sympathetic paralysis due to oedema and dilatation of the wall of the internal carotid artery, and the narrowing of the carotid syphon seen at angiography[4] supports this notion. A "cold spot" over one eye was reported in two of five cases studied by thermography, which is consistent with reduced flow in the internal carotid artery and its ophthalmic branch, which supplies the area of the forehead. The presence of adjacent "hot spots" suggests that external carotid dilatation may contribute to the pain.[5] Anthony and Lance[6] have recently claimed that in a migraine attack the plasma serotonin level falls and plasma histamine is unchanged, whereas in a cluster headache the serotonin level is unchanged and the whole blood histamine is raised.

The current concept of cluster headache has therefore moved from a migraine variant to a distinctive clinical picture in which local histamine release could explain both the observed vascular effects and the pain. What remain obscure are the initial release mechanisms and the cause of the periodicity, so that if histamine is to be accepted as the rogue of the piece further information is needed.

The diagnosis is based on a carefully taken history, which should exclude migraine, trigeminal neuralgia, sinusitis, and psychogenic facial pain. In the occasional difficult case oral nitroglycerin will reproduce an attack within 30 to 60 minutes during a cluster.[7] Radiographs and angiography have no place if the history is adequate. Symonds[8] introduced treatment with ergotamine, which is much more effective than it is in migraine. It is given one hour *before* the expected attack by intramuscular injection (0·25-0·5 mg) or by suppository (1-2 mg) as prophylaxis. Success depends on accurate observation of the timing and may necessitate two or three doses per day for the duration of the cluster. Treatment should be omitted for one day every week to see if the cluster has ended. Patients who do not respond to ergotamine may be relieved by methysergide, 3-6 mg per day, or by pizotifen, 1·5-3 mg per day, for the duration of the bout.

In few conditions are patients so grateful for relief of their symptoms.

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Gastric ulcers and achlorhydria

"The idea that the secretion of gastric acid is necessary for the production of ulcer of the stomach is by no means new . . . although not worked out by exact methods," wrote Ewald[1] towards the end of the last century. The pithy dictum "no acid—no ulcer" was formulated by Schwarz in 1910.[2] Its truth has rarely been questioned since. Many studies have reported the invariable presence of gastric acid in patients with chronic peptic ulcer. Conversely, the absence of such ulcers in patients with pernicious anaemia has strengthened the concept that acid is necessary for the development of gastric ulcers. Support for the truth of the dictum is available from other sources. When radiation therapy is used to induce achlorhydria in patients with peptic ulcer, healing occurs, and there is no recurrence of ulceration so long as achlorhydria persists. Acute superficial mucosal ulcers may be found in patients with achlorhydria, but these lesions do not become chronic and they apparently heal rapidly.[3]

Until the introduction[4] of the maximal stimulus to secretion of acid in 1953 tests measuring acid secretion were of limited value. It is true that, when standard but submaximal doses of histamine were used, some patients were reported with benign gastric ulcers who were apparently achlorhydric. In fact, hypochlorhydria is a common accompaniment of chronic gastric ulceration and reflects the mucosal lesions of gastritis. If a maximal stimulus had been used, whether in the form of histamine, ametazole hydrochloride (Histalog), or pentagastrin, some acid would have been found in these patients—or so it has been assumed.

More recently, and at a time when the usefulness of measuring the secretion of gastric acid in patients with gastric ulcers has been questioned,[5] a few patients have been described with benign gastric ulcers in whom there has been achlorhydria after maximal stimulation. Wald and Burbige[6] gave details of two such patients and a case had previously been recorded by Isenberg *et al.*[7] The acid studies were performed punctiliously and repeated on several occasions preoperatively, each time failing to show the presence of titratable acid. The ulcers were examined endoscopically, and in two of the three cases detailed histological sections became available after surgical resection, confirming that there was no evidence of malignancy. The gastric mucosa of one patient was reported as atrophic, and in another biopsies showed atrophic gastritis and a greatly reduced number of parietal cells.

This combination of benign gastric ulcer and achlorhydria, if genuine, must be an extreme rarity. It is just possible that the methods used on these patients were not sufficiently sensitive to induce or detect acid secretion. Patients have not so far been described in whom pentagastrin was used as the stimulus, nor in whom a continuous infusion of histamine was given. Whereas there is little difference in the results from giving histamine or pentagastrin by the same route, the one-shot technique gives an acid output only about 90% of that obtained by continuous infusion of histamine.[8] Alternatively it might be postulated that gastric ulceration might be present in a patient with only transient achlorhydria. It has been suggested that sufficient back diffusion of hydrogen ions through an extensively damaged mucosa might take place to render particular acid secretion tests invalid. Whatever the explanation, accounts of such patients are likely to remain few. Endoscopic examination and biopsy of gastric ulcers have now largely superseded the performance of acid secretion studies in clinical diagnostic practice. If acid studies are performed in patients with gastric ulcers, the finding of achlorhydria after a maximal stimulus should signify that the ulcer is almost certainly, though perhaps not inevitably, cancerous. The cases recently described may represent genuine exceptions to Schwarz's dictum, but further examples are necessary before its absolute validity is finally questioned.

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