THE FUNDUS OCULI AND THE DETERMINATION OF DEATH *

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Pathologists and clinicians alike are made aware all too frequently of the need for a simple and readily available method for determining whether death has occurred. While a condition of "suspended animation" does not exist in the literal sense, respiratory and circulatory activity may be so reduced in both rate and intensity as to escape detection by the usual procedures. Because the possibility of resuscitation may be at stake, time may not permit the use of the electrocardiograph or similar apparatus. In addition to establishing the fact of death, reasonably accurate determination of the moment of death may be of prime significance in the subsequent adjudication of the descent of property.

It has long been known that complete cessation of blood flow to the bulbus oculi results in rapid and striking morphologic changes in the retina and its vessels. These changes were first studied by Bouchut[1] (1863), who advanced them as constituting the earliest and most accurate sign of death. Many subsequent investigators have corroborated this assertion. Several, in addition, have either intimated or stated emphatically that such changes unequivocably indicate cardiac standstill; and thereby have shifted attention somewhat from their mortal significance to the possibility of their serving as a guide to resuscitability.

It is my purpose to emphasize, as others have done previously, the importance, speed, accuracy, and ease of observation of the fundus oculi; to summarize all preceding investigations; to illustrate photographically the appearance of the retina during and after death; and to correlate the illustrated fundic changes with simultaneous electrocardiographic tracings.

Changes in the Fundus with Cessation of Cardiac Activity

Three cardinal changes, occurring more or less concomitantly but at different velocities and with different degrees of intensity, comprise the so-called ophthalmoscopic signs of death. In reality, these are actually signs of cessation of blood flow to the bulb and, inferentially, of cessation of cardiac output.

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Disappearance of the Central Retinal Artery and Its Branches. As the circulation slows down with gradual failure, the arterial blood columns become "granular"[2,3] due to visibility of clumped, rapidly moving erythrocytes. They may eventually form "beads," or become "striated" at the instant of heart standstill; at the same time the caliber becomes markedly reduced. As a rule the arterial pattern disappears rapidly, perhaps within the following minute or less,[4-6] although it is reported to have persisted for much longer periods with the initial onset of peripheral arteriolar haziness.[7-10] Arterial blood, when seen, remains brighter than that in the veins.[8]

Color Changes in the Retinal and Choriodal Coats. The retina and choroid normally have an intense reddish color due to the rich network of choroidal capillaries which can be seen easily through the transparent retina. At the instant when circulation stops, the choroid becomes pale yellow-orange,[7-11] and the optic disk, normally a pale rose color,[5] immediately becomes ghastly white and more sharply outlined against the now paling retina.[5,8] Grayish retinal turbidity begins to appear rapidly,[8,9] at first in the posterior pole of the globe around the disk, gradually progressing to the periphery where some red tinting may be perceptible even hours after death. The macula appears as a red spot on a field of delicate gray-yellow, then gray-white, and deep gray after several hours; the amount of pigment, which varies with race and individually, determines the degree of yellow or brown overlay.

Retinal Venous Changes. As in the arteries, circulatory deceleration results in "granularity" of the blood columns flowing through retinal veins. Almost without exception these columns break into segments ("beads,"[2] "striations"[3]) within a few seconds after circulatory stagnation.[2,5,6] Such formations are seen chiefly in the larger veins on or near the disk,[8] although the site is variable.[3] The segments and intersegmentary clear spaces continue to move smoothly for approximately 10 to 20 minutes after their evolution[6] and finally become completely static. They remain visible in this fashion for several hours. During this period they usually return to their original shape and position after external pressure upon the globe, which causes only temporary blanching.[3] Hours later the final picture is one of magenta colored venous segments on a uniformly gray retina which shows no trace of an artery. These striking vascular phenomena, especially the rapidly occurring venous segmentation, constitute the less subtle and more objectively cogent components of the ophthalmoscopic triad.

Historical Review

Just 1 year after Helmholtz invented the ophthalmoscope, the first documented studies on mortal retinal changes were conducted by E. Bouchut,[1,12,13] (1863) who, under the pseudonym of Pierre Durand, received a prize offered by the Marquis d'Ourches for the most nearly infallible sign of death. He stressed rapid disappearance of retinal arteries, the uniformly pearl-gray fundus, and the so-called pneumatosis or segmentation of venous columns, which results, in his opinion, from immediate post-mortem release of gases present in the blood. He supported this contention by referring to frequent gaseous segmentation of meningeal vessels observed at necropsy as manifesting a generalized somatic phenomenon. These findings were reported by him to the Academy of Science, Paris, in 1864 and first published in 1868 ("A Memoir on Some New Signs of Death Furnished by the Ophthalmoscope"). Bouchut[1] also published another brief account in 1876 together with the earliest extant illustration, a single colored drawing reproduced here as Figure 8.

