compared, using paired t tests, with those who were current non-smokers, individually matched for age (to within 5 years), sex, and diagnosis. One hundred and eight matches were successfully obtained. Epidermal growth factor output for smokers was 64% (44·0 to 92·2%) of the value for non-smokers ($p<0·05$) in 60 control pairs and 43% (28·3 to 64·8%) of the value for non-smokers ($p<0·01$) in 48 pairs of patients with ulcers.

Comment

Our data show that smoking is associated with reduced salivary secretion of epidermal growth factor and an increased prevalence of peptic ulceration in patients attending for endoscopy. Cigarette smoking could thus predispose to peptic ulcer by depressing salivary secretion of epidermal growth factor. Depression of submandibular epidermal growth factor concentration in a small group of patients with gastric and duodenal ulcers has been reported.[4] Our data suggest that reductions in epidermal growth factor secretion in patients with ulcers are likely to be attributable to the higher proportion of patients with ulcers who are smokers.

Smoking is associated with depressed prostaglandin synthesis, while nicotine can enhance platelet aggregation, vasoconstriction, and increased concentrations of circulating catecholamines.[5] Carbon monoxide may depress oxygen transport.[5] However, neither the mediator nor the mechanism by which smoking depresses production of salivary epidermal growth factor is clear. Epidermal growth factor is produced by Brunner's glands in the duodenum, as well as by a cell lineage which develops at the site of gastrointestinal ulceration. If smoking also depresses local mucosal synthesis or secretion of epidermal growth factor this might represent an additional factor predisposing to duodenal ulceration as well as a plausible explanation for the link between smoking and Crohn's disease.

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Safety of subaqua diving with a patent foramen ovale

Stephen J Cross, Sian A Evans, Lesley F Thomson, Hai Shiang Lee, Kevin P Jennings, Thomas G Shields

Department of Cardiology, Aberdeen Royal Infirmary, Aberdeen AB9 2ZD
Stephen J Cross, MRCP, *registrar*
Hai Shiang Lee, MRCP, *registrar*
Kevin P Jennings, FRCP, *consultant*

Hyperbaric Medicine Unit, Aberdeen Royal Infirmary
Sian A Evans, BM, *senior house officer*
Lesley F Thomson, MB, *senior house officer*
Thomas G Shields, FRCP, *consultant*

Correspondence to:
Dr Cross.

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Patent foramen ovale may be a risk factor for developing some forms of decompression sickness.[1,2] Venous nitrogen bubbles are thought to evade the pulmonary filter by passing through these intracardiac shunts and to form emboli, which enlarge to cause symptoms. Should potential and current divers be screened for the presence of a patent foramen ovale, and if one is found should they be prevented from diving?

Subjects, methods, and results

Seventy eight subaqua divers who had not had any form of decompression sickness were examined by contrast echocardiography.

Microbubble contrast was generated in 8-10 ml of agitated normal saline by the two syringe and three way tap method.[3] This contrast medium was injected rapidly into a right antecubital vein while an apical four chamber view of the heart was being observed with a Vingmed CFM 700 imaging system.

Up to six injections of contrast medium were given: three during quiet respiration and three during the strain phase of a Valsalva manoeuvre. A patent foramen ovale was said to be present if contrast medium was seen in the left heart within five cardiac cycles of the right heart becoming opaque. The procedure was stopped if a patent foramen ovale was shown or after the sixth injection.

Twenty four divers were found to have a patent foramen ovale and another two late (presumed intrapulmonary)[4] shunts. These divers were recreational (16), professional (six), or a mixture of the two (four). The professional group comprised police (two), biologists (four), fish farmers (two), and ex-Royal Navy divers (two) (table).

All subjects claimed in general to control their decompression profiles by using conventional tables or decompression computers. Nine of the professional and five of the amateur divers admitted to having performed dives with factors possibly associated with an increased risk of developing decompression sickness.

The diving record of one former professional diver in particular was notable. He had been diving for 30 years, including some time as an experimental diver with the Royal Navy. He had a large shunt demonstrable on the first injection of contrast medium—that is, without a provocative manoeuvre having been performed. He had performed 637 dives with compressed air to a maximum depth of 60 m, 141 closed circuit oxygen dives, 327 dives with nitrogen-oxygen, 14 surface oriented dives to 100 m with helium-oxygen, and 47 bell dives to 180 m with helium-oxygen. He had spent 1743 days in saturation at an average depth of 200 m (maximum 330 m). All this was in addition to a "large number" of emergency mine disposal dives (recorded in a missing log book). He had not experienced any form of decompression sickness.

Comparison of professional and amateur divers with demonstrable right to left shunts. Values are means (SD)

	Professional (n=10)	Amateur (n=16)
Age (years)	35·9 (7·1)	32·7 (8·2)
No of years diving	12·8 (10·9)	7·5 (5·8)
No of dives	650 (446)	236 (316)

Comment

As patent foramen ovale might be related to some forms of decompression sickness,[1,2] the question arises whether these shunts are relevant in subjects who have not had decompression sickness.

