Retinal angiography in divers

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Abstract

Objective—In the light of previous reports, to examine the possibility that professional diving might cause abnormalities of the retina and choroid.

Methods—The retinal fluorescein angiograms of 55 Royal Naval divers and 24 non-diver servicemen were compared.

Results—No differences were found between divers and non-divers and the prevalence of abnormalities was not correlated with diving experience.

Conclusion—In contrast to previous reports, there seem, at least in naval personnel, to be no ocular consequences of diving.

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Polkinghorne *et al* examined the ocular fundi of an unspecified mixture of 81 sport and commercial divers and 22 non-diver controls by means of fluorescein angiography.¹ The divers had significantly more retinal pigment epithelial defects and the prevalence of defects increased with the number of years of diving experience and with histories of decompression illness. Capillary anomalies and a reduction in capillary density at the fovea were also found in divers. No visual deficit was identified as a consequence of these changes. Holden *et al*² compared 26 sport divers with more than 10 years' experience with seven divers with only one year's experience. The prevalence of retinal pigment epithelial defects was the same in both groups and a trend towards more vascular abnormalities in the experienced diver group (12 of the 26 seasoned divers compared with one of the seven novices) was not significant. It was suggested that the conservativeness of the diving carried out by this group might have avoided the ocular effects noted by Polkinghorne *et al*.¹

In the context of diving, transient ischaemia seemed the most likely cause of the ocular changes recorded.¹ The retinal changes in divers were of concern in view of the possibility that they might be paralleled by brain damage corresponding with that suggested by psychometric, electroencephalographic, and histological studies.^{3–6} The presence of "silent" bubbles within the carotid circulations of divers decompressing conservatively from innocuous dives⁷ and especially from saturation dives⁸ is well recognised. The concept of

arterial gas embolism in the pathogenesis of cerebral decompression illness is consistent with the finding of transient gas bubble emboli in the retinal and choroidal circulations of monkeys who had been subjected to decompression stress.⁹ These were associated with electroencephalographic slow wave changes similar to those reported in humans in association with decompression illness and after apparently normal dives.^{10 11}

Methods

Royal Naval male clearance divers and non-diver servicemen from the Portsmouth area were identified from pay records. Men with a history of serious systemic, ophthalmic, or neurological non-diving related illness were excluded. Of the 56 divers and 24 controls entered into the study, one diver was unable to tolerate the procedure and was excluded from the analysis. The divers and controls were of similar age (mean (range) 33 (22–55) *v* 36 (22–54) years) and duration of naval service (16 (6–38) *v* 18 (4–39) years). Twenty three of the divers had participated in non-service commercial or sport diving and 13 had shared 16 episodes of relatively mild or transient diving related illness; non-neurological decompression illness (8), gas poisoning (6), dysbaric osteonecrosis (1), and otic barotrauma (1). Twenty divers had participated in saturation diving.

Each volunteer completed a questionnaire detailing his medical and diving history. The information from divers' log books was entered into a computerised data bank for dive exposure analysis. Data were collated by workers who had no knowledge of participants' diving status.

Visual acuities were recorded with a Snellen's chart and visual fields were analysed with a Friedman field analyser. Colour vision was assessed with the 15 plate Ishihara test.

Pupils were dilated with 1% tropicamide and 10% phenylephrine for colour photography and fluorescein angiography. The negatives were analysed by two independent macula specialists who were blind to subject status. Each recorded the material as normal or abnormal in terms of retinal vascular defects and retinal pigment epithelial defects. The results were compared and those cases in which there was disagreement were resolved by consensus.

The procedure for capillary density measurement in the parafoveal region was taken from the method of Polkinghorne *et al*.¹ The

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Fluorescein angiogram of a diver showing the mottled hyperfluorescent appearance of a retinal pigment epithelial defect.

angiogram negative taken at the time of maximal capillary filling was projected on to a circular grid and the number of capillary intersections through its equally spaced eight radii was recorded independently by two physicians. The scores were averaged. Angiograms with clear capillary detail at each of the radii were accepted. This resulted in the rejection of 25 diver and 13 non-diver records in this part of the study. In the resulting smaller group the divers ($n = 30$) and controls ($n = 11$) were of similar age (mean range 32 (23–51) *v* 33 (23–48)) and duration of naval service (15 (5–35) *v* 13 (3–32)).