Meunier[14] (1868) and Poncet[15] (1870) verified the above observations. The latter, working independently and ignorant of Bouchut's efforts,[1,4] noted absence of the characteristic signs in syncopic patients. By 1873 many observers had become sufficiently skilled in ophthalmoscopy to observe the intra-ocular alterations at death. On the other hand Weber,[16] in 1876, noted persistence of intact arterial blood columns along with venous blood segmentation. Becker,[17] too, noted full arteries. Gayet[7] studied eyes immediately after decapitation and observed orange-yellow fundi (not described by Bouchut), thin arterial remnants, and irregularly occurring peripheral "beading" of venous blood. Also, he reported that in two bodies which had remained for several hours at a low temperature (6° to 10° C.) after death, the retinal vessels appeared normal. He therefore concluded that retinal vascular signs are not pathognomonic of death. Doe[18] insisted that they are.

Usher[2] (1896), working with humans, monkeys, dogs, cats, and rabbits, correlated not only the "granularity" of retinal circulation with a generalized circulatory deceleration, but also the eventual "beading" of venous blood and the disappearance of arteries with complete cardiac standstill. He was the first to state that "granular movement," a result of ophthalmoscopically magnified (20 ×) individual and clumped red cells, does not necessarily mean death, and that recovery is possible. Absence of fundic signs in an excessively

chloroformed, apparently dead monkey, which recovered unexpectedly, served to emphasize his point. He devised experiments to implicate a relative increase in intra-ocular pressure as a cause of "beading"; and he insisted, contrary to Aldridge,[19] that retinal arteries empty retrogradely after death.

In the face of conflicting opinions of contemporaries, Gayet[7] (1900) concluded that further study led to less dogmatism about infallibility of the signs and that ". . . the former condition of the patient's health, certain individual peculiarities, the mode of death, and, finally, the surroundings in which the body is kept . . ." are all factors which influence the significance of any ocular sign. Gowers[20] (1904) briefly mentioned the signs and cited Gayet's work.

Albrand,[8] in 1904, emphasized the purely mortal aspects of retinal changes and presented the detailed descriptions of opacification of the media as well as of retinal color changes after death. He stated that arterial blood, which usually disappears quickly after death (within 5 minutes at most, but usually much earlier), is of lighter color than venous blood which tends to become gradually darker as tissues usurp liberated oxygen. Irregularly narrowed veins persist for hours, with coagulated segments seen early in the physiologic depression, separated by fluid and by fine strands of erythrocytes. He included colored drawings of each eye of a cadaver at 45 minutes and at 6 hours after death, the former showing persistent, narrowed arteries and veins (segmented on the disk) on a yellow-orange fundus; the latter, only two dark venous trunks near the papilla upon a gray background. Albrand asserted that these changes allow for conclusive determination of onset of death, time of death, position of the body at death (congestion producing a lingering red tinge in the more dependent eye), and the type of death involved, either rapid or slow. His criteria for the last point are questionable.

In 1906 Albrand-Sachsenberg[9] placed emphasis on the use of funduscopic signs in determining resuscitability. If even the feeblest heartbeat and a barely vital circulation are present, regardless of all appearances of death, the characteristic funduscopic changes do not occur; arterial volume is usually markedly reduced and the arteries are traversed by pulsation waves coincident with heartbeats. Such may be the case with severe anemia, syncopal conditions, pathologic sleep, and electrocution (which often produces an ominous, immediate, but transient and completely reversible state of apparent death). Albrand-Sachsenberg argued that it is the duty of every physician who examines corpses to differentiate real from apparent death with the

ophthalmoscope, to institute resuscitative measures if indicated, and to obviate by frequent examinations the premature abandonment of resuscitative efforts or the remote possibility of premature interment. Although funduscopic signs may vary in detail from case to case, he was convinced that the over-all picture is sufficiently distinct, even with severe concomitant changes of diabetes, nephritis, or arteriosclerosis, to warrant its universal application. Albrand-Sachsenberg conceded that emboli occluding the central retinal artery during life also may bring about similar retinal changes, but at a slower rate.

