THE ELECTROENCEPHALOGRAM AFTER CARDIAC ARREST*

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EXPERIMENTAL STUDIES have shown that the mortality from ischaemic brain damage can be reduced by hypothermia used within three hours following the injury.[10,11] Clinical observations support these findings, and there is reason for believing that in some cases recovery was only possible with the help of hypothermia. Therefore it has been recommended that hypothermia should be used in all cases of cardiac arrest with central nervous system damage.[2,4] The problem in clinical practice is the recognition of cerebral damage early enough to institute treatment at a time when it is most beneficial.

The E.E.G. has been recorded in humans at various intervals following accidental cardiac arrest.[1,3,5,13] With an increasing number of observations it has become evident that the E.E.G. can only be considered a diagnostic aid, that cortical potentials must be interpreted in relation to functional changes following injury, and that there is no single electrical wave pattern that, in itself, can be regarded with certainty as being incompatible with recovery.

The following E.E.G. changes can be observed in dogs after cardiac arrest: Anoxia causes the electrical activity of the cortex to disappear—there is electrical silence or a "flat" E.E.G. After a short period of anoxia, recovery of the electrical activity of the cortex proceeds in a characteristic fashion (Figure 1). The "recovery pattern" begins with slow waves with a low amplitude; these are irregular and increase in frequency and amplitude (Figure 1, graph 2). Superimposed there appear faster waves, first intermittent, later continuous, which increase in frequency and gradually reach the range of beta frequency. Simultaneously the slow waves diminish and finally disappear (Figure 1, graph 3). This leads to a pattern of normal frequencies but with a low voltage (Figure 1, graph 4). Lastly the voltage gradually increases to the pre-anoxic level (Figure 1, graph 5).[13] There are modifications—e.g. the first return of electrical activity may occur in bursts with intermittent periods of absence of activity[3] but in principle there is a similar sequence of events.

An accidental observation in man (Figure 2) revealed the same pattern.[13] This patient had an episode of ventricular fibrillation in the course of a thyroidectomy, with successful resuscitation after a period of circulatory arrest of 1 minute and 50 seconds; low-voltage slow waves, a pattern of irregular slow waves, increasing in amplitude and frequency, superimposed fast activity, a fading of the slow-wave activity, and finally a return to a normal pattern were observed. The patient had to be reanaesthetized for the completion of the operation and made a complete and uneventful recovery.

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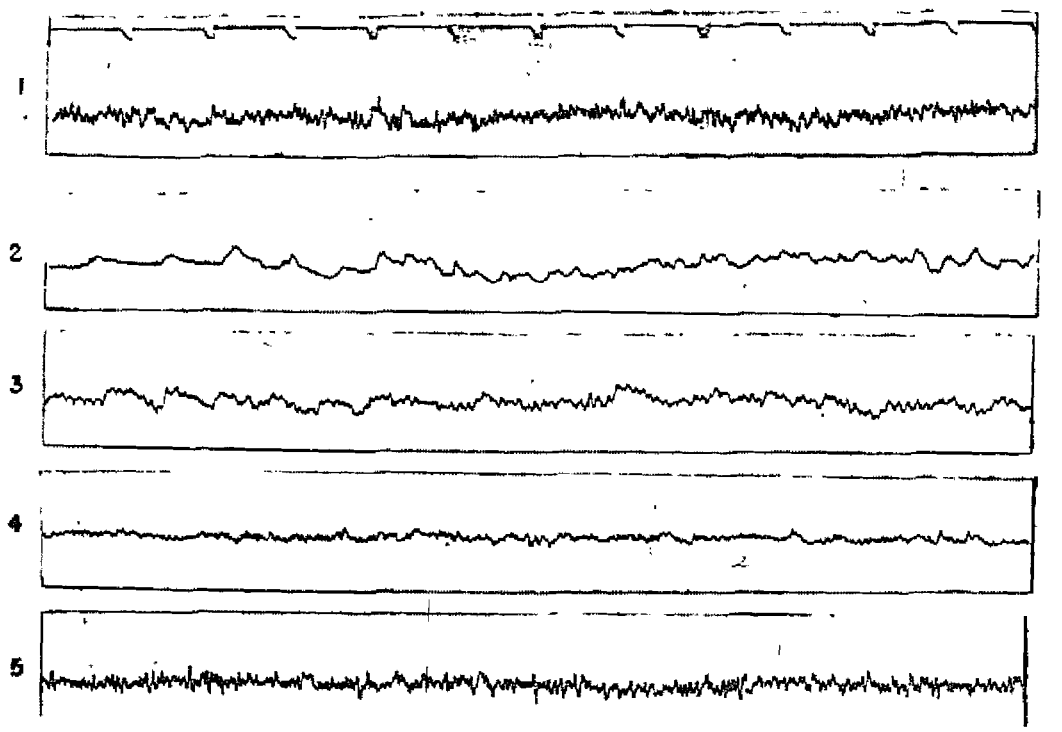

