Exercise or Heating

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A male patient had cholinergic urticaria accompanied by anaphylaxis induced by exercise or a hot bath. Key words: Cholinergic urticaria; Anaphylaxis; asthma. (Received January 30, 1984.)

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Cholinergic urticaria is elicited by warm water, exercise, fever, and emotions (1). It may be associated with systemic manifestations such as abdominal pain, nausea, diarrhoea, headache, and asthma (2, 3, 4). Recently, a small series of patients suffering exercise-induced anaphylaxis have been reported (5, 6). This syndrome comprises urticaria and hypotension, sometimes also accompanied by asthma or cardiac arrhythmias. Warm baths, showers or fever could not induce the anaphylactic symptoms (5). In the present case both exercise and passive heating provoked an anaphylactic reaction.

CASE REPORT

A 21-year-old man with atopic dermatitis and mild asthma developed in relation to exercise symptoms of cholinergic urticaria, comprising pruritus, sweating, flares, hives of varying size (initially few mm urticarial papules with a halo later confluent), and facial angio-oedema.

The symptoms increased gradually over weeks, and further the patient noticed dizziness and a feeling of fainting in relation to the attacks. The patient was admitted for further investigation.

During a hot bath, he developed a severe attack of cholinergic urticaria. Few minutes later he developed asthma, abdominal discomfort, dizziness and loss consciousness. The patient was placed in Trendelenburg’s position. Immediately after, the pulse was weak but frequent and the blood pressure immeasurable.

He was treated with intravenous injections of adrenaline, aminophylline and steroids and he recovered. Prophylactic treatment with oral theophylline was instituted.

Test procedure

In order to evaluate the possible prophylactic effect of long term-treatment with various antimediator drugs, the patient was exercised 11 minutes on a bicycle ergometer with increasing loads to nearly exhaustion. The one-second forced expiratory volume (FEV$_1$) and the blood pressure was used as objective variable.

These exercise tests were performed with an interval of at least 3 days. The first and the last tests were performed without any medication except theophylline. The patient had then multiple exercise tests with varying medications. First he was loaded with a combination of H$_1$ antihistaminic (mepyramine 50 mg 3 times daily), beta-sympathomimetic (terbutaline 5 mg 3 times daily), H$_2$ antihistaminic (cimetidine 200 mg 3 times daily) and anticholinergic (glycopyrrone 2 mg 3 times daily).

After each test one drug was withdrawn in the order listed and the subsequent test was performed 3 days later.

Few minutes after every test, the patient developed symptoms of cholinergic urticaria and asthma. There was no clinical effect of any of the drugs. FEV$_1$ was low before all tests 2.0 l/s ±0.31 (± 1 SD) (minimal normal value 3.2 l/s), increased to 2.28 l/s ±0.37 immediately after the test, and then dropped to a minimum level of 1.7 l/s ±0.27 after 5 minutes.

This sequence was seen in all tests. No drop in blood pressure was observed, and the patient did not complain of dizziness.

The total IgE (blood sample taken at admission) was found to be elevated to 5491 IU (normal range 0-20 IU).

DISCUSSION

Exercise-induced anaphylaxis according to Sheffer et al. (5) is a distinct form of physical allergy differing from cholinergic urticaria by the vascular collapse and the somewhat larger wheals (10-15 mm). They pointed out that increases in the core body temperature, i.e. by fever or a warm bath, could not induce the attacks. Kaplan et al. (6) later described 2 patients who suffered from dizziness, light-headedness, syncopal episodes and hypotension induced by exercise.

These 2 patients both had punctate urticaria lesions typical of cholinergic urticaria. The present case developed urticaria, asthma and hypotension also after a hot bath.

It was not possible to induce the symptoms of dizziness or record a drop in the blood pressure during bicycle-ergometer exercising. However, several authors have noted similar difficulties in reproducing these symptoms in the laboratory (3, 5).
Transient increase in plasma histamin levels have been documented in patients with cholinergic urticaria and exercise-induced anaphylaxis (2, 3, 5), but we found no clinical beneficial effect of H1 or H2 antihistamine drugs. Further no therapeutic effect was seen during treatment with betasymptomimetics and anticholinergics.

The anaphylactic attack provoked by the warm bath was most severe, and could have been life threatening. For this reason we find it important to warn patients with exercise induced anaphylaxis of the possibility of a severe anaphylactic reaction after heating.

REFERENCES

Psoriasis Provoked by β-Blocking Agents

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23 patients suffering from psoriasis and being treated with β-blocking agents were compared to a control group regarding psoriasis activity. Seven out of the 23 were affected by psoriasis after introduction of the β-blocking drug. The mean age of onset was significantly higher (p<0.001) than that of the control group, which supports the provoking effects of β-blocking agents. Remission occurred in 3 out of 4 patients after medication was stopped.

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Ten years ago Ridley (1) reported psoriasiform dermatoses as a side effect of treatment with the β-blocking agent practolol. Two years later Søndergaard and co-workers (2) reported aggravation of psoriasis due to the same drug. Healing or marked improvement occurred, however, when practolol treatment was stopped. In the recent years other β-blocking agents have been reported to induce psoriasiform dermatoses as a side effect (3). Provocation of psoriasis has not been reported. We therefore call the attention to our findings that 7 out of 23 psoriatrics on β-blocking agents got their psoriasis after the drug was introduced.

PATIENTS AND METHODS

23 patients suffering from psoriasis and taking β-blocking agents, were interviewed about activity of their psoriasis in relation to the medication (group 1). As controls served 25 patients with psoriasis not