Kahn[3] (1913) was concerned with venous segmentation which he attributed to post-mortem coagulation and the squeezing out of serum to accumulate between the coagula. He rejected the "pneumatotic" hypothesis and devised various *in vitro* experiments to substantiate his views. He discussed briefly 3 cases which manifested venous segmentation at varying intervals after death and in various retinal quadrants.

On the basis of 12 cases, Würdemann[10] (1920) considered that the most pronounced and pathognomonic sign is a sickly yellowish discoloration of the fundus and blanching of the disk as heart action diminishes. Arterioles become constricted, straighter, and disappear in approximately ½ hour after death, at which time venous segmentation becomes evident due to coagulation of blood. Thus one has positive evidence of death plus an indication of elapsed time after death. Two colored drawings show most of these changes, but segmentation is not represented.

Ginestous and Lande[4] (1929) observed retinal changes after decapitation, described the classical color changes, and illustrated diagrammatically the venous segmentation which they compared to the appearance of bubbles of air in a colored-alcohol thermometer. They attributed this change to the instantaneous release of gases at death and even suggested the possibility of immediate putrefaction.

Pines[11] (1931) stated that blood "breaks up" in the arteries in 15 to 20 minutes after death, and that this occurs later in the veins. All this, he admitted, was contrary to textbook descriptions and purportedly a result of post-mortem regurgitation of static blood through a dilated aortic valve. He preferred to think of intersegmentary vascular spaces as being filled with air ("pneumatosis"). Royo-Villanova Morales[5] (1934) was in agreement with others as to color changes and maintained that only veins, segmented by bubbles of air, remained after death.

The most recent significant contribution was that of Salsbury and

Melvin[6] (1936). After observations on moribund patients and well planned experiments on dogs, they concluded that slowing of the general circulation produces "granularity" of the retinal venous blood stream; that absolute cessation of blood flow to the globe, as by bilateral carotid arterial occlusion, proximal aortic occlusion, cardiac arrest, or ventricular fibrillation, is necessary for the disappearance of the arteries and segmentation of venous columns within a matter of seconds; that venous segments continue to move toward, and fall strikingly over, the edge of the physiologic depression for at least 10 minutes *after complete stagnation* of blood; and that functional restoration of circulation totally abolishes these alterations in a few seconds. Emphasis was centered upon use of the signs—above all, the moving segments—as a clinical guide in resuscitation. They concluded by citing the need for electrocardiographic correlation with these changes.

Materials and Methods

Ophthalmoscopic photography dates back at least to 1899[21] at which time photographs in black and white were, in general, equal to the best drawings, especially when touched up with water colors.[8] The type of ophthalmoscopic camera used for the present work was evidently devised by Mawas[22] in 1933 and built by Nordenson and Zeiss. Utilizing a carbon arc as source of light, a prismatic system directs a focused beam through the pupil to illuminate the retina. The resultant image is viewed through a reflex lens apparatus to permit fine adjustment of focus. The equipment was portable and was moved to the bedside in each case.

The patient was arranged in a semi-reclining position with his head facing the objective lens at a distance of approximately 10 cm. Application of a few drops of 1 per cent neosynephrin solution insured against pupillary constriction during film exposure. For some patients the lids were taped open and the corneas kept moist by frequent gentle ejections of physiologic saline solution from an ophthalmic irrigator. The carbon arc system was supplied with electric power through a portable rectifier. Limb leads from a portable Burdick direct-writer electrocardiograph were attached to the patient, who was generally unconscious of the proceedings. Each exposure at one eighth of a second on 35 mm. Kodak Ektachrome film was indicated on a continuously running electrocardiographic (EKG) record. Efforts were made to begin photographing the fundus as respirations ceased and to continue to take serial pictures at short intervals thereafter to demonstrate the rapidity with which changes developed.

Results

Six attempts were made; in only 2 cases was the start of the photographic series sufficiently well synchronized with the onset of the patient's death and the resultant serial pictures sufficiently clear to permit complete objective evaluation of the sequence of events.

Case 1

The first patient was a 65-year-old white woman who died of lymphosarcoma a few minutes before the equipment had been set up. The first picture (Fig. 3-A) was taken hastily about 4 minutes after the last EKG oscillation and, consequently, care was not taken to have the disk in view. Arteries had disappeared, but segmented retinal veins persisted. Lacking control pictures of the living fundus, the degree of retinal pallor could not be evaluated. The venous segments appeared to be separated by serum rather than by air, and in some areas fine red streaking could be seen along venous walls in the clear zones. The second picture (Fig. 3-B) was taken 15 minutes after the first and showed essentially the same features (the discoloration was probably an artifact). Movement of segments was not looked for in this case, the first human fundus to be photographed after death in this hospital.