FIGURE 1. Return of E E G after anoxia: dog in very light thiopentone anaesthesia, ventilated with N_2 until the E E G. became "flat" 1, before anoxia, 2, 30 seconds after the end of anoxia—first return of electrical activity, 3, 2 minutes later, 4, 5 minutes later—low amplitude, normal frequency, 5, 1 hour later—complete recovery

Recovery may be expected in practically all patients showing this pattern—and this is readily substantiated by the rapid reappearance of reflexes, responses to stimuli, and finally the return of consciousness

If the patient remains unconscious and an E E G is recorded some time after an arrest, the following may be seen.

1. There may be no electrical activity. While this is in most instances a very bad sign, it must be remembered that in principle a "flat E.E.G." does not necessarily represent a dead cerebral cortex. It has been shown that a very low blood flow through the brain may be adequate to maintain the integrity of the cortex for some time without signs of electrical activity or cortical function.[6] The return of a normal blood flow after such a period of low flow results in rapid recovery. Thus, the duration of a flat E.E.G. is not of great significance unless accompanying factors are known.

2. A so-called file pattern may be present. It consists of a low-voltage, fast activity pattern with frequencies above 25 per second, resembling the cross-section of a file.[5] This pattern was observed in many of the patients dying with irreversible brain damage.[1,3,5,13] On the other hand, Pearcy and Virtue[9] have reported a "file pattern" subsequent to a period of arrest of the cerebral circulation under hypothermia, particularly when there was a wide area between the two electrodes. In their cases, the voltage gradually increased, leading to complete recovery. A low-voltage pattern during normal recovery (as shown in Figure 1, graph 4) could be interpreted as a "file pattern," and that reduces the prognostic significance of this wave-form.

