# THE ELECTROENCEPHALOGRAM IN PATIENTS UNDERGOING OPEN HEART SURGERY WITH HEART-LUNG BYPASS 

Harold T. Davenpont, F.F.A.R.C.S., G. Anfel, M.D., F. R. Sanchez, M.D.

Some authorities state $1,2,3$ that the electroencephalogram shows little change during open heart surgery using heart-lung bypass. It would be of value to find the relationship between the use of a heart-lung bypass apparatus and the electrical cerebral function in these circumstances. We therefore describe the encephalographic findings observed during such operations and have attempted to make some conclusions from these studies. Eighteen patients operated on at The Montreal Children's Hospital were studied. The surgical techniques had been used previously at this institution in seventy other operations and one of us (G. A.) had had experience reading electroencephalograms during heart surgery over the last three years.

## Method

By means of a Grass Polygraph we made simultaneous recordings of arterial pressure, electrocardiogram and electroencephalogram. The arterial pressure was always measured directly by catheterization of a systemic artery (radial artery or internal mammary artery). The electroencephalogram was recorded from bipolar electrodes, one frontal and one central parietal. Symmetrical electrodes on the right and left sides were attached to make possible the verification of any aspects of the tracings from each cerebral hemisphere. We began recording the electroencephalogram soon after the patient was anaesthetized. From the begiming of the operation to the point of the catheterization of the venae cavae the electroencephalogram was recorded intermittently, but after such catheterization recording was continuous to the end of the operation. The speed of the recording was mostly 10 mm . per second and only rarely was it 25 mm . per second for detailed observation.

[^0]In the eighteen patients studied, two had two separate periods of heart-lung bypass at the same operation. In this way, we had twenty tracings during bypass for study.

## Resulets

We divide the changes in the electroencephalogram observed during the different periods of the operations as follows: (1) initial modifications at the beginning of the bypass; (2) modifications observed during the remainder of the bypass period; (3) modifications noticed at the end of the bypass, and, (4) the characteristics of the tracings at the end of the operation.
Initial Modifications. In all instances the electroencephalogram became altered at the moment the bypass was started and in seventeen there were detrimental changes of relative intensity. In only three instances was there improvement of the tracing at this time. These alterations occurred after a period from 30 to 150 seconds from the beginning of the heartlung bypass. Any abnormality in the electroencephalogram came on more quickly than any improvement.

The detrimental change which appeared at the begiming of the bypass was one of three types according to the degree of alteration.

Type I-Sencht Altemations: Twice the abnormality consisted of the appearance of slow waves superimposed on the previonsly existing rhythm. This alteration disappeared after several minutes, returning to the previous wave type with minimal variations in voltage or frequency (fig. 1).

Type II-Monerate Alterations: On five oceasions, waves with a frequency of 1 to 2 cycles per second ( $\mathrm{c} / \mathrm{s}$ ) and an increase in voltage appeared, but in addition to these new waves there was persistence of background patterns present before the onset of the bypass. These patterns became less but never disappeared, and soon became predominant again (fig. 2).
Type III-Marked Alterations: The electroencephalogram described as type III showed a constant sequence of changes. This sequence was observed in ten subjects. The only difference was in


Fic. 1. A 58 year old patient with an interatrial septal defect (no. 5). (a) Normal E.E.G. before the bypass. (b) Slight alteration of the electroencephalogram and negligible change of the blood pressure one minute after the bypass began (type I).


Fig. 2. A 4 year old patient with tetralogy of Fallot (no.3). (a) Normal electroencephalogram before the bypass. Blood pressure 95 mm . Hg. (b) Moderate alteration of the electroencephalogram (type II) and blood pressure lowered to 40 $\mathrm{mm} . \mathrm{Hg} 21 / 2$ minutes after the bypass began.


Fig. 3. A 4 year old patient with a large ventricular septal defect (no. 2). (a) Normal electroencephalogram before the bypass. Blood pressure $75 \mathrm{~mm} . \mathrm{Hg}$. (b) Severe alteration of the electroencephalogram (type III) 35 seconds after the bypass began. First change shows slower waves of higher voltage. Blood pressure 50 mm . Hg . (c) Alteration type III, third change, showing slow regular high voltage waves $21 / 2$ minutes later. (d) Some recovery with more rapid, smaller, irregular waves $21 / 2$ minutes later. Blood pressure $65 \mathrm{~mm} . \mathrm{Hg}$. (c) Nearly normal electroencephalogram $61 / 2$ minutes later, Blood pressure 75 mm . Hg. The total duration of altered patterns was 10 minutes.
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Fig. 4. A 4 month old patient with a congenital aortic stenosis (no. 9), (a) Marked suppression of cerebral activity with severe hypotension before the bypass. (b) An improvement of the electroencephalogram appearance with a rise in blood pressure 3 minutes after the bypass began.
the duration of the successive changes in each patient.

