into two parts—one was retained for bacteriological examination and the other dispatched frozen to the Torry Research Station at Aberdeen for chemical analysis. The total bacterial count was less than 500 organisms/g, consisting largely of coagulase-negative staphylococci. The Torry Research Station reported that the fish seemed in good condition but was very lightly cured (salt/wet weight 0.9%, as opposed to the usual 3% to 5%). The result of the mouse inoculation test for *Clostridium botulinum* was negative. The histamine content of the meat was 1480 mg/l (normal < 50 mg/l).

Comment

The manifestations of scombroid fish poisoning closely resemble those of a histamine reaction and include nausea, abdominal pain, diarrhoea, flushing, headache, rashes, vomiting, palpitations, and pruritus. In severe cases burning of the mouth, dizziness, and respiratory distress have been reported.³ The onset is rapid—a few minutes to a few hours—and recovery is usually complete four to eight hours later. One death only was reported in three series totalling 1660 cases.²

The toxins are formed by the action of contaminating bacteria on fish muscle, which in scombroid fish is particularly rich in histidine. Histidine is decarboxylated by bacterial enzymes to histamine at temperatures between 20 and 30°C.⁴ Fish with histamine concentrations of 100 mg or more/100 g of flesh are usually toxic.⁵ Nevertheless, as Aiso (cited by Halstead²) has shown that 450 to 500 mg of histamine taken by mouth by human volunteers evoked no response at all, the true nature of scombrototoxin remains unknown, though antihistamines have been shown to give symptomatic relief.

The fish referred to in our report may have contained up to 250 mg of histamine. Though few bacteria were found at examination, bacterial growth presumably occurred before and perhaps after the demonstrably inadequate curing.

Prevention is a matter of adequate refrigeration or preservation as soon after catching as possible. Though most affected fish appear normal, those showing any putrefactive changes around the gills or tasting sharp or peppery, whether cooked or uncooked, should be discarded.

We are grateful to the staff of the Department of Microbiology of the Torry Research Station, Aberdeen, for the chemical analysis of the fish.

¹ Kelly, J, *Municipal Engineer*, 14 January 1977, p 52.

² Halstead, B W, *Poisonous and Venomous Marine Animals. II Vertebrates*. Washington DC, US Government Printing Office, 1967.

³ Merson, M H, *et al*, *Journal of the American Medical Association*, 1974, **288**, 1268.

⁴ Geiger, E, *Food Research*, 1944, **9**, 293.

⁵ Simidu, W, and Hibiki, S, *Bulletin of the Japanese Society for Science and Fisheries*, 1955, **21**, 365. Cited by Halstead.²

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Abnormal cerebrovascular regulation in hypertensive patients

Hypertensive patients are known to have an increased risk of cerebrovascular accidents. The reasons for this are not fully understood, although it has been postulated that the normal control mechanisms of the cerebral vasculature may have failed. Patients with chronic hypertension show normal total and regional cerebral blood flow under resting, normocapnic conditions. Nevertheless, the response of cerebral blood flow to a change in P_{aCO_2} is frequently employed as a test of the integrity of the cerebral vasculature.

Patients, methods, and results

Newly diagnosed essential hypertensive patients were considered for inclusion in the study. Hypertension was defined as a mean diastolic pressure

of >100 mm Hg for blood pressure recordings measured on three separate occasions. Patients were excluded if they were known or suspected to be suffering from any other conditions apart from hypertension, or if they were on any form of medication. Ten patients fulfilling these criteria were matched for age and sex with normal volunteers.

Cerebral blood flow was measured by the ¹³³Xenon inhalation method of Wyper *et al*.¹ Details of the procedure have been described fully elsewhere.² Recordings were made at normocapnia and during inhalation of 5% CO₂.

At normocapnia there was no significant difference between cerebral blood flow or P_{aCO_2} in the two groups (table). Under hypercapnic conditions

Comparison of values for 10 hypertensive patients and 10 age-matched normotensive control subjects. Values are means $\pm$ SE of mean. (Mean arterial pressure calculated as diastolic pressure plus one-third pulse pressure)

	Normotensive	Hypertensive
Cerebral blood flow at normocapnia (ml/100 g/min)	45.9 $\pm$ 2.0	46.6 $\pm$ 1.6
Cerebral blood flow at hypercapnia (ml/100 g/min)	55.0 $\pm$ 3.0	46.5 $\pm$ 1.9
P_{aCO_2} at normocapnia (kPa)	5.61 $\pm$ 0.16	5.43 $\pm$ 0.09
P_{aCO_2} at hypercapnia (kPa)	6.77 $\pm$ 0.21	6.36 $\pm$ 0.13
% Reactivity	11.8 $\pm$ 1.8	-2.0 $\pm$ 3.8
Age (years)	48.3	50.0
Mean arterial blood pressure (mm Hg)	91.4 $\pm$ 3.1	134.3 $\pm$ 4.2

Conversion: SI to traditional units— P_{aCO_2} : 1 kPa $\approx$ 7.5 mm Hg.

a comparable increase in P_{aCO_2} was achieved for each group. Cerebral blood flow rose satisfactorily in the normal subjects, but there was a mean fall in the patients. Individually all normal subjects showed a good response of cerebral blood flow to change in P_{aCO_2} , while in only five patients did the blood flow rise and in five it actually fell in response to hypercapnia. Analysis by Fisher's exact test shows this difference to be significant ($P=0.016$).

Cerebrovascular reactivity was calculated as a percentage using the formula: % Reactivity = [Exponential (log change in CBF/(change in P_{aCO_2}) - 1) $\times$ 100]. A significant difference between the two groups was found, with $P<0.02$ (Mann-Whitney test).

Comment

These results indicate significant impairment of cerebrovascular reactivity to CO₂ in a group of hypertensive patients. Previous studies have given conflicting results in hypertensive patients.³⁻⁵ Nevertheless, in Tominaga's study⁵ several subjects had either concomitant medical problems or were taking various medications.

Novack *et al*³ measured cerebral blood flow by the nitrous oxide method, which reflects total cerebral blood flow, while the method we used measures mainly flow in cerebral grey matter. Iliff *et al*⁴ showed an abnormal response to hypocapnia, but gave little clinical information about any concomitant disease. The results of our controlled study receive support from other work we have performed, in which a further 13 (unmatched) hypertensive patients with no known vascular problems showed similar findings.

Various anatomical changes occur in the cerebral vasculature of the hypertensive patient. The mechanisms whereby cerebral vessels respond to changes in P_{aCO_2} are controversial, but our results indicate that there may also be a functional disorder in these vessels. Possibly this might be important in the pathogenesis of strokes, and it is of interest that the same functional abnormality has been reported in diabetic patients.²

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¹ Wyper, D J, Lennox, G A, and Rowan, J O, *Journal of Neurology, Neurosurgery, and Psychiatry*, 1976, **39**, 141 and 147.

² Dandona, P, *et al*, *British Medical Journal*, 1978, **2**, 325.

³ Novack, P, *et al*, *Journal of Clinical Investigation*, 1953, **32**, 696.

⁴ Iliff, L D, *et al*, in *Blood Flow and Metabolism in the Brain*, ed by A M Harper *et al*, pp 11-29. Edinburgh, Churchill, 1975.

⁵ Tominaga, S, *et al*, *Stroke*, 1976, **7**, 507.

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