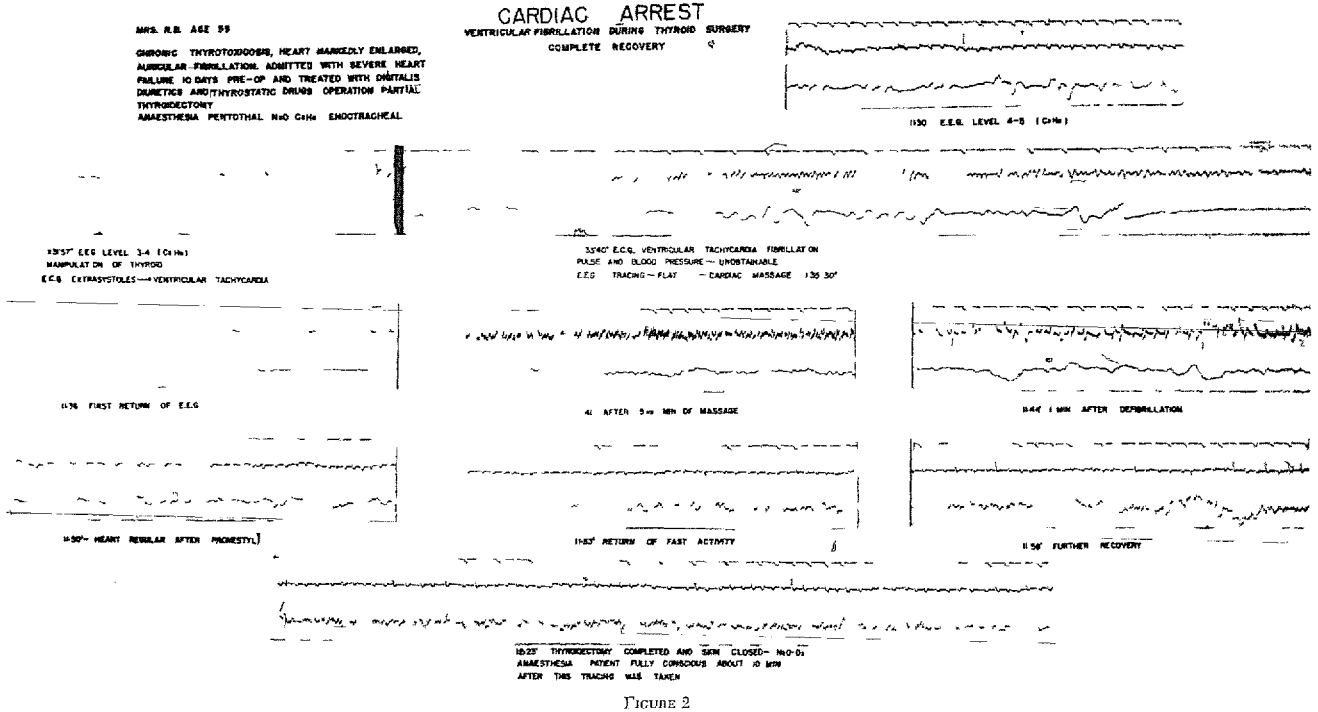
CARDIAC ARREST
VENTRICULAR FIBRILLATION DURING THYROID SURGERY
COMPLETE RECOVERY
MRS. R.B. AGE 59
CHRONIC THYROTOXICOSIS, HEART MARKEDLY ENLARGED, AURICULAR FIBRILLATION. ADMITTED WITH SEVERE HEART FAILURE 10 DAYS PRE-OP AND TREATED WITH DIGITALIS DIURETICS AND THYROSTATIC DRUGS. OPERATION PARTIAL THYROIDECTOMY
ANAESTHESIA PENTOTHAL N2O C2H4 ENDOTRACHEAL
1930 E.E.G. LEVEL 4-8 (C2H4)
1957' E.E.G LEVEL 3-4 (C2H4)
MANIPULATION OF THYROID
E.C.G. EXTRASYSTOLES → VENTRICULAR TACHYCARDIA
3540' E.C.G. VENTRICULAR TACHYCARDIA FIBRILLATION
PULSE AND BLOOD PRESSURE — UNOBTAINABLE
E.E.G. TRACING — FLAT — CARDIAC MASSAGE 135 30'
1936 FIRST RETURN OF E.E.G
4' AFTER 5 MIN OF MASSAGE
1944' 1 MIN AFTER DEFIBRILLATION
1950' HEART REGULAR AFTER PROMESTYL
1953' RETURN OF FAST ACTIVITY
1956' FURTHER RECOVERY
1027 THYROIDECTOMY COMPLETED AND SKIN CLOSED— N2O-O2
ANAESTHESIA PATIENT FULLY CONSCIOUS ABOUT 10 MIN
AFTER THIS TRACING WAS TAKEN

FIGURE 2

3. Convulsions may occur either during anoxia (as in the Adam-Stokes Syndrome) or shortly after reoxygenation. They are usually generalized and are not incompatible with recovery. Convulsive discharges, seen some time after the return of the blood or oxygen supply to the brain, which are irregular, complex, and bizarre in their configuration and are associated with muscle twitches, represent probably evidence of cortical damage.

In repeated E.E.G. recordings in six patients who did not recover after cardiac arrest, all showed some types of convulsive discharges.[13] They were particularly pronounced in a 20-month-old boy (Figure 3), who was brought to us because of persistent unconsciousness after a period of arrest reported to be less than one minute. He had been treated successfully with closed-chest massage. Frequent twitching motions in the arms and legs appeared several hours after the arrest and produced muscle artefacts in the E E G. The E E G. consisted of recurrent convulsive discharges.