First Change Soon after beginning the bypass the waves became abruptly of higher voltage and became slower -5 to $6 \mathrm{c} / \mathrm{s}$. The duration of this period was generally about 10 to 15 seconds.

Second Change: The voltage then became greater and a sinusoidal wave form became evident, with a rate of $3 \mathrm{c} / \mathrm{s}$.

Third Change, A period of greatest disturbance followed. The tracing was slow ( $1 / 2$ to $1 \mathrm{c} / \mathrm{s}$ ), of high voltage ( 300 to 500 microvolts) and perfectly regular.

After this third change there was a recuperative phase, in which the brain waves became smaller, faster and irregular. They were of a frequency of 4 to $5 \mathrm{c} / \mathrm{s}$ and became gradually faster -10 to 12 $\mathrm{c} / \mathrm{s}$ and sometimes 14 to $15 \mathrm{c} / \mathrm{s}$.

The total duration of these three changes of wave character was 2 to 15 minutes and the duration of each change, except the first, was most variable (fig. 3).

In three patients the electroencephalogram previous to the bypass was flattened and of poor quality and this was substituted during the bypass by a more normal, vanied pattern. The frequency of the background waves accelerated to 10 to $12 \mathrm{c} / \mathrm{s}, 2$ to $21 / 2$ minutes after the onset of the bypass (fig. 4).

Modifications during the Remainder of the Bypass Period. After the period of initial abnomality (which occurred seventeen times) the electroencephalogram became stable on thirteen occasions, being formed generally by association of waves of 5 to $6 \mathrm{c} / \mathrm{s}$ and waves of 10 to $15 \mathrm{c} / \mathrm{s}$ (fig. 3, e). The proportion of frequencies of 5 to $6 \mathrm{c} / \mathrm{s}$ was, however, greater than before the bypass; that is, the total tracing had less waves for a given interval of time. A slightly lower voltage and an angulated appearance of the wave form could also be noticed. These slower frequencies were marked in the last minutes of the bypass and particularly just after the tournquets around the venae cavae were released.

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TABLE 1
Changes in the Electroencephalogram Before, During and After Brpass in 18 Patents

| $\begin{aligned} & \text { Patient } \\ & \text { No. } \end{aligned}$ | Age (years) | Diagnosis | Before Bypass |  | During Bypass |  |  |  |  |  | After Bypass |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  |  | Complication | Blood Pressure | Chanes of EEC |  |  |  | Tmproved EEG | Blood Pressure | Abnormality | Reactivity |
|  |  |  |  |  | 1 | II | III | $\begin{aligned} & \text { Duration } \\ & \text { (minutes) } \end{aligned}$ |  |  |  |  |
| 1 | 6 | V.S.D. | 0 | 86 |  |  | $+$ | 8-10 |  | 55 | $\pm$ | $+$ |
| 2 | 4 | V.S.D. | 0 | 90 |  |  | $+$ | 9 |  | 50 | $+$ | 0 |
| $3 *$ | 4 | Tert Fallot | 0 | 95 |  | $+$ |  | 5 |  | 40 |  |  |
|  |  |  | $+$ | 40 |  |  |  |  | $+$ | 75 |  |  |
| 4 | 5 | V.S.D. | 0 | 100 |  |  | + | 5 |  | 60 | $\pm$ | $+$ |
| 5 | 58 | A.S.D. | 0 | 100 | $+$ |  |  |  |  | 100 | $\pm$ | + |
| 6 | 6 | Tetr Fallot | $+$ | 88 |  | $+$ |  | 3 |  | 50 | + | 0 |
| 7 | 62 | V.S.D ${ }^{\text {a }}$ | 0 | 115 |  |  | $+$ | 15 |  | 60 | Nullstadium | -Death |
| 8 | (20 months) | Pulmonary valve stenosis | 0 | 88 |  | $+$ |  | 30 |  | 50 | + | $+$ |
| 9 | (4 months) | Aortic valve stenosis | $+$ | 20 |  |  |  |  | + | 50 | $+$ | 0 |
| 10 | 11 | V.S.D. re-op. | $+$ | 45 | $+$ |  |  | 6 |  | 55 | $+$ | $\pm$ |
| 11 | (11 months) | Pulmonary valve stenosis | $+$ | 65 |  |  | $+$ | $10+35$ |  | 25 | $+$ | $+$ |
| 12 | 34 | Pulmonary valve stenosis | + | 150 |  | $+$ |  | 2.5 |  | 50 | 0 | ? |
| 13* | 3 | Pulmonary artery stenosis | $+$ | 80 |  | $+$ |  | 35 |  | 50 |  |  |
|  |  |  | $+$ | 30 |  |  |  |  | + | 80 | Nullstadium | - Death |
| 14 | 4 | Tetr. Fallot | 0 | 80 |  |  | + | 5 |  | 50 | $\pm$ | $+$ |
| 15 | (7 months) | A.P.V. connections | + | 20 |  |  | $+$ | 45 |  | 10 | $+$ | 0 |
| 16 | 6 | A.S.D. | 0 | 110 |  |  | $+$ | 2 |  | 60 | $\pm$ | 0 |
| 17 | 3 | V.S.D. | 0 | 125 |  |  | $+$ | 5 |  | 60 | $\pm$ | $\pm$ |
| 18 | 8 | A.S.D. | 0 | 85 |  |  | $+$ | 5 |  | 50 | $\pm$ | $+$ |