Case 2

A 36-year-old white woman died in coma which had continued for 8 months. She had a huge craniopharyngioma. Again, equipment was not operable at the instant of death. One of four pictures, taken about 25 minutes after respirations ceased, satisfactorily showed a hazily "beaded" vein on a very pale retina devoid of arteries (Fig. 4). Segmentation occurred early in this instance (as observed ophthalmoscopically without EKG control) and was quite remarkable: segments and intersegmentary spaces were short and moved smoothly with a velocity of approximately one to two disk diameters per second in all large venules to the physiologic depression, where they disappeared as over a precipice. Looking like converging trains of boxcars viewed from above, the moving segments presented the most awesome ophthalmoscopic dynamic picture which I have observed. The venous segments continued to move for about 20 minutes after the last breath. Their ends appeared somewhat concave suggesting that the clear spaces in the larger venous tributaries contained compressed gas.

Case 3

A 35-year-old Negress succumbed to metastatic carcinoma of the colon. Faulty adjustment of the carbon light source caused underexposure with totally unsatisfactory photographic results. Ophthalmoscopic examination revealed findings entirely similar to those in case 2.

Case 4

This patient was a 70-year-old white man with massive bilateral cerebral infarcts. The EKG tracing immediately after cessation of respiration (Fig. 1, A-C) showed greatly diminished frequency of heartbeat; a simultaneous photograph (Fig. 5-A) demonstrated hazy granularity, or possibly blurring of moving segments, of arteries only. Veins and retinal color appeared normal (shadow was caused by the untaped upper lid). The photograph reproduced as Figure 5-B, taken by chance just 6 seconds after the last EKG oscillation, revealed arteries disappearing in the direction of flow (the originally "granular" artery was no

longer visible). Venous blood on the now paler and more distinctly outlined disk was irregular in outline due to "granular" flow or moving segments. The entire retina was paler. The subsequent eight serial pictures were ruined by a technical error in exposure. Figure 5-C represents the same fundus approximately 15 minutes after the first picture of the series, but on the opposite side of the disk—a change resulting from hurried rearrangement and repositioning after the error in exposure was discovered. Venous segmentation, though not marked, was then unmistakably evident. Contrary to expectations, the arteries were still apparent; they were thin without "beading." Only in this case of the entire study did such a finding occur. It could not be ascertained whether the arteries refilled after emptying (noted earlier on the other side of the disk), or whether some arteries simply failed to "bead" and disappear.

Case 5

The cause of death of this 59-year-old Negress was esophageal carcinoma with metastases. The photograph (Fig. 7) was taken 15 minutes after clinical death, with no EKG control, and demonstrates remarkably well the venous segmentation with some streaking of erythrocytes between the red segments and, in areas, against the venous walls. The segments continued to move for approximately 12 to 13 minutes after respirations ceased. Light digital pressure upon the globe temporarily reversed direction of the moving segments in some venules, with resumption of the usual flow upon release of pressure.

Case 6

The uremic state of this 51-year-old Negress resulted from chronic pyelonephritis; death occurred immediately after the last of four terminal convulsions. Venous segmentation was observed within seconds after the last EKG activity. Figure 6-A shows indistinct but relatively normal appearing vessels and corresponds to the EKG record of Figure 2-A which showed a frequency of two beats per second. Although electrical heart activity was still present (Fig. 2-B), the frequency of seven beats per minute was too slow to prevent venous segmentation and diminishing caliber of arteries (Fig. 6-B, 45 seconds after 6-A). Figure 6-C represents the same fundus 45 seconds after Figure 6-B; arteries are barely evident.

Case 7

The fundus of a cadaver was examined in the necropsy room 3 hours after death and appeared exactly as depicted by Bouchut[1] (Fig. 8). Merely touching the globe with a finger obliterated the pink-purple venous segments at the disk; removal of the finger resulted in return of segments to exactly their original sizes and positions.