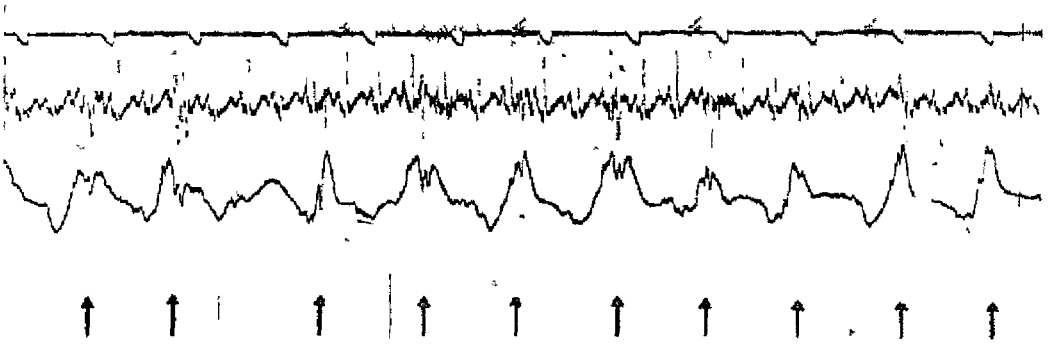

FIGURE 3 Patient 20 months old, 5 hours after cardiac arrest. Convulsive discharges of irregular configuration. Synchronization between spikes (E E G.) and muscle twitches (E C.G. disturbance) indicated by arrows.

In this patient thiopentone temporarily suppressed the convulsions and revealed a file pattern for a short period (Figure 4). Finally the convulsive discharges ceased and the patient's condition deteriorated rapidly. He died three days after the arrest.

In another 2-year-old child (Figure 5) a rather complex electrical discharge regularly preceded the patient's respiration. No other muscle movements were noticed. The patient died two days after the arrest.

The E.E.G., used in this fashion is only of limited value. It will confirm our clinical impression of a quick recovery or it may show an abnormal pattern in patients with irreparable brain damage, but it does not indicate those patients on the border of irreversible cortical damage at a time when treatment may be possible. In order to approach this problem one must consider the E.E.G. as a function of the cerebral cortex and look at anoxia as a disturbance that produces a chain of interrelated events.

Following the interruption of blood flow (Figure 6), the function of the cortex will survive for a few seconds; this has been called the *survival time*.[8] After that all measurable function ceases; yet for a certain time the brain can be revived—