[^1]TABLE 2
The Physiological Data Recorded m 18 Patieste Undergong Meart Lung Bypass and in Whom Detaled Obervations of the Electronvecphadogram Were Made

| $\begin{gathered} \text { Patient } \\ \mathrm{No} \end{gathered}$ | $\begin{aligned} & \text { Weight } \\ & \text { ong } \end{aligned}$ | Morke | During Bypass |  |  | pH |  |  | $\mathrm{CO}_{2}$ |  |  | HCO |  |  | Bypase Duration (minutes) | Lowest Rectal Temperature (degree C ) |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  |  | $\begin{aligned} & \text { Arterialo } \\ & \text { (wat cent) } \end{aligned}$ | $\begin{aligned} & \text { benous } g^{2} \\ & \text { Eater certy } \end{aligned}$ | Blood | B.B. | DB. | $A B$ | B.1. | D. ${ }^{\text {a }}$ | AB | 18.B. | D.B. | AB |  |  |
| 1 | 16 | 125 | 100 | 65 | $60-75$ | 7.55 | 7.31 | 7.3 | 21 | 35 | 18 | 18.5 |  | 16 | 34 | 34.5 |
| 2 | 16 | 106.3 | 100 | 54 | $-75$ | 7.58 | 7.39 | 7.4 | 21 | 35 | 21 | 20 | 20.5 | 15 | 89 | 33 |
| 3 | 13.6 | 95.6 | 100 | 82 | $-70$ | 7.53 | 7.30 | 722 | 19 | 30 | 36 | 17 | 18 | 14.2 | 115 | 31.8 |
| 4 | 15 | 100 | 100 | 81 | $65-70$ | 7.50 | 7.39 | 7.50 | 23 | 36 | 25 | 21.5 | 21 | 19.5 | 20 | 31.8 |
| 5 | 45 | 91.1 | 93 | 74.5 | 100 | 7.57 | 7.31 | 7.52 | 22 | 48 | 24 | 20.5 | 22 | 20 | 35 | 35 |
| 6 | 15.8 | 101.3 | 100 | 70 | $50-65$ | 7.44 | 7.22 | 7.31 | 23 | 48 | 24 | 16.5 | 18 | 12.6 | 58 | 32.5 |
| 7 | 20 | 100 | $\square$ |  | 70-100 | 7.36 | 723 | 7.40 | 38 | 50 | 27 | 20.5 | 19.5 | 17 | 25 | 33.8 |
| 8 | 12 | 83.3 | 100 | 70 | $45-90$ | 7.44 | 7.51 | 7.46 | 26 | 17 | 18 | 18 | 15 | 11 | 33 | 32.5 |
| 9 | 3.39 | 88.5 | - | - | 40 | - | - -8 | - |  | 1 | 1-- |  | 10. |  | 29 | 34 |
| 10 | 24 | 95.8 | 99 | 70 | 55 | 7.54 | 7.28 | 7.4 | 26 | 4 | 175 | 22 | 19.5 | 17 | 54 | 34 |
| 11 | 8 | 100 | 100 | 56 | 25 | 7.48 | 735 | - 14 | 24 |  |  | 18 |  |  | 35 | 37.5 |
| 12 | 65 | 76.9 | 89 | 74 | 110 | 7.58 | 7 | 7.44 | 28 | 50 | 36 | 26 16 | 26. | 23 | 24 49 | 35 |
| 13 | 12 | 75 | 100 | 68 | 5068 | 7.24 | 7.07 | 6.85 | 28 | 54 | 62 | 16 | 13.7 | $18^{92}$ | 49 +1 | 34 345 |
| 14 | 17.5 | 97.1 | 100 | 81 | $30-50$ | 7.47 | 7.20 | 744 | 24 | 5 | 24.5 | 18 | 20 | 18 | 41 | 34.5 |
| 15 | 4.8 | 83.3 |  | - | 1540 | - | -18 |  |  |  |  |  | 18 | $1-1$ | 57 | 32, |
| 16 | 17 | 94.1 | 100 | 79 | 75.90 | 742 | 7.18 | 7.35 | 30 | 33 | 52 | 19 | 18 | 17.5 | 29 | 33.1 |
| 17 | 12 | 91.7 | 100 | 86 | $62-75$ | 7.50 | 7.28 | 74 | 19.5 | 35 | 21 | 16 | 16 | 15 | 33 | 32.5 |
| 18 | 24.5 | 150 | - | - | 50-70 |  |  | 7.39 |  |  | 29 |  |  | 14 | 27 | 33.5 |