Discussion

Conditions for Successful Ophthalmoscopic Photography

Mechanical accessibility of an adequate amount of light to the retina is the obvious necessary basis of ophthalmoscopy. As a rule pupils dilate somewhat during the process of death, sufficiently to render the routine use of mydriatic solutions unnecessary. In photography with a carbon arc light source, however, iridal light reactions must be prevented. If pupils remain too small at death, e.g., in cases of morphine poisoning, post-mortem use of mydriatics will prove to be effective until iridal rigor occurs in 3 to 4 hours after death.[3]

Clarity of intra-ocular media is another factor of great importance. Extensive, severe, corneal scarring, inflammatory exudation, cataracts, or opacification of the vitreous body during life preclude retinoscopy. Rarely, however, are such obstacles encountered bilaterally; since fundic changes of death are usually similar in both eyes,[8] unilateral examination ordinarily suffices. Frequent digital closure of the lids after death may not maintain corneal moistness and transparency during prolonged examination. The resulting rapidly developing haziness of the corneal surface is best dispelled by applying a drop or two of normal saline solution every 30 to 60 seconds. Plain water has been advocated,[3,10] but protracted use of other than physiologic saline solution may itself eventually cause corneal turbidity. This simple maneuver is *extremely* important, for it allows one to view the retina in very sharp detail for hours, until parenchymal turbidity of the cornea becomes manifest.[8]

Severe, extensive, bilateral intra-ocular diseases also may create insurmountable obstacles to ophthalmoscopy. It already has been mentioned that the usual intra-ocular pathologic changes seldom create confusion with the mortal signs[9]; but large retinal detachments, intrabulbar hemorrhages, large neoplasms, and perhaps unusually severe hemorrhagic exudative retinopathy make ophthalmoscopy impossible.

General Comments

In general, after "EKG death" the arteries are either completely empty or rapidly emptying, presumably in the direction of flow, although evacuation can conceivably take place in both directions simultaneously. The single exception to this usual arterial behavior at death was noted in case 4 of this study. With slowing of heart rate, arterial "granularity" sometimes becomes manifest in the larger branches; that it occurs at all has been questioned by some workers,[6] but not flatly refuted.

Venous irregularity due to marked "granularity" or to moving segments is observed almost invariably within several seconds after cardiac output ceases,[6] as is borne out by the photographs. The first change usually occurs in the large trunks and finer venules on the disk[8] where early segmentation is the rule. Distribution of segments over the fundus varies from case to case[3,23] and some authors insist that this feature may be undetectable early in rare instances.[3,7,10,11,23]

The ultimate cause of venous blood segmentation, a clumping of erythrocytes as circulation loses speed,[2,6] remains obscure. Deceleration and inertia may in themselves be the answer, with post-mortem

equalization of the arterial and venous pressures and a "milking" arteriolar peristalsis accounting for segmental motion[6,23] which is occasionally unapparent in old and debilitated animals.[23] It is interesting to speculate just what rôle, if any, a generalized tendency for body blood to "sludge" during morbid states[24] may play in the genesis of segmentation. Another mechanical factor may be fundamental, a relative increase in intra-ocular pressure. Usher[2] claimed to have caused transitory segmentation during life by injecting saline solution into the eye and, conversely, to have prevented it in animals at death by removing the cornea. It is true that while intra-ocular pressure begins to fall after death,[8] it remains considerably elevated for a time relative to intravascular pressure which is nil; this consideration lends weight to Usher's theory. The possibility of segmentation with glaucoma during life, then, becomes a plausible assumption, but to my knowledge this has not been reported. It may be either that local, intra-ocular, compensatory, vascular pressure changes serve to restore the pressure balance, or that segmentation does occur during late stages of the disease when retinal visualization is no longer practicable.

The only known vital process which produces retinal vascular changes similar to those of generalized circulatory standstill is occlusion of the central retinal artery,[9] either by thrombosis or by embolism. However, the changes under such circumstances tend to occur more slowly, with accompanying edema and hemorrhages. Both causes are rare, and simultaneous bilateral occurrence especially so.

Probably, moderate variations in temperature have no effect upon the appearance of the signs. The results of experiments on hypothermic rabbits strongly negate earlier observations[2] on human corpses. Six anesthetized rabbits with normal rectal temperatures of 39° to 40° C. were subjected to hypothermia. In 3 of them, which evidently froze to death, retinal pallor and venous segmentation with "granularity" were obvious on and around the disk at the time of removal from the bath; rectal temperatures were 14°, 20°, and 21° C., respectively. Arteries had disappeared. Two animals survived shorter exposures, having temperatures of 29° and 32° C. Rapid intravenous injection of calcium chloride solution was followed almost instantaneously in both animals by pallor and segmentation as described. In no instance during this experiment was segmentation as clear-cut as is usual in human eyes, but rather tended to be more "granular" and "sludged" without motility. After 1 hour in the bath, one rabbit with a temperature of 15° C. appeared to be dead on inspection and palpation. Retinal vessels were thinned but continuous. Typical changes of actual

death, however, resulted from a rapid injection of calcium chloride. As a control, one rabbit anesthetized with intravenous pentobarbital was sacrificed by means of intravenous calcium gluconate, without being immersed in an ice bath; segmentation was definite and rapid.