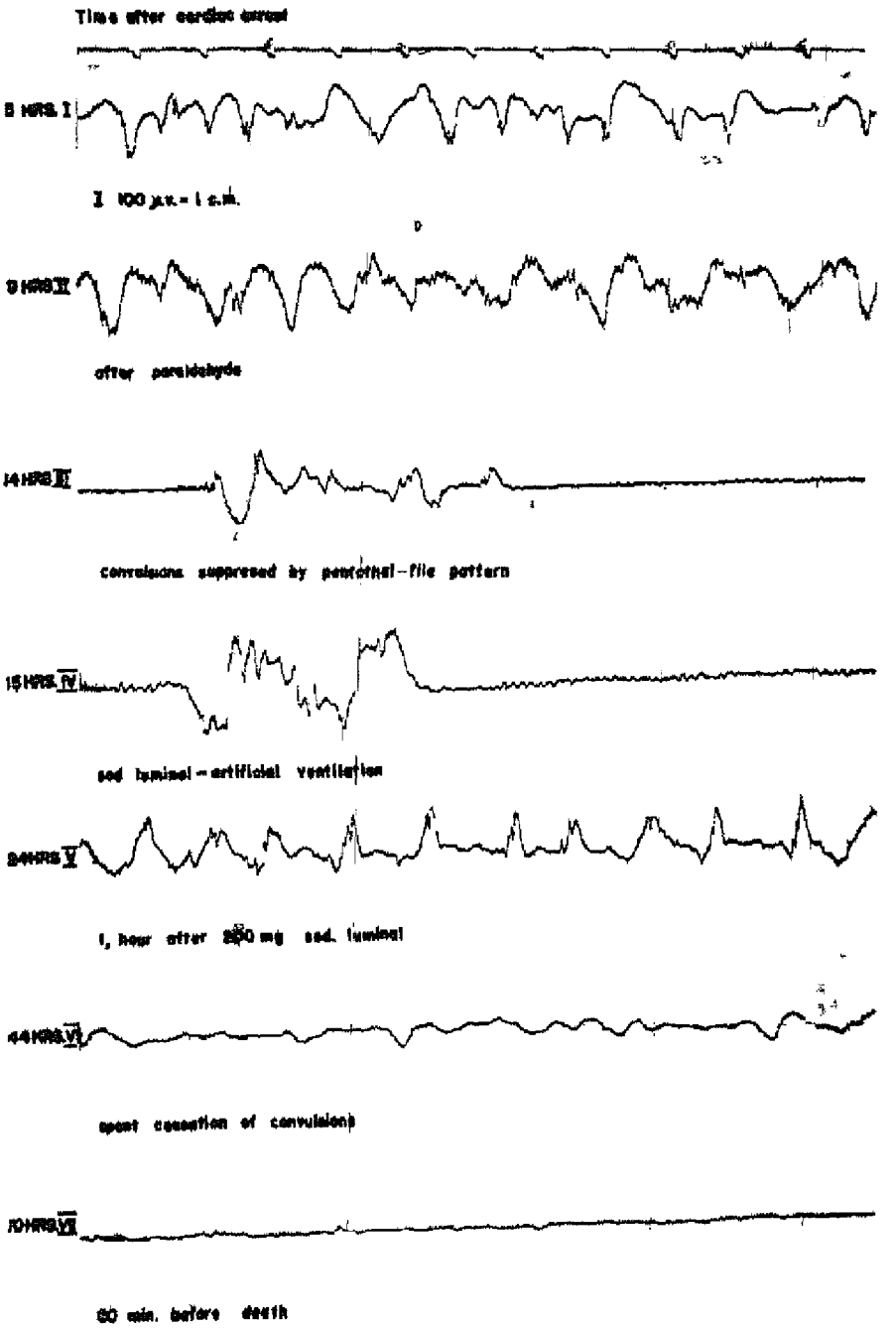

FIGURE 4 Same patient as Figure 3 E E G recordings at different times after cardiac arrest

the *revival time* For the human brain a revival time between 3 and 5 minutes is usually assumed If the blood flow is not restored during this time there will be cellular death and irreversible brain damage On the other hand, a return of the blood flow during the revival time will bring about a gradual recovery of function (*recovery time*) after a period of latency, here indicated as *latency of recovery* From experimental observation there is good evidence that these three intervals,

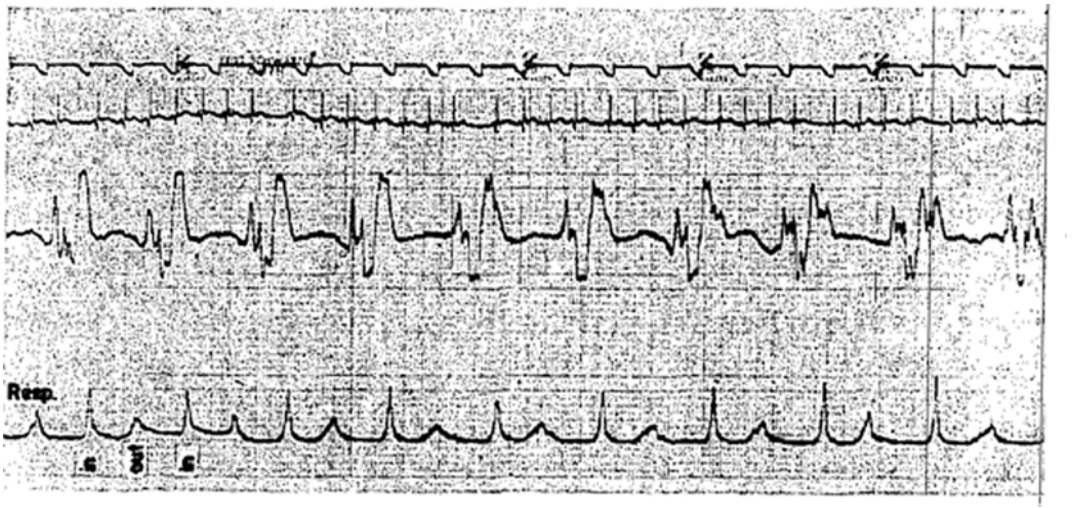

FIGURE 5. Patient 2 years old, 16 hours after cardiac arrest, regular complex spike and wave pattern superimposed on activity with low amplitude and high frequency. Note synchronization between spikes (E.E.G.) and inspiration (lowest tracing) and the absence of muscle artefacts (E.C.G., upper tracing.)