B.B. D.B., A.B = Before, during, after bypass.

In the remaining four electroencephalograms, further important alterations occurred during the bypass. In one patient (No. 2) the bypass period was prolonged ( 89 minutes). A secondary slowing was apparent after the fiftieth minute of bypass. In two other patients (no. 11 and 15) after a slight recuperation following the initial alterations, serious changes occurred in relation to severe hypotension (under 25 mm . of Hg ). One of the tracings showed numerous periods of depression of 15 seconds duration (no. 15). Slight rises in blood pressure were concomitant with the electroencephalographic improvement and the aggravation of changes in the tracings coincided with recurrence of hypotension. The fourth patient (no, 13), 3 years old with localized stenosis of right and left pulmonary arteries, had serious hypoxia before the bypass period. The total bypass time was 49 minutes. The blood pH was 7.24 previous to the bypass and 7 to 7.07 during the bypass; the arterial $P_{\mathrm{cos}}$ changed from 28 to 54 . The electroencephalographic tracing was flattened for 40 minutes.
Modifications Immediately After the Bypass. When the bypass was discontinued a deterioration of the electroencephalographic pattern was evident on ten occasions. In one tracing there was a slight improvement and in nine others no important change was present.
However, if the immediate post-bypass electroencephalograph was compared with the one obtained before the bypass there was a modification in all but one. In eight of these, the alterations were marked and in eleven, moderate (fig. 5).

Tracings Near the End of the Operation (60 to 90 minutes after the bypass). In eight of the eighteen patients studied the electroencephalogram became normal or slightly subnormal (slow rhythm) at the time of chest closure. In eight patients many slow waves were clearly present. In two patients a heart deficiency gave an irreversible extinction of the brainwaves (Creutzfeldt's Nullstadium) leading to death. An electrical awakening, represented by an acceleration of the brain wave with lower voltage, was apparent in all except these latter two patients at the moment of spontaneous respiration. On nine occasions we
saw the reappearance of reaction to stimulii (pinching, etc.).

The physiological and biochemical data are presented in tables 1 and 2 .

## Discussion

During these surgical procedures the variables are innumerable and the electroencephalogram will not only be dependent upon the efficiency of the mechanical devices employed for the heart-lung bypass.

It is probable that the modifications showed by the electroencephalogram were an expression of the cerebral suffering and show the sensitivity of nervous centers to a physiological change.