Although the dive profiles tended to vary between this particular group of professional and amateur divers (the professional divers tended to perform shallower multiple dives, with frequent ascents for any one dive), most had a substantial exposure to diving over several years. None had decompression sickness. Nevertheless, we found a 31% incidence of patent foramen ovale (or a 33% incidence of right to left

shunt) in this group. This compares favourably with a postmortem study of normal hearts that showed the incidence of patent foramen ovale to be about 27%.[5]

We conclude that the presence of a right to left shunt in a diver without a history of decompression sickness may be irrelevant. On current information, there is probably not a case for the routine screening of divers (or, indirectly, potential divers) for shunts.

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Plantar power: reproducibility of the plantar response

J Maher, M Reilly, L Daly, M Hutchinson

Department of Neurology, Adelaide Hospital, Dublin 8
J Maher, MRCPI, *senior house officer*
M Reilly, MRCPI, *registrar*
M Hutchinson, FRCP, *consultant neurologist*

Department of Public Health Medicine and Epidemiology, University College, Dublin
L Daly, PHD, *senior lecturer*

Correspondence to:
Dr Hutchinson.

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Babinski's sign is an established part of neurological examinations, regarded widely as the key clinical sign of pyramidal tract disturbance. The specificity and sensitivity of the sign have never been studied as there is no standard test to indicate both the anatomical and the physiological integrity of the descending pathways. There is, none the less, much evidence linking the presence of the sign with pure corticospinal tract lesions.[1,2]

To be of practical diagnostic value all clinical signs must be both valid and reproducible. In view of the inherent difficulties in documenting the validity of Babinski's sign we examined its reproducibility by assessing the intraobserver and interobserver variation in its detection by 24 physicians.

Patients, methods, and results

Twelve patients with multiple sclerosis (five), cerebral infarction (two), spondylotic myelopathy (two), Wilson's disease (one), diabetes mellitus (one), and epilepsy (one) were chosen for the study. Twenty four physicians (seven consultants and 17 experienced non-consultant hospital doctors) were divided into six groups of four. Each group member examined the same two patients (four plantar responses) on two separate occasions, making a total of 192 examinations. The extent of agreement within each group of four physicians in the first examination of two patients was assessed (interobserver variation). Intraobserver variation was based on comparison of the first and second examinations of the four plantar responses by the 24 physicians. Patients were screened so only their feet were visible, and physicians were told that they had to examine four patients (rather than two patients on two occasions as was the case). Each plantar response was graded as upgoing or downgoing; an equivocal response was not accepted.

For statistical analysis the κ statistic was calculated. This is a measure of agreement and can be interpreted as the percentage agreement beyond chance. A κ value of 100% implies perfect agreement between observers, a value of 0% agreement due purely to chance. A κ for interobserver variation was calculated in each of the six groups of four physicians using the results from the first examination. The overall κ was based on averaging these six estimates. For intraobserver variation a κ statistic was calculated for each of the 24 physicians based on the duplicate interpretations of the four plantar responses. These 24 κ values were averaged to give an overall measure of intraobserver variation. Details are to be found in Fleiss.[3] Results were expressed as 95% confidence intervals.

Interobserver variation—In only 50% of the examinations was there total agreement between the four doctors (table). The average percentage agreement beyond chance was 16·7% (0·4% to 33·0%).

Agreement between physicians on nature of plantar responses (interobserver variations) and by each physician examining four plantar responses on two occasions (intraobserver variation)

Interobserver variation		Intraobserver variation	
Extent of agreement between four physicians	No of plantar responses	No of plantar responses that agreed	No of physicians agreeing
Complete	12	4	14
3/1 split	7	3	6
2/2 split	5	2	3
		1	1
Total	24	Total	24

Intraobserver variation—Fourteen physicians agreed completely with their assessments of all four plantar responses on the two separate occasions (table). The κ statistic averaged over the 24 physicians was 59·6% (39·6% to 79·6%).

Comment

Our data indicate that experienced physicians often disagree about the nature of the plantar response. Though intraobserver consistency was somewhat more impressive, our results clearly call into question the clinical value of this established sign. Other explanations for our findings are that (*a*) the physicians were not experienced in eliciting the sign or (*b*) the plantar responses in our patients were intrinsically variable. However, we deliberately chose physicians with some years of clinical experience, all of whom were members or fellows of a royal college, and the patients studied were those from a neurology ward who were deemed by us to have definite plantar responses of one or other type.

A high frequency of extensor plantar reflexes, without other pyramidal tract signs, has been reported in a population of elderly patients with psychiatric disease.[4] McCance *et al* noted that the reproducibility of the sign was poor and cautioned against overemphasising its value in this age group. Nathan and Smith counselled that the plantar reflex should be interpreted only as part of the complete physical examination[5] and our study reemphasises the difficulty in basing clinical decisions on any single physical sign.

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