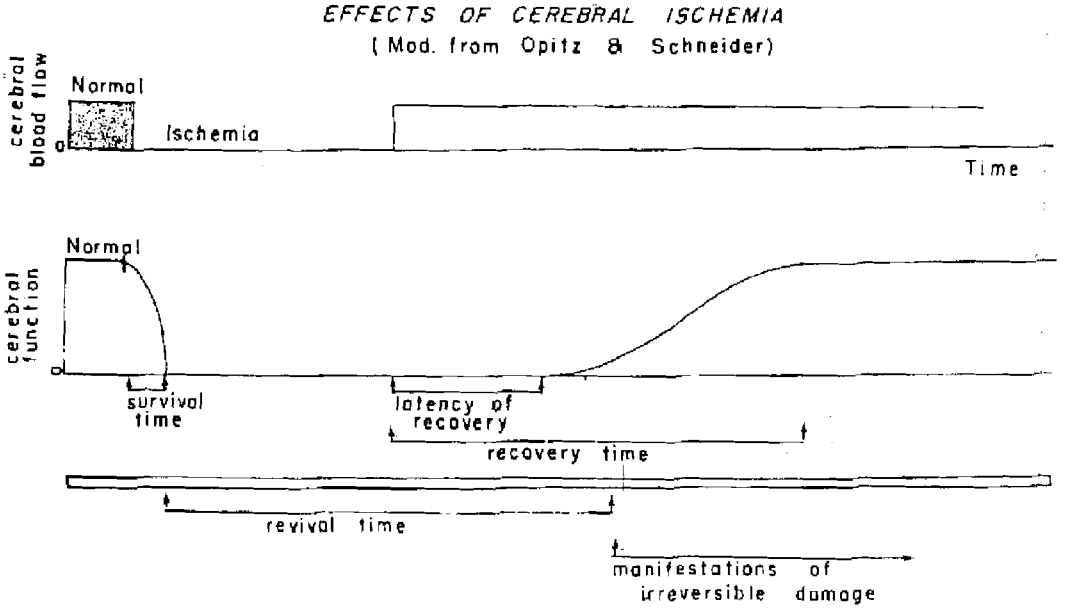

FIGURE 6. Schematic presentation of effects of cerebral ischaemia.[8] Top, indication of cerebral blood flow; middle, indication of corresponding changes in cerebral function; bottom, indication of revival time.

the duration of ischaemia, the latency of recovery, and the recovery time are related in a certain way.[12]

Brechner[3] found a logarithmic relationship between the duration of ischaemia and the latency of recovery. Hirsch *et al.*[7] reported an increase in latency of recovery proportional to the duration of ischaemia up to about three minutes; with a longer duration of cerebral ischaemia, the latency of recovery rose steeply.

Schneider[12] found that there is also a good correlation between latency of recovery and the recovery time. With an increasing latency of recovery, the recovery time rises in an exponential fashion.

It would appear that the latency of recovery could be a useful clinical index, superior to an estimate of the time of ischaemia, which is notoriously prone to gross inaccuracies. The time at which cerebral blood flow has returned—i.e. when effective cardiac action has been restored or effective massage begun—can readily be observed and the interval to the return of cortical potentials determined accurately. This time interval could then be used as a basis for the selection of treatment. Brechner[8] feels that if this latency exceeds 15 minutes, hypothermia should be started without delay, and this appears the best approach to this problem in the light of our present knowledge.

If one considers the E.E.G. as being a function of the cerebral cortex, one might assume that other cortical functions could be used to determine this latency of recovery, e.g., the pupillary reflex, the corneal reflex, or other reflex responses. I am not aware of any clinical data that would substantiate this, nor can I offer any figures at this time. But theoretically it should be possible to use any manifestation of cortical function for the determination of a latency of recovery and thus to obtain a useful practical index without complicated equipment.