We were confronted with probable ischemic hypoxia of the brain, with the exception of case 13 where we had hypoxic hypoxia. The effects of anoxia and ischemia were unable to be differentiated by the electroencephalographic recording. They consisted of a sequence of changes which began by an amplification and slowing of the tracing as was shown first by Berger in 1934.5 If the hypoxia was slight or moderate, no further modifications of the electroencephalogram appeared. When, however, the hypoxia was severe, the slow waves were followed by a suppression and in the worst instances by total extinction. A description of these electrical changes, as observed in the individual brain cells of cats, was given by Creutzfeldt: ${ }^{6}$

In this study we were interested in the aetiology of these numerous changes of the electroencephalogram as well as in their incidence and prognostic significance.

Aetiology of Electroencephalographic Changes at the Beginning of the Bypass. It seems that these were dependent on (a) cerebral conditions present previous to the bypass and (b) new conditions created by the bypass.
Condmons Previous to the Bypass: These can be dependent upon factors such as anaesthesia, age of the patient, nature of the heart anomaly and events occurring before the bypass.

Anaesthesia was that evolved over the last few years at this Hospital for all chest surgery, and modified to encompass the bypass period. Sedation with pentobarbital, morphine and
scopolamine and, as required, rectal thiopentone, ensured that the patient arrived in the operating room without anxiety.

Induction of maesthesia was with mitrous oxide and ether, slowly administered until conditions were ideal for orotracheal intubation. All patients were hyperventilated throughout the period of open chest with 50 per cent nitrous oxide and 50 per cent oxygen, and the muscle relaxant, Landexium, enabled complete control of respiration. Just prior to the bypass more relaxant was given and throughout the total bypass movement of the lungs was suspended and they were filled with a mixture of 50 per cent helium and 50 per cent oxygen, mantained at a pressure of about 10 cm . of water, the degree of distension depending on the surgical exposure.

Minor changes of techmique, such as no sedation of infants and the use of a relaxant for tracheal intubation of adults, were individualized and occasionally Neostigmine was given at the end of the operation to comiteract persistent relaxation.
After the induction, the electroencephalographic tracing was generally stable and showed the characteristic pattern of light anaesthesia under nitrous oxide, which is represented by waves varying from 15 to $18 \mathrm{c} / \mathrm{s}$ with some slower waves 5 to $10 \mathrm{c} / \mathrm{s}$. This light type of anaesthesia made the appreciation of other modification of the electroencephalogram possible. The initial tracing of this type was unchanged throughout the pre-bypass period in eleven of the eighteen patients.

There were two adults, the remainder were children of four months to eleven years of age and of these nine were from four to six years of age.

Contrasted with the normal findings without anaesthesia, the tracings did not usually show characteristics that could be assigned to age. Only in the two youngest infants (four and seven months of age) could we appreciate a difference by the presence of slow waves of 2 to $3 \mathrm{c} / \mathrm{s}$ with a few rapid waves.

From our previous experience we know that in some patients with chronic hypoxic diseases (e.g., heart failure, tetralogy of Fallot, or pul monary stenosis) the tracings showed extreme slowness even when the anaesthesia was clinically light. In the present study two patients


Fic. 6. A one year old patient with a ventricular septal defect (no. 10). (a) The electroencephalogram appearance before bypass during excessive haemorrhage, while catheterizing the superior vena cava, Previous blood pressure $90 \mathrm{~mm}, \mathrm{Hg}$. (b) The electroencephalogram 30 seconds later (blood pressiure stable but low). (c) Recovery apparent 3 minntes later.
with tetralogy of Fallot (no. 3 and 6) had slightly slower waves than usual as did two patients with pulmonary stenosis (no. 8 and 11).

In nine of the twenty bypass electroencephalograms studied some incident occurred before the bypass which led to electrical changes in the electroencephalogram (fig. 6).

Without any complication, the catheterization of the superior vena cava usually caused minimal changes in the electroencephalogram. Important disturbances were shown only twice (no. 6 and 15) and these could be assigned to temporary obstruction of the superior vena cava with impairment of retum venous How: (fig. 7, a, b, e).

New Conditoons Createi by the HeartLung Bypass: A sereen pump oxygenator of the Gibbon type ${ }^{9}$ was used during these operations. Arterial flow varied from 80

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Fis, 7 . A 6 ycar old patient with tetralogy of Fallot (no, 6). (a) Slower, larger electroencephalogran waves appearing at the moment the superior vena cava was catheterized. (b) Marked change in the clectroencephalogram shown one minute later. (c) Some improvement apparent 5 minutes later.
to $100 / \mathrm{ml} / \mathrm{kg}$. minute. The oxygenated blood was returned via the right femoral artery.