As to the effect of high temperatures, most of the patients photographed had low-grade fevers; and early segmentation in case 6 (Fig. 6-B) is indisputable in spite of excessive hyperpyrexia at death (over 107° F., rectally). It seems reasonable to conclude that the usual behavior of retinal vessels when heart output stops is not modified by variations in body or environmental temperatures within relatively wide range.

In conclusion, these rapid signs—above all, "granularity" and segmentation of columns of venous blood—signify only one thing: *absence of cardiac output,* due either to a weak, non-functional beat, or to complete arrest, ventricular fibrillation, or complete heart block (patients with Stokes-Adams syndrome could conceivably show the signs).[6] When seen, they indicate cessation of blood flow to the eyes and therefore cessation of flow to the brain, and, obviously, complete cessation of flow to all organs—or practical death. It is this period of terminal agony (vascular narrowing and "granularity") and so-called clinical death (segmentation *with* movement) which is of utmost importance in resuscitable cases.[25] No known clinical or laboratory aid other than the ophthalmoscope can determine so quickly whether circulation has stopped recently enough to allow for resuscitation without severe residual central nervous system disintegration: the electrocardioscope[25] and the EKG indicate merely that flow or heart action has stopped, with no indication as to elapsed time. The EKG may, in addition, show normal or abnormal oscillations (injury currents) up to an hour or more after complete standstill.[26] If venous segmentation with movement is seen ophthalmoscopically, it is reasonable to assume that cardiac standstill could have been no more than 10 to 20 minutes antecedent to examination, and vigorous resuscitative measures should be attempted. After segmental movement has stopped, resuscitation is virtually hopeless despite any undertaking.[6]

In this connection, Wegner's work[27] (1933) is of special interest. He determined that the retina can endure complete occlusion of its blood supply for 22 minutes without suffering any permanent functional impairment after restoration of flow. The critical time range in which cells began to disintegrate, producing visual impairment, was from 22 to 45 minutes. After cardiac standstill the venous segments continue to move as long as 20 ± minutes. This may serve as an indi-

cation not only of the possibility of retinal restitution, but also that similar vascular phenomena in other, invisible organs of the body, including the heart, may maintain the entire organism in a resuscitable state for that period of time.

The ophthalmoscopic signs may find useful application as follows:

1. In general clinical practice: to determine a state of death; to differentiate between real and apparent death and to determine resuscitability of victims of fainting, pathologic sleep, electrocution, deep coma, profound shock; narcosis (barbiturates, chloroform), asphyxiation, or drowning; to differentiate real shock from cardiac arrest or fibrillation during surgery; and to determine death in patients in respirators.

2. In forensic medicine: to estimate time of death in minutes (moving segments) or hours (static segments and retinal color); and to determine the position of the head at death.

Summary

There are ophthalmoscopically observable changes in the color of the retina and in the caliber and content of the retinal vessels which are indicative of impending and of actual death. Of special importance are "granularity" of venous blood and of moving or static segmentation of the columns of red blood cells in the veins. These changes are illustrated by colored photographs which are synchronized (for the first time) with electrocardiographic tracings.

These changes provide a readily available means of determining the fact of death, of differentiating apparent and actual death, of estimating (within a certain range) the elapsed time after death, and, of great importance, of indicating the possibility of success from efforts at resuscitation.