However, the use of neurological signs to determine the recovery from cerebral anoxia can be misleading. As an example, Table I gives a coarse neurological

TABLE I

TRANSIENT RECOVERY IN TWO CASES AFTER CARDIAC ARREST WITH IRREVERSIBLE BRAIN DAMAGE

	Case H							Case D				
	6*	12	24	36	48	60	70	1*	6	12	24	36
Consciousness	−	−	−	−	−	−	−	?	(+)	(+)	?	−
Light reflex	(+)	(+)	(+)	+	(+)	−	−	(+)	+	+	+	?
Size of pupils, mm.	1	1	2	2	3	3	3	2	1	1	2	3
Lid reflex	+	(+)	−	−	−	−	−	(+)	+	+	+	−
Pain stimuli	+	(+)	(+)	+	−	−	−	+	+	+	(+)	−
Coughing, swallowing	+	(+)	−	?	−	−	−	+	+	+	+	−
Spontaneous respiration	+	+	(+)	(+)	(+)	−	−	+	+	+	+	−
Convulsive movements	(+)	(+)	+	+	−	−	−	−	−	−	+	(+)

Signs: + present, (+) weak but present, ? doubtful, +) unequal, − absent.
*Hours after cardiac arrest.

assessment of the two children whose E.E.G. records of convulsive discharges have been shown earlier. Both made a slow but steady recovery to a remarkable degree of reflex activity; one even appeared to respond to the calling of his name. Then after reaching a point where one almost had hope for the return of consciousness, both deteriorated and died within two and three days after the arrest. One had the impression that certain elements of the brain were able to function for a period of time after they have been irreversibly damaged.

Similar observations have been made experimentally and there appears to be a marked discrepancy in the effect of oxygen lack on the processes involved for the survival of the nerve cell and those for neuronal and synaptic conduction. Von Harreveld[14] believes that this may be due to damage of structures involved in the manufacture of complex enzymes systems. The store of enzymes present at the end of an ischaemic period may enable a nerve cell to recover and function to a limited extent but the insufficient replacement of these enzymes will subsequently lead to the death of such a neurone.

Summary

There is no specific E.E.G. pattern that indicates with certainty irreversible brain damage. Following a short period of cerebral ischaemia, the E.E.G. shows a definite pattern of recovery. Failure of this recovery pattern to occur may indicate brain damage, as does the appearance of a "file pattern" and of convulsive discharges. However the whole recovery must be viewed as a functional process, and while a single E.E.G. record may be of little significance, frequent recordings can aid in the prognostic assessment of the case.

There is a relationship between the duration of ischaemia and the time interval between restoration of blood flow and return of function. The duration of this latency of recovery is in turn related to the total recovery time and this is proposed as a more suitable basis for assessing the patient's chance of recovery than an estimate of the time of ischaemia. The use of hypothermia is probably indicated if the electrical activity has not returned after 15 minutes.[3] It appears possible that apart from the E.E.G. a similar relationship could be established using the return of reflex activity to determine this latency of recovery, thereby making an assessment of the expected speed of recovery possible.

However, attention is drawn to the observation that a considerable reflex activity may return temporarily after several hours in spite of irreversible damage of the brain.

Acknowledgment

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Résumé

Il n'existe pas de tracé électroencéphalographique spécifique indiquant de façon certaine des lésions cérébrales irréversibles. A la suite d'une courte période d'ischémie cérébrale, l'électroencéphalogramme montre un tracé caractéristique de réveil. Si ce tracé n'apparait pas, il peut s'agir de lésions cérébrales comme l'indique un tracé en lime et l'apparition de crises convulsives. Toutefois, toute la récupération doit être envisagée comme un processus fonctionnel et, si un tracé n'offre qu'une valeur relative, de nombreux tracés peuvent aider à établir le pronostic d'un cas.

Il existe une relation entre la durée d'une ischémie et le temps écoulé entre le retablissement du débit sanguin et le retour de la fonction. D'autre part, le délai de ce retour à la fonction est en relation avec le délai de l'amélioration générale et c'est là la base pour estimer les chances de guérir son malade plutôt que la durée de l'ischémie. Si l'activité électrique cérébrale n'est pas manifeste après 15 minutes, l'emploi de l'hypothermie est probablement indiqué.

A part l'électroencéphalogramme, il semble possible d'établir une relation semblable en employant le retour de l'activité réflexe pour produire le délai de la récupération et ainsi en faisant un critère de la vitesse de guérison.

Toutefois, il faut attirer l'attention sur le fait qu'une activité réflexe considérable peut réapparaître temporairement après plusieurs heures en dépit de l'existence de dommages cérébraux irréversibles.

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