Table 1 Number of subjects with retinal pigment epithelial defects by number of dives performed

	Controls	Divers (number of dives)			All
		<300	300–449	>449	
Normal	18	16	14	11	59
Abnormal	6	5	4	5	20
All	24	21	18	16	79
% Abnormal	25	24	22	31	25

Table 2 Estimates of regression coefficients in logistic regression of abnormality rates

Term	Estimate	Standard error of estimate	t Value
Constant	-1.096	0.993	-1.10
Age	-0.0038	0.0297	-0.13
Number of dives	0.00057	0.00124	0.46

The horizontal and vertical diameters of the foveal avascular zones in the 30 divers and 11 non-divers were measured concurrently with the determination of capillary density by the two independent physicians. The size of the foveal avascular zone was calculated by assuming it to be oval in shape. The mean of the two assessments was taken.

STATISTICAL METHODS

The relation between rates of abnormality (retinal pigment epithelial defect) and explanatory variables—for example, age, number of dives performed—have been investigated by logistic regression modelling.¹² For example, a logistic model may relate the probability (P) of an abnormality to age and number of dives as follows:

$$\log (P/(1-P)) = a + b (\text{age}) + c (\text{number of dives})$$

where a is a constant and b and c are the regression coefficients for age and number of dives which are to be estimated.

Regression methods have been used in the analysis of capillary counts and foveal avascular zones.¹³

Unless otherwise stated in the results section, significant results are at the 5% level.

Results

VISUAL ACUITIES AND FIELDS

All subjects had corrected visual acuities of 6/9 or better in each eye except for one control and three divers who had astigmatism and whose acuities were no worse than 6/18, 6/9. Visual fields were normal in all the controls and divers. All were able to recognise 15 out of 15 colour plates bilaterally except for two non-divers. One of these failed one plate with each eye and the other passed two out of 15 with one eye and three out of 15 with the other.

RETINAL PIGMENT EPITHELIAL DEFECTS

Six (25%) of the 24 controls and 14 (25%) of the 55 divers had retinal pigment epithelial defects. None of the divers or non-divers had microvascular defects.

The overall rates of abnormality (retinal pigment epithelial defect) for controls and divers (table 1) were similar ($\chi^2 = 0.42$, 3 degrees of freedom, $P > 0.9$).

Logistic regression analyses, incorporating age and number of dives as explanatory variables, showed no significant variation in abnormality rate when the variables were considered jointly or singly. Table 2 shows the estimates of regression coefficients of age and number of dives when both variables were included in the logistic model.

The rates of abnormality (retinal pigment epithelial defect) were similar for the 24 controls, 20 divers who had saturation diving experience, and 35 divers who had no such experience (table 3).

Logistic analysis of the abnormality rates with age as an explanatory variate showed the relations for three groupings—24 controls, 20 divers with saturation diving experience, 35

Table 3 Number of subjects with retinal pigment epithelial defects according to their participation in saturation diving

	Controls	Divers		All
		Non-Saturation	Saturation	
Normal	18	26	15	59
Abnormal	6	9	5	20
All	24	35	20	79
% Abnormal	25	26	25	25

divers without saturation diving experience—which were not significantly different and abnormality rates were not related to age. By a similar method of analysis, no significant differences in abnormality rates were found between the 24 controls, the 13 divers with histories of diving related illness, and the 42 divers without histories of diving related illness (table 4).

There was no evidence that abnormality rate was related to having participated or not participated in non-service diving (table 5).

An overall logistic analysis of abnormality rates with linear additive terms representing diver, saturation diving, and previous history of diving related illness indicated no significant factors.

CAPILLARY DENSITIES AND FOVEAL AVASCULAR ZONES

The mean (SD, range) total number of capillary intersections through eight radii for the 30 divers who underwent capillary counting was 54.8 (9.1, 37.5–75.5) and for the 11 non-divers was 58.4 (10.6, 45.5–78.0). A small difference existed between the capillary scores of the two independent assessors (mean (SEM)

difference of 1.6 (0.5)). The mean vertical and horizontal diameters of the foveal avascular zones were 0.47 mm and 0.46 mm respectively for the divers and 0.48 mm and 0.45 mm for the non-diver controls. These approximate to the foveal avascular zone diameter of 0.5 mm given by Polyak.¹⁴ The mean (SD, range) foveal avascular zone areas for the 30 divers was 0.17 (0.06, 0.05–0.34) and for the 11 non-divers was 0.17 (0.05, 0.09–0.26). No significant difference was found between foveal avascular zones computed from the sets of radii from each of the two independent assessors (mean (SEM) 0.002 (0.002)).