Efficient oxygenation was obtained by passing 97 per cent oxygen and 3 per cent carbon dioxide over the blood film in the artificial lung. The atterial blood samples taken from the patients before bypass usually revealed a high $p \mathrm{H}$ and low $\mathrm{P}_{\mathrm{CO}}$ level, this respiratory alkalosis being due to hyperventilation. Arterial blood samples taken during late bypass showed a return to normal level of $P_{\text {ro }}$ and a reduction of pH .8

At the beginning of the bypass, soon after the pump oxygenator took over the patient's circulation, a drop of atterial blood pressure usually occurred. (14 times in 20 periods of bypass.) This drop in pressure was of short duration and then a stable pressure was established. After a period varying from 10 to 90 seconds the blood pressure was usually over

50 mm . of mercury throughout the rest of the bypass. In a few instances the blood pressure during bypass went below 50 mm . of mercury, for short periods of time. This happened during the bypass in infants under twenty months of age and 12 kgm . of weight (no. $8,9,11$ and 15).
Body temperature was continuously recorded by means of a rectal thermocouple. The lowest recorded temperature was 31.8 C . To prevent an excessive drop in body temperature the oxygen was heated before going to the artificial lung by passing the gas through water heated to 85 C . The patient was also warmed by a heated mattress.
The principal factor responsible for the alterations of the electroencephalogram seemed to be changes in mean arterial pressure. It remains to be determined why changes of arterial pressure of similar magnitude may not lead to electroencephalographic changes of identical severity or duration in different patients. The physiological variations we recorded during the bypass do not seem to explain this difference. It is well known that a fall in body temperature has influence on the electroencephalogram, but considering the range of temperature in our patients $(37.5 \mathrm{C}$. to 31.8 C .) no correlation is thought justifiable.

Beyond a possible individual susceptibility, the previous condition of the brain before bypass seems to be of importance. In the ten patients where important alterations of the electroencephalogram (type III) occurred at the beginning of the bypass, there were eight in whom no serious incident arose before the bypass. This suggests that when there was no complication before the bypass the brain was maintained in a stable condition and underwent only small physiological adjustments. Thus, when the bypass was begun, because of the hemodynamic disturbances then created, a severe imbalance was produced. The gravity of this disorder and the difficulty of adaptation of the brain were manifested in the electroencephalogram. An adaptation was made after several minutes ( 2 to 15 ) but usually the arterial blood pressure remained low. The different speed of recuperation could be explained by variations of the cerebral vasomotor activity or it may have been caused by a diminution in consumption of oxygen during
the bypass ( 50 per cent in the experiments of Creech, et al). ${ }^{.}$

On the other hand, in two patients in whom alterations of Type III were recorded some complication did occur in the preparatory period during catheterization of the vena cava (no. 11 and 15). In these bypass was accompanied by a serious depression of blood pressure 25 and 15 mm . of merenry mean respectively).

Of the seven patients wheren the alterations were slight to moderate at the begiming of the bypass (types I and II) there were four in which complication before the bypass produced permanent or temporary alterations in the electroencephalogram. These complications were produced by mechanical factors (e.g. Clamping of the pulmonary atery or obstruction of the superior vema cava) or by hypotension (due to haemomage) or by inadequate cardiac output (after a previons episode of hypotension or a period of bypass).
There were three patients of this group in whom no complication was noticed before the bypass. In one we could see a small rise of blood pressure at the begiming of the bypass and the other two were patients with chronie hypoxia in which the hypoxia could have al ready stimulated compensatory mechanisms.

In the three patients in whom the electroencephalogram improved, there had been a complication before the bypass and the bypass raised the blood pressure. Two of these had had low blood pressure following a previous bypass period.

In the presence of one or several of the above-mentioned factors we might suppose that the brain, under repeated insults, had to bring forces into play to combat the circulatory disturbances and the metabolic upset. We can believe that at the moment when the bypass was started the brain was already adapted to precarious conditions. The compensation mechanisms for this physiologic acclimatiotion' ${ }^{10}$ are produced at the expense of physiological reserves but the retum to normality then becomes problematic.
Interpretation of the Electroencephalogram During the Remainder of the Bypass. After the period of initial alterations the electroencephalogram tracing usually improved. The waves were not, however, similar to those
previous to the bypass. Slow waves still remained in the majority. Substitution of cardiac systole by a nonpulsatile artificial circulation may be enough to explain this difference.

