Photoelectric plethysmography
Ocular blood flow measurements in dogs

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Ocular blood flow in dogs was monitored with the use of a specially designed photoelectric plethysmograph transducer. The ocular pulse waves demonstrated were (1) of low amplitude, (2) coincident with cardiac systole, and (3) rapidly affected by stimuli known to influence cerebral blood flow. Changes in ocular pulse amplitude correlated with concurrent blood gas analyses (arterial *CO₂* and *O₂*). Hypercarbia and hypoxia increase the ocular pulse amplitude, while hypocarbia and hyperoxia decrease the ocular pulse amplitude. Variations in ocular pulse amplitude measured by photoelectric plethysmography may mirror changes in cerebral and ocular blood flow.

Key words: ocular pulse, photoelectric transducer, reflectance photometry, ocular blood flow, ocular plethysmography, carotid and cerebral blood flow.

Most of our knowledge concerning ocular blood flow has been gained from indirect methods of measurement, since direct methods are rarely feasible.¹ Photometric estimations of ocular blood flow have been performed for many years.²⁻⁵ Several authors have described methods for detecting the ocular pulse with the use of the photoelectric plethysmograph,⁶⁻⁸ but little is known about the actual significance of this pulse. The eye, supplied by the first branch of the internal carotid artery, must certainly mirror cerebral blood flow. Reported here are some controlled studies on dogs which are an attempt to correlate the height of the ocular pulse with stimuli known to affect the cerebral vasculature.

Materials and methods

Medium-sized mongrel dogs (approximately 15 to 30 kilograms) were used in this series of experiments. As far as could be determined, all were normal and healthy and lacked any apparent ocular abnormalities.

The apparatus consisted of an ocular plethysmograph transducer, plethysmograph amplifier, pressure transducer, and a two-channel Sanborn Twin-Viso recorder (Model No. 60-1300 B).⁵ The ocular photoelectric plethysmograph (P.E.P.) transducer used for the detection of the ocular pulse waves by photometrics consisted of a special corneal contact lens mounting (approximately 18 mm. in length, 12 mm. in diameter, and weighing less than 4 Cm.) with a self-contained light source (Chicago Miniature No. 7153, 5 volts), a photosensing cell (Clairex-CL 903 L), and a low vacuum attachment for suction when desired (Fig. 1). The 5 volt incandescent light source was driven at 2.7 volts to decrease heat emission. The spectral response curve of the photosensor peaks at 7,350 Å, a desirable sensitivity for hemoglobin study. The signal amplifier used was specially designed with

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Fig. 1. Ocular photoelectric plethysmography (P.E.P.) system utilizing a corneal contact lens with self-contained light source and photosensor.

12 fixed-gain positions and a separate power source for the light in the ocular P.E.P. transducer.

The instrumentation utilizes the optical principle of reflectance photometry in which both incident and reflected light pass through the dilated pupil. Diffuse light passes through the contact lens into the eye and interacts with the retinal and choroidal vasculature by being absorbed, transmitted, or reflected. It is the reflected light that is sensed by the photocell (Fig. 1). The photocell is most sensitive in a spectral zone of minimal absorption by hemoglobin. Incident light is either reflected directly from the vascular space or transmitted to the sclera, from which it is then reflected back through the vascular medium. Transitory changes in blood flow through the retinal and choroidal vessels lead to variations in the density of the reflective medium (i.e., hemoglobin). As the photosensor acts as a densitometer, a momentary volume-pulse increase (at systole) results in an increased ocular pulse amplitude as measured by increased output of the photoelectric system. Recorder deflection is thus a function of change in light reflectance.

Dogs were anesthetized with intravenous sodium pentobarbital (30 mg. per kilogram) and intubated. One pupil was dilated with two drops of 1 per cent cyclopentolate solution.

The femoral artery was catheterized and connected to a Statham pressure transducer with a three-way stopcock arrangement for blood pressure monitoring and arterial blood sampling for gas analysis. The ocular P.E.P. transducer was then placed on the dog's cornea. Baseline levels of arterial oxygen concentration ($O_2$), carbon dioxide concentration ($CO_2$), pH, peripheral pulse pressures, and ocular pulse amplitudes were then taken. The pulse amplitude in all instances was measured in millimeters of vertical recorder deflection at a fixed gain.

To measure the effects of increased $CO_2$ (hypercarbia) and relative hypoxia, a rebreathing system was established. Dead space was increased 130 ml. by adding a length of polyethylene tubing to the dog's endotracheal tube. During a four-minute rebreathing cycle, $CO_2$, $O_2$, pH, ocular pulse amplitudes, and peripheral pulse pressures were measured at 30 seconds, 1, 2, 3, and 4 minutes. The dog was then removed from the rebreathing system and allowed to breath ambient air. The same variables were then monitored for another five minutes.