The mean of the two independent assessors for capillary densities and foveal avascular zones for each man have been used in obtaining the following results. Analyses by regression methods indicated that the number of capillaries was not significantly related to age or the number of dives performed. With or without allowance for age, no significant differences were found between the mean numbers of capillaries in the 11 controls and the 30 divers. Within the 30 divers, the mean number of capillaries for the 13 saturation divers and the 17 non-saturation divers was not significantly different. Only one significant factor was found when the linear additive model included only indicator (yes = 1, no = 0) terms for diver, participation in saturation diving, participation in non-service diving, and possession of a history of diving related illness (table 6 gives estimated coefficients associated with indicator terms).

The model indicated a higher expected number of capillaries in divers with a history of diving related illness compared with divers without such a history. The expected number of capillaries in non-divers was nearer to the expected value for divers with a history of diving related illness.

With regression methods, foveal avascular zone areas were not found to be significantly related to age or number of dives performed. No significant factors were found when the regression model included age and the indicator (yes = 1, no = 0) terms for diver, participation in saturation diving, participation in non-service diving, and the possession of a history of diving related illness. In the model with age and the indicator all regression estimates were less than one standard error of their estimates.

Discussion

This study, like that of Polkinghorne *et al*,¹ involved a modest number of divers and controls, a limitation that would tend to favour the emergence of negative findings. However, the suggestion that diving is associated with retinal abnormalities and a reduction in capillary density at the fovea¹ was not supported. The greater capillary density in the group with histories of diving related illness has little biological plausibility.

It is difficult to reconcile the retinal studies of divers performed so far. The methodology for this study was deliberately based on that of

Table 4 Number of subjects with retinal pigment epithelial defects according to possession of history of diving related illness (DRI)

	Controls	Divers		All
		Non-DRI	DRI	
Normal	18	33	8	59
Abnormal	6	9	5	20
All	24	42	13	79
% Abnormal	25	21	38	25

Table 5 Number of subjects with retinal pigment epithelial defects according to their participation in service diving only (Ser) or service and non-service diving (non-ser)

	Controls	Divers		All
		Ser	Ser + non-ser	
Normal	18	24	17	59
Abnormal	6	8	6	20
All	24	32	23	79
% Abnormal	25	25	26	25

Table 6 Estimates of regression coefficients in logistic regression of abnormality rates

Term	Estimate	Standard error of estimate	t Value
Constant	58.41	2.70	21.60
Diver (yes)	-6.86	3.67	-1.87
Saturation diving (yes)	4.72	3.50	1.35
Non-service diving (yes)	-3.34	3.63	-0.92
History of diving related illness (yes)	8.83	3.77	2.34

Polkinghorne *et al*¹ for ease of comparison of the two investigations. Although there were fewer divers than in the other study,¹ they were a more homogenous group. Controls, being servicemen of comparable position and experience with similarly high levels of general health to the divers, were likely to be more representative than those of the other study.¹ In our study population the lifetime dose of the proposed hazard was available from divers' logs in terms of the number of dives or dive hours accumulated, whereas in the study of Polkinghorne *et al*¹ this was achieved only crudely in terms of years of diving experience. The quantity and pattern of diving performed by the naval divers and by subjects in the other study may have differed. Although their means of assessing experience was crude, it is unlikely that subjects performed more dives or dived deeper than the population reported here. Furthermore, the absence of a dose effect among the naval divers makes it unlikely that the different results of the two studies could be ascribed to a relative lack of exposure in this group. Although the amount of non-service diving performed by the naval divers admitting to it was small, the absence of positive findings in this group adds further contrast with the other study, which investigated a mixture of sport and commercial divers. The main difference between the data from the two studies is the higher prevalence of changes in the control group. Although an explanation for this is not apparent, the finding does not detract from the conclusion that in this group diving is not associated with lesions in the ocular fundus.

Service diving has a reputation for safety and incidents of decompression illness are perhaps less likely to pass unrecorded than in commercial practice where income is more tenuous. Recreational diving is less well regulated than professional diving and some of its practitioners indulge in manifestly unsafe procedures. If Royal Naval diving is accepted as

being characterised by an emphasis on safety and conservativeness, our findings would support the contention of Holden *et al*² that adherence to sound well established diving practices may protect against the ocular fundus lesions reported by Polkinghorne *et al*.¹ Furthermore, as the ocular and cerebral vasculature are closely related, the cerebral vasculopathy reported in divers⁶ might also be avoided.

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