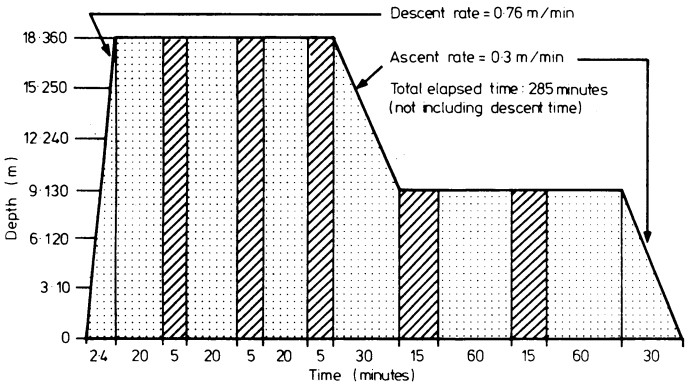
Spinal decompression sickness with delayed onset, delayed treatment, and full recovery

Decompression sickness is caused by nitrogen bubbles formed in the body of divers and people working with compressed air who leave a compressed environment too fast. The bubbles most commonly afflict the joints, skin, brain, spinal cord, and lungs. Decompression sickness, if untreated, may cause permanent functional damage and is potentially fatal. We report an unusual case of decompression sickness affecting the brain and spinal cord in which neurological symptoms and signs first appeared five hours after the dive. Symptoms were partially relieved by oxygen, dextran 40, and dexamethasone but disappeared fully only during hyperbaric treatment.

Case report

A 20-year-old healthy male diver worked at 20 metres for 40 minutes and ascended with one three-minute decompression stop at three metres. Two hours later he complained of headache, sore throat, and malaise, and five hours later of pain in the left knee and left elbow, headache, and confusion.

On examination his temperature was 38·5°C and a mild follicular tonsilitis was found. There were no other abnormalities. Two hours later sensory loss in the right thigh, mild paraparesis, and loss of sensation around the anus developed. He received 20 mg dexamethasone intravenously, 500 ml dextran 40, and sodium lactate (Hartmann's solution); oxygen was given by mask. Two hours later he was admitted to the Israeli Naval Hyperbaric Institute fully conscious and co-operative but unable to stand. Neurological examination showed hypoaesthesia in the L3 dermatome on the right, patellar reflex weak bilaterally, Achilles reflex normal bilaterally, abdominal reflexes missing, and severe paraparesis. Laboratory findings were normal, as were findings on chest and spinal radiography and electrocardiography. He was put into a recompression chamber and compressed to 18 metres breathing 100% oxygen (according to US Navy table No 6, (figure)). After 35 minutes he showed signs of improvement. After three hours 20 minutes at a depth equivalent to nine metres neurological examination showed no abnormalities.



US Navy therapeutic recompression table 6 (reserved for severe decompression sickness). Stippled areas represent periods of breathing oxygen; hatched areas represent periods of breathing air.

Comment

The unusual characteristics of this case were the long delay preceding the onset of symptoms[1] and the rapid and complete relief of symptoms on recompression despite the delay in starting treatment. The onset of spinal symptoms was probably facilitated by dehydration owing to fever, with resultant haemoconcentration and high viscosity and coagulability. So-called "silent bubbles" exist in the body of a diver even after dives made within accepted limits. Small numbers of bubbles are usually cleared in the lungs without producing symptoms. The bubbles may, however, be coated with thrombocytes.[2] Aggregation of other blood elements follows, and vessels are progressively occluded by clots surrounding bubbles.[3] Venous drainage of the spinal cord is in the form of a large, valveless venous lake in which the flow is normally sluggish and therefore vulnerable to obstruction by bubbles.[4]

Our patient was treated with oxygen, dexamethasone, and dextran 40. Rehydration was achieved with sodium lactate. Normobaric oxygen enhances dissolution of bubbles[5]; dexamethasone helps relieve oedema in the brain and spinal cord; dextran inhibits aggregation of thrombocytes and improves microcirculation; and sodium lactate expands blood volume and thus improves circulation and helps to relieve ischaemia and mobilise trapped bubbles. Recompression to 283·7 kPa (2·8 atmospheres) diminishes bubble size and thus relieves circulatory obstruction. Oxygen at high pressure improves oxygenation of ischaemic tissue and relieves oedema.