We have not noticed evidence of cerebral air embolism in these patients but believe that synchronous bilateral tracings are necessary to detect any bit massive amounts of air.

The influence of hyperventilation on cercbral oxygenation $t 1$ is recognized, but the degree necessaty to be significant is undecided as is the effect of mised oxygen tension in the bood. And we do not know the significance of temporary interference with cerebral cellular activity under operating conditions, e, e, hypothermia, annesthesia.

The intemption of anaesthesia durng the bypass will not explain the slowing in the electroencephalogram becuuse electrical awakening upon withdrawal of this type of anaesthesia is nomally matked by acceleration of the waves and not by slowing. It is of interest that all patients treated with the bypass technique have had no sedation or anaesthetic added during the bypass but have no memory of the event. We cannot explain this fortunate phenomenon.

Electroencephalogram After the Bypass. The alterations which were present after the bypass were related to the lowering of aterial blood pressure and all were improved or disappeared when the pressure became nomal.

In nearly all patients the electroencephalogram after the bypass remained changed in comparison to the electroncephalogram previous to the bypass. This change varied from a few to an excess of slow waves.

This slowness may have been favoured by the amaesthetic drug given after the bypass, but it is more probable that the changes were dependent on the special conditions under which the bran functioned during the bypass. Halley et al. 22 have shown an increase in cerebral volume after bypass which suggests the possibility of cerebral oedema.

Relation of the Electrocticephalogram to the Recotery. The electrocncephalogram will not give the whole prognosis of recovery but only one aspect of it. The electroencephalogram is only the cerebral reflection of the physiopathological conditions that develop during surgery.

If we try to establish some prognostic rela-
tion we may say that prognosis is not related to the gravity of the alterations that appear at the beginning of the bypass but with the succession and combination of events during the whole procedure. It seems that the total duration of the periods of slow waves before, during and after the bypass is very significant.

After the bypass sixteen patients regained full consciousness, but those who had a prolonged abnormal tracing or no electroencephalographic response to stimulii had a grave prognosis as regards total recovery. In our observations of three children in whom the tracings showed slow waves for periods of 70 , 72 and 105 mimutes respectively (no. 9, 13 and 15), death occurred soon after the end of the operation without an apparent surgical explanation.

Failure of nommal electrical activity to reappear at the end of the operation was a grave sign and in five of sis patients was followed by death sometime in the postoperative period. The absence of reactivity does not explain the death itself, but it is a sign of serious cerebral disturbance which may lead to vital brain stem damage.

## Summary

The electroencephalogram and intra-arterial blood pressure were recorded continuously throughout twenty periods of heart-lung bypass during the surgical correction of congenital heart defects in 18 patients.

Some change always occurred in the electroencephalographic waves when the patients underwent a period of heart-lung bypass. Examples of the changes are presented with interpretation and discussion of their significance.

The wave form may be influenced before the bypass by anaesthesia, age of the patient, nature of the heart anomaly and surgical events. At the beginning of the bypass we have recognized four types of initial modifications of wave form their degree and duration is associated with mean blood pressure changes and may be related to the state of the brain just before the bypass. Throughout the remainder of the bypass time and afterwards the majority of patients had an electroencephalogram which was different from that recorded before the bypass.

If the abnormality of wave form was marked and prolonged and if there was no cerebral electrical response to stimulii after the operation, even if consciousness is regained there may be damage to the brain of a magnitude sufficient to affect the whole recovery process.

The changes recorded electroencephalographically may be cumulatively detrimental to the patient's welfare. Perfection of the bypass techniques may be possible by correlating these electrical changes with the detailed clinical happemings. Also, study of all aspects of cerebral function with psychological and psychophysical tests will be necessary to indicate the value of the electroencephalogram during these operations.

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[^1]:    * Two periods of Bypass, V.S.D. = Ventricular Septal Defect. A.S.D. - Interarterial Septal Defect, A.PV. = Anomalous Pulmonary Venous. encephalographic change is indicated. After bypass the change in appearance of the electroencephalogram compared with that before bypass and the response to peripheral stimulation is indicated.

[^2]:    The Mark Co., Randolph, Mass.