To measure the effects of increased $O_2$ (hyperoxia) and hyperventilation, each dog was connected to a respirator (Bird Corp., Mark 7) and administered 100 per cent oxygen at 20 to 25 respirations per minute for five minutes. During this five-minute period, the pertinent variables ($CO_2$, $O_2$, pH, ocular pulse, and peripheral pulse pressure) were measured at 30 seconds, 1, 2, 3, 4, and 5 minutes. The respirator was then disconnected, the animal was allowed to breath ambient air, and the same variables were again monitored for another four to five minutes. The effects of hypercarbia and hyperoxia were induced several times to demonstrate the reproducibility of the data.

Results

Representative data are illustrated in Figs. 2 and 3. During hypercarbia and hypoxia, as the $CO_2$ increased, the ocular pulse amplitude was observed to increase markedly (approximately 200 per cent) (Fig. 2). Immediately upon removing the dead-space tubing, there was a sharp decrease in pulse amplitude (approximately 80 per cent) coincident with the increasing $O_2$ (Fig. 2). Due to the manner in which the dog was made hypercarbic (i.e., by increasing anatomical dead space), not only was $CO_2$ elevated, but $O_2$ was reduced significantly—indicating concurrent hypoxia.

During the hyperoxia-hyperventilation procedure, the magnitude of change of the ocular pulse was not as large ($\approx$ 30 per cent), but as the $O_2$ increased ($CO_2$ decreased), the amplitude of the ocular pulse decreased (Fig. 3). When the respirator was disconnected and the animal allowed
to breath ambient air, the ocular pulse increased approximately 125 per cent, while the \( \text{PO}_2 \) decreased (Fig. 3). Response patterns were clear and reproducible.

Ocular pulse amplitude varied directly with \( \text{PCO}_2 \) and inversely with \( \text{PO}_2 \). The observed changes in ocular pulse amplitude showed little if any latency after \( \text{PCO}_2 \) and \( \text{PO}_2 \) changes, indicating a direct and in-phase relationship to the stimuli influencing blood flow.

The ocular pulse wave form obtained during a typical hypercarbic-hypoxic procedure is shown in Fig. 4. The wave form and amplitude of the ocular pulse varied also with eye movement, respiration, and the position of the P.E.P. transducer on the cornea.

**Discussion**

Total cerebral blood flow tends to remain relatively constant. The principal local regulatory mechanism maintaining the integrity of the cerebral blood flow involves \( \text{PCO}_2 \) in the blood and brain tissue. Only in severe hypoxia does the \( \text{PO}_2 \) play a more important role. A rise in the \( \text{PCO}_2 \) will cause an immediate vasodilatation, leading to a decrease in cerebral vascular resistance and an increase in flow rate. In hypocarbia, as expected, the response is in the reverse direction. A fall in \( \text{PCO}_2 \) results in vasocon-
Fig. 3. Timebase: ON indicates hyperventilation-hyperoxia phase; OFF indicates recovery phase with ambient air. (Top) Arterial carbon dioxide (\(P_{CO_2}\)) and oxygen (\(P_{O_2}\)) tension during hyperventilation-hyperoxia and recovery; (middle) ocular pulse amplitude (millimeter of vertical recorder deflection) and peripheral pulse pressure (millimeter of Hg), (systolic minus diastolic) during hyperventilation-hyperoxia and recovery; (bottom) arterial pH during hyperventilation-hyperoxia and recovery.

Ocular pulse height is related directly to the effects of stimuli influencing cerebral blood flow; increased flow corresponds to an increased ocular pulse.

The ocular pulse amplitude is both a qualitative and a semiquantitative monitor. It is not an absolute value and varies with position, respiration, eye movement, and from subject to subject. When the baseline value for a particular subject is known, however, variations in the ocular pulse amplitude during any given stimulus are quantifiable in terms of percentage change.

Ocular F.E.P. may have several impor-
Fig. 4. Ocular pulse wave form (typical) during hypercarbia-hypoxia obtained from a dog's eye with the use of the P.E.P. transducer. Pulse amplitude (millimeter of vertical recorder deflection at a fixed gain) vs time (recorder speed 2.5 cm per second).

Important clinical applications. It may allow inferences about the carotid system, ocular vasculature, and intraocular pressure. Study of the ocular pulse wave contour may lend itself to diagnosis of certain cardiovascular conditions. Finally, ocular P.E.P. will allow direct monitoring of the patient undergoing a procedure which transiently compromises cerebral and ocular blood flow.

Mr. Harold Balyoz constructed the electronic equipment.

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