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PHYSIOLOGY

Liebreich's Sign for Defective Colour Vision among Artists

LIEBREICH[1] pointed out that in a London exhibition of 1871 a painting showed roofs an oxen red on the sunny side but green where shadowed. He suggested that it indicated that the painter was a red–green colour vision defective. Angelucci[2] called this "Liebreich's sign" when he described the works of six painters known to be red–green defectives, whose pictures showed this characteristic. Trevor-Roper[3] and Kalmus[4] also mention Liebreich's sign.

I have examined a number of paintings by the red–green defective artists mentioned elsewhere[5–8], and by six of the art students discussed later[9], together with paintings by seven more art students. The resulting data are shown in Table 1, each type of defective being noted separately. Of the ninety-eight pictures which did not show Liebreich's sign twelve had no shadows because they were abstract designs. Liebreich's sign is not more often shown by deutans than by protans because the observed difference is not statistically significant.

Table 1

Type	Artist No.	No. of pictures	Liebreich's sign +	Liebreich's sign −
DA	5	39	6	33
EDA	5	25	4	21
D	1	5	2	3
Totals	11	69	12	57
PA	2	19	0	11
EPA	5	17	7	10
P	1	12	1	19
Totals	8	48	8	40
Undiagnosed	1	1	0	1
Grand totals	20	118	20	98

Among six paintings reproduced in colour by Rabkin[10], which are copies by red–green defectives of other pictures also shown, only Plate 4, by a deuteranomal, and Plate 6, by a protanomal, show Liebreich's sign. In Plate 10 a green shadow in the original has been more emphasized in the copy by a protanomal, while in Plate 12 green shadows of the original have become brown in the copy by a deuteranomal.

It appears that Liebreich's sign is sometimes present in the paintings of red–green colour vision defectives, but, at least in contemporary artists and students of art, it is too infrequent to be a reliable guide.

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Effect of Laparotomy on Plasma Renin Activity in the Rabbit

WHILE trying to find an effective stimulus for the release of renin in the rabbit, we inserted a catheter into the left lumbo-adrenal artery and pushed the tip into the left renal artery close to the kidney. With no further manipulation the plasma renin activity increased within a few hours and subsequently fell to normal or less than normal values during the next few days. This unexpected observation encouraged us to investigate the changes in plasma renin brought about by opening the abdomen.

Eight rabbits were anaesthetized with 30 mg/kg of sodium pentobarbitone given intravenously. A 4-in. incision was made in the midline of the abdomen and the peritoneum was opened. The incision was covered with gauze swabs soaked in warm saline. After 20 min the abdomen was closed in two layers, and blood samples were taken by puncture of the central artery of the ear. Three samples were taken from each animal: after anaesthesia but before incision; 4 h after incision; and 48 h after the first sample. The volume of blood taken was 4·5 ml. with 30 U of heparin/ml. to prevent clotting. A normal diet with free access to water was allowed before and after operation. For comparison arterial blood samples were taken from four rabbits at time intervals which were the same as for the operated animals, but these rabbits were not anaesthetized nor were they operated on. A further four rabbits were anaesthetized and blood samples were taken at similar intervals.

Plasma was separated by centrifugation and assayed by a method described in more detail elsewhere[1]. Each sample of plasma was treated in the following way: 2 ml. of plasma was added to 1·0 ml. of soybean trypsin inhibitor (1 mg/ml.). To this mixture was added 1·0 ml. of 4×10^{-2} molar disodium ethylene diamine-tetraacetic acid (EDTA), and 0·1 ml. of 4×10^{-1} molar dimercaprol, which was in arachis oil. Soybean trypsin inhibitor and EDTA were in 0·1 molar sodium phosphate buffer, *p*H 6·0, containing chlorhexidine gluconate, 0·01 per cent w/v. The EDTA, dimercaprol and chlorhexidine inactivate plasma angiotensinases, and soybean trypsin inhibitor prevents the formation of plasma kinins. To this buffered plasma mixture was added sufficient renin substrate to give a final substrate concentration of 900–1,000 mμg/ml. The final mixture was incubated for 6 h at 42° C. The supernatant collected after snap freezing or heating at 100° C was assayed to find the concentration of angiotensin.

Renin substrate was prepared from plasma of rabbits 48 h after bilateral nephrectomy. Ammonium sulphate was added to the plasma to 60 per cent saturation. The precipitate was separated by centrifugation and redissolved in a quarter the volume of distilled water. This