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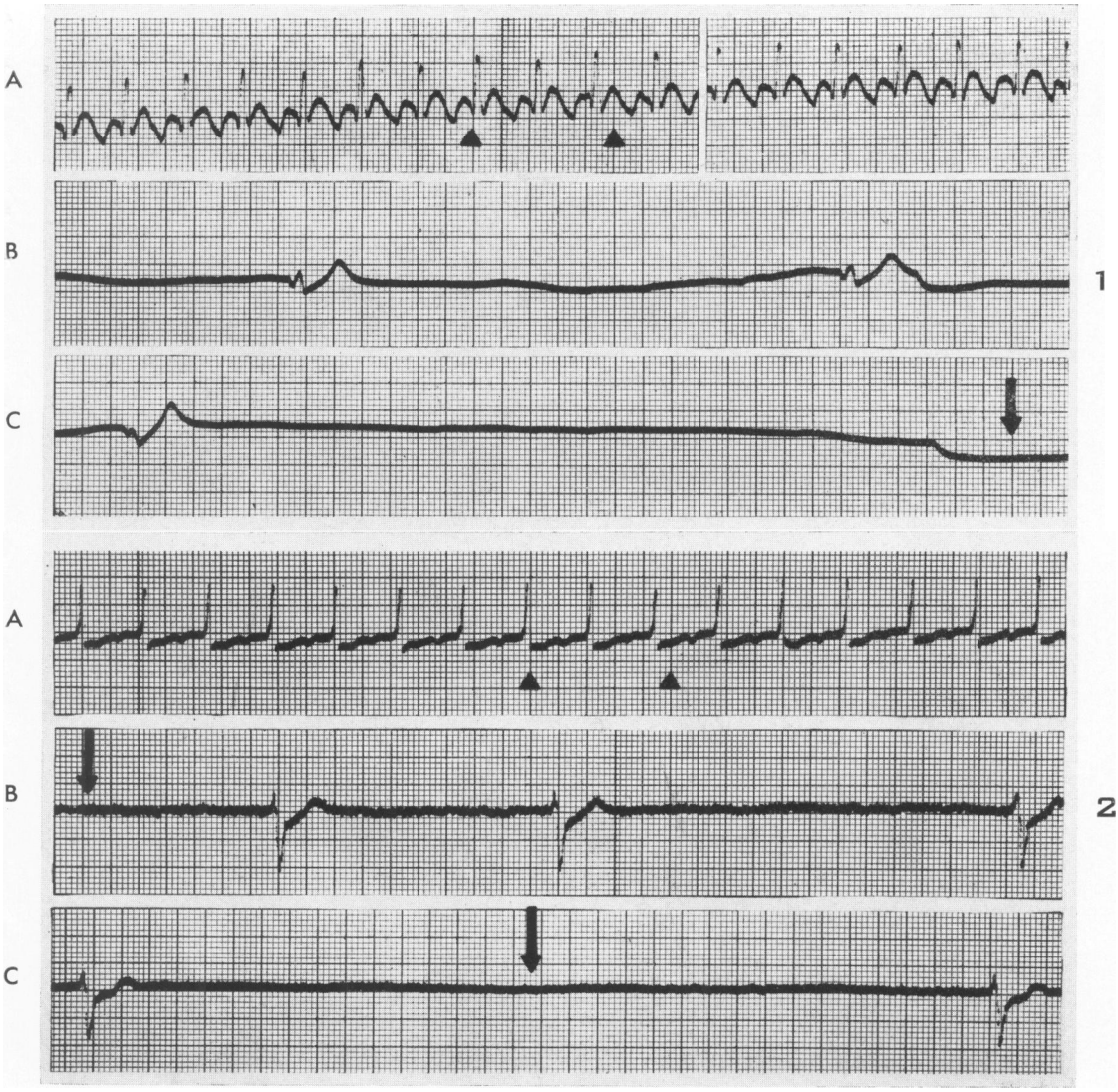
[*Illustrations follow*]

LEGENDS FOR FIGURES

FIG. 1. EKG tracings in case 4. *A*. Several minutes before respirations ceased. Triangles delimit an interval of 1 second. *B*. Approximately 45 seconds after respirations ceased. Although the exact time was not indicated, Figure 5-A was taken during this period. *C*. The last EKG oscillation. Arrow indicates time of Figure 5-B, approximately 30 to 40 seconds after Figure 5-A.

FIG. 2. EKG tracings in case 6. *A*. Approximately 3 hours before death; 1 second interval between triangles. This was the EKG pattern with Figure 6-A. *B*. Immediately after the final convulsion; respirations were undetectable. Arrow marks point at which Figure 6-B was taken. *C*. The last two EKG oscillations. Arrow shows when Figure 6-C was taken, 45 seconds after Figure 6-B.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C
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FIG. 3. Fundus in case 1. *A*. Approximately 4 minutes after "EKG death." *B*. Fifteen minutes after A.