Hyperbaric treatment is the primary and specific treatment for decompression sickness, even when there is considerable delay after the accident. Pharmacotherapy may be of benefit. The prognosis of paralysis due to decompression sickness is poor without treatment.

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Prolactin-like immunoreactivity in human small-intestinal mucosa

Several peptide hormones are present in both the brain and the gastroenteropancreatic endocrine system.[1] The possibility that prolactin is another "brain gut" peptide was suggested by the persistently raised serum concentrations of prolactin (>1000 mU/l) found in some patients with coeliac disease[2] with no other obvious cause for the hyperprolactinaemia.[3] We sought prolactin-like immunoreactivity in small-intestinal biopsy specimens using a simple technique for showing gastroenteropancreatic endocrine cells.

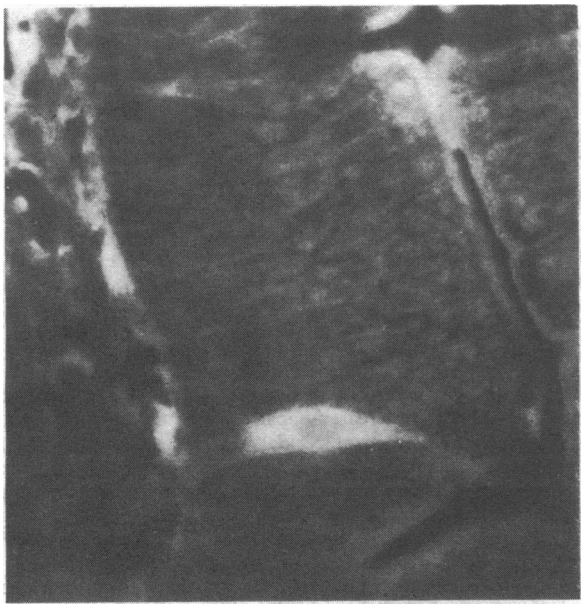
Methods and results

Small-intestinal biopsy specimens were obtained with a Watson capsule from the distal duodenum and the proximal jejunum in patients undergoing investigation for possible coeliac disease. The specimens were orientated on monofilament nylon gauze with the luminal surface uppermost, placed immediately in modified Susa fixative, and examined under a dissecting microscope (modified Susa fixative consisted of 76 mmol (12·5 g) trichloroacetic acid, 43 mmol (2·5 g) sodium chloride, 100 ml 40% formaldehyde, and 25 ml glacial acetic acid, made up to 500 ml with distilled water). The period of fixation varied from a few hours to two weeks with no resultant loss in peptide hormone immunoreactivity.

The fixed tissue was washed in several changes of 70% ethanol and processed as per routine paraffin technique. Serial 5 μ sections were cut from paraplast-embedded tissues, dewaxed, and rehydrated in phosphate-buffered saline pH 7·4 with resultant unmasking of immunoreactive sites. Sections were stained using an indirect immunofluorescence technique with rabbit antihuman prolactin as primary antibody (Tenovus Cancer Research Laboratories, Cardiff) and swine antirabbit IgG conjugated with fluorescein isothiocyanate as secondary antibody. The specificity of this antibody was examined with respect to human growth hormone and adrenocorticotrophic

hormone in human anterior pituitary tissue and to gastrin, gastric inhibitory polypeptide, glucagon, motilin, somatostatin, secretin, and vasoactive intestinal peptide in serial sections of human upper small-intestinal mucosa.

Cells reacting with prolactin antiserum were found in biopsy specimens of small-intestinal mucosa from both patients with and patients without coeliac disease. The cells were located in the crypts of Lieberkühn with occasional cells in the lateral epithelium of villi or on the luminal surface of specimens from patients with coeliac disease. The figure shows a prolactin-immunoreactive cell from the neck of a crypt in a specimen obtained from a patient without coeliac disease. It is a typical intestinal endocrine cell with a long apical process reaching to the luminal surface. Fluorescence was located fairly uniformly throughout the cells apart from the centrally located nucleus. Background fluorescence appeared to be within blood vessels rather than nerves.