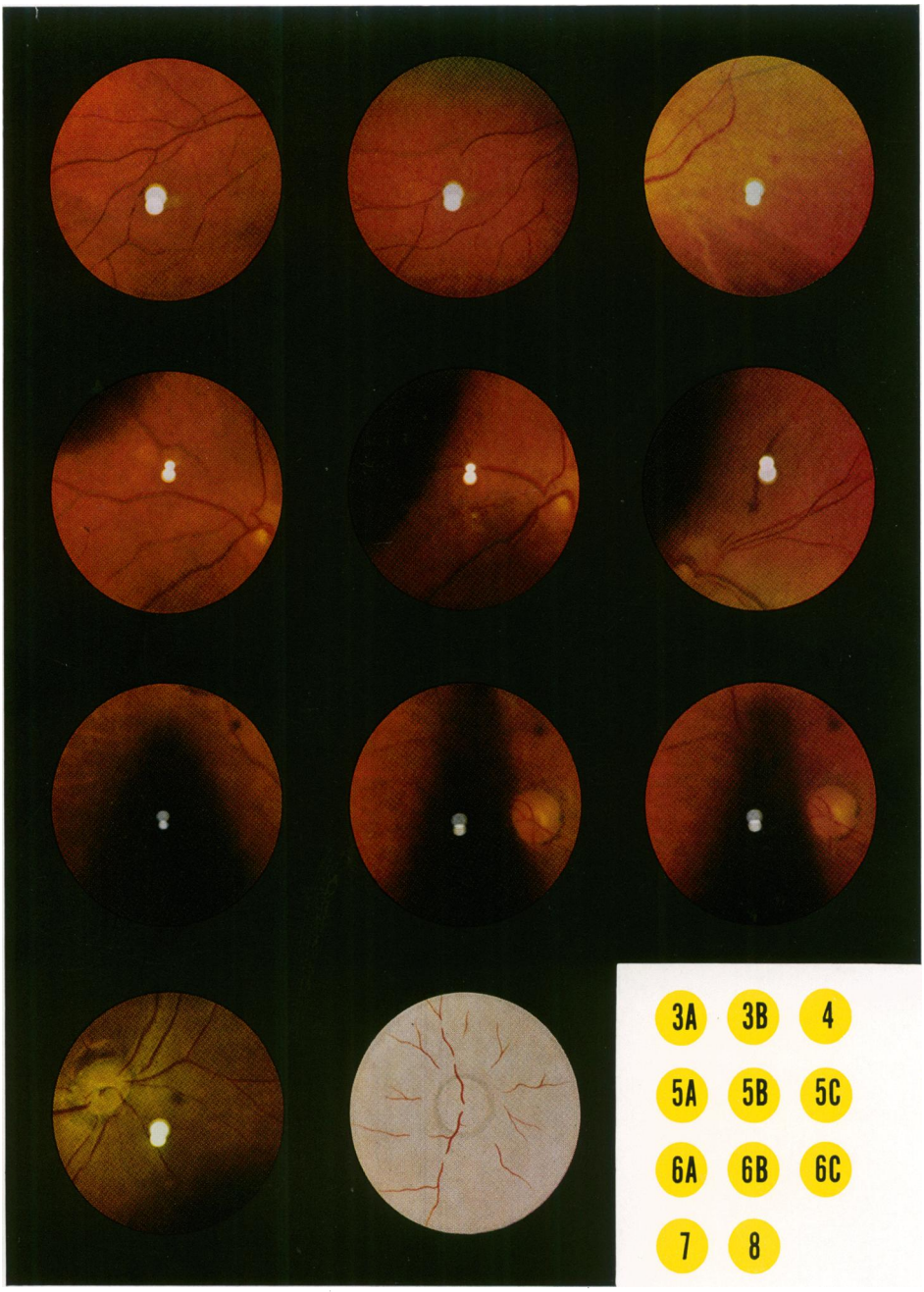
FIG. 4. Fundus in case 2. Approximately 25 minutes after respirations ceased.

FIG. 5. Fundus in case 4. *A*. Approximately 45 seconds after respirations ceased. EKG frequency about 15 oscillations per minute (see Fig. 1-B). *B*. Same fundus 30 to 40 seconds later, 6 seconds after last EKG oscillation (see Fig. 1-C). *C*. Same fundus 15 minutes later, opposite side of disk.

FIG. 6. Fundus in case 6. *A*. Approximately 3 hours before death (see Fig. 2-A). *B*. Same fundus and generally the same site with the disk now visible. Respirations had stopped; EKG frequency very low (see Fig. 2-B). *C*. Same fundus 45 seconds later, at the time of "EKG death" (Fig. 2-C).

FIG. 7. Fundus in case 5. Fifteen minutes after clinical death.

FIG. 8. Photographic reproduction of Bouchut's original drawing.[1]


3A
3B
4
5A
5B
5C
6A
6B
6C
7
8