Single prolactin cell in epithelium of normal human jejunal villus showing immunofluorescence. Modified Susa fixation. ×400 (original magnification).

Specificity studies on human anterior pituitary tissue showed that the prolactin antiserum stained a different population of cells from that stained by primary antisera to human growth hormone or adrenocorticotrophic hormone. In the small-intestinal mucosa prolactin staining was considerably reduced by prior incubation of Tenovus antiserum with human prolactin, whereas when gastrin, gastric inhibitory polypeptide, glucagon, motilin, secretin, somatostatin, and vasoactive intestinal polypeptide were used no reduction in the intensity of fluorescence was observed. In addition, serial sections stained with primary antisera to the gastroenteropancreatic hormones showed populations of cells whose distribution or morphology or both did not correspond to the prolactin immunoreactive cells.

Comment

Using an antiserum directed against human pituitary prolactin we showed cells containing prolactin immunoreactivity in human small-intestinal mucosa. The immunoreactivity was intracellular though extranuclear and was typical of that seen in other populations of cells of the intestinal endocrine system. The distribution of the staining was unlike the "internalised" prolactin seen within the Golgi apparatus of hepatocytes.[4] The cellular distribution in the gastrointestinal tract has yet to be determined.

Isolation and characterisation of gut prolactin immunoreactivity should allow specific antibodies to be raised for radioimmunoassay and ultimate elucidation of its biology in man.

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Intraspinal opiates and itching: a new reflex?

Facial itching is a common, unwelcome, and unexplained side effect of intraspinal opiates. Opiates release histamine from mast cells in body tissues, but it is not known whether histamine is wholly or even partly to blame for the itching referred so constantly and so specifically in the distribution of the trigeminal nerve. There is mounting evidence in favour of a central, enkephalinergic mechanism for some kinds of itch. Itching induced by butorphanol, a morphine-like analgesic that does not raise blood histamine concentrations, can be prevented by naloxone.[1] Naloxone has also proved successful in the treatment of generalised itch in patients with liver disease.[2,3] We report on three patients in whom facial itching provoked by intrathecal opiate responded to intravenous naloxone.

Case reports

Two women aged 35 and 41 years were scheduled for elective cholecystectomy. Both received 1·0 mg intrathecal morphine sulphate in 0·1 ml saline isobaric with cerebrospinal fluid at the L3-L4 interspace 30 minutes before induction of general anaesthesia.

On recovery from anaesthesia two hours after receiving the intrathecal morphine the first patient scratched her nose vigorously and complained of facial itching, most pronounced in the nostrils. Naloxone 0·4 mg intravenously rapidly (two minutes) abolished the itch but not the analgesia. The second patient complained of nasal itching three hours after the injection of intrathecal morphine. Naloxone 0·4 mg intravenously provided immediate, complete, and permanent relief, analgesia being unaffected.

A third patient also underwent cholecystectomy. She was given 1·0 mg diamorphine hydrochloride intrathecally and complained of facial itching five hours later. Intravenous naloxone 0·4 mg successfully abolished the itch.

Comment

Our results support the view that a central enkephalinergic component may play a part in opiate-associated itching. They do not so readily explain how intraspinal opiates consistently induce itching referred in the distribution of the trigeminal nerve. We suggest that this phenomenon may be neural in origin, the result of a spinal reflex transmitted through a medullary itch centre associated with the spinal nucleus of the trigeminal nerve.

In some animals there is evidence of a "scratch centre," which lies in the caudal part of the floor of the fourth cerebral ventricle.[4] In man itching of the nostrils is an "absolutely specific" clue to the presence of a cerebral tumour that has infiltrated the floor of the fourth ventricle.[5]

Like the substantia gelatinosa of the spinal cord—where intraspinal opiates act to produce analgesia—the spinal nucleus of the trigeminal nerve is rich in opiate receptors. At the level of the third or fourth cervical segment of the cord the spinal nucleus and tract of V become continuous with and indistinguishable from the substantia gelatinosa and the tract of Lissauer. Cells in the grey matter of the dorsal horn have further, indirect access to both trigeminal nuclei by spino-trigeminal interneurones that travel in the anterolateral quadrant of the cord (D Bowsher, personal communication). These might be