Some observations on the fluttering midline echo in echoencephalography

A ballistocardiac effect and suggested cause of rupture of the septum pellucidum

D. N. WHITE AND C. O. JENKINS

From Queen's University, Kingston, Ontario, Canada

SUMMARY In cases of hydrocephalus, echoes from the region of the cerebral median sagittal plane may show a fluttering variation both in amplitude and range. Evidence is presented that, in the case studied, these movements arose from the falx cerebri and that they were caused by ballistocardiac forces presumably setting the CSF in the enlarged lateral ventricles into resonance within the enlarged cranium. Similar movements would be expected in the lateral ventricular walls as well as the septum pellucidum when the latter is imperforate. It is suggested that the lowering of the resonant frequency of the ventricular CSF in cases of hydrocephalus with both large ventricles and large heads allows ballistic and acceleratory forces applied to the hydrocephalic head to cause large pressure changes between the two lateral ventricles with consequent lateral movement of the midline structures separating them and possible rupture of the septum pellucidum, as is commonly found in hydrocephalus.

When an ultrasonic transducer, coupled to an A-mode display system, is applied to the parietal region of the skull of some patients with hydrocephalus, an echo appears in the midline that exhibits a peculiar fluttering fluctuation in amplitude. This flutter which, on the expanded display, can be seen to occur not only in amplitude but also in range, is reminiscent of a flag fluttering in a strong breeze.

Although the phenomenon of the fluttering M-echo has been known to echoencephalographers for a long time, it appears to have been mentioned only briefly in the literature (McKinney, Kato, Pou, and Thurstone, 1966). They believed it resulted from a tear in the septum pellucidum but stated that a similar fluttering echo had been seen in two patients at a depth corresponding to that of the lateral wall of the lateral ventricle and which therefore could not be arising from the septum pellucidum. With this exception, this strange and striking phenomenon does not appear to have been studied.

PATIENTS

It has been seen only in patients with hydrocephalus. Recently, we examined 14 patients with documented hydrocephalus, who were inmates of an institution caring for people with subnormal intelligence. Seven of them were seen to have a fluttering midline echo. The age of these seven ranged from 2 to 45 years. They all had obviously large heads. Only one of the other seven in whom it was not seen had an obviously large head, and that was curiously deformed, being flattened from side to side, so that the transverse diameter was smaller than normal. Thus the fluttering echo occurs in patients whose hydrocephalus was active before closure of the sutures, and whose ventricles are enlarged especially in the coronal plane, with probably a very thin cerebral mantle. The large ventricles show on the A-mode display as wide anechoic areas on either side of the fluttering echo. It was not seen in patients who did not have this anechoic area. It was not seen in one patient with large ventricles but a normal sized head and who had had a ventricular shunt performed.

Another patient, aged 51, was studied in more detail by means of equipment capable of writing out, on line, fluctuations in the amplitude or range of one cycle of an echo pulse. The equipment used will be described elsewhere (Clark, White, Curry, and Campbell, 1971). This one patient had had hydrocephalus from childhood and had a large head and
large ventricles (Fig. 1). She had never been subject to any ventricular shunting procedure.

The fluttering echo could be displayed only when the transducer was placed on the lateral surface of the skull and directed medially. It could not be displayed when the transducer was placed on the frontal, vertical, or occipital regions of the skull. It must therefore be arising from an interface which is perpendicular to the bitemporal or biparietal axis—that is, which lies in the sagittal plane. It was optimally displayed when the transducer was placed in the parietal area and directed medially. This was above and behind the conventional above-pinna position from which it was less well displayed. As the transducer moved anteriorly, though the M-echo was still present, the fluttering echo diminished in amplitude until only the M-echo remained, exhibiting the normal cardiac-induced amplitude pulsations. The single sagittal midline interface from which it arose was therefore presumably of greater area in the biparietal axis, less in the bippina axis, and least in the bitemporal axis anteriorly. Its extent in the other coronal axes, where the transducer axis could not be kept normal to the median plane, could not, of course, be determined.

It is suggested that only the falx cerebri could provide a reflecting interface that would fulfil all these criteria. Confirmation of this explanation of the source of the fluttering echo is suggested by the fact that the echo could be displaced quite markedly from its normal midline position by causing a rapid lateral flexion of the head in the coronal plane. It was then seen, momentarily, to lag behind the direction of movement quite markedly—as much as several millimetres of tissue. A lesser displacement accompanied rotation of the head around the vertical axis and no obvious displacement followed movements in the horizontal plane. Its behaviour with movements in the vertical plane was not studied.

It is significant that large displacements of the fluttering echo in the coronal plane are followed by displacements to either side of the midline for a short period, suggesting that the displaced interface is part of a highly damped oscillatory system.

**SOURCE OF FLUTTERING ECHO** The fluttering echo, unlike the M-echo, was never seen to be paired even on the expanded display. It always consisted of a single pulse train, suggesting that it arose from a single thin interface rather than a number of parallel interfaces as is the case with the M-echo (White, Clark, Campbell, Chesebrough, Bahuleyan, and Curry, 1969a; White, Clark, and Campbell, 1969b). It was always seen precisely in the midline when the patient's head was upright. The originating interface therefore must be in the cerebral midline.

In all cases it was easy to display at very high amplitude, usually higher than the M-echo itself, which could be distinguished from it by the M-echo's regular variation in amplitude with cardiac systole. It could be displayed from a fairly wide area on the lateral surface of the skull. Both these features suggest that it arose from a single interface of large area. They also suggest that, while this interface could be a component of the multiple median and paramedian interfaces which we believe give rise to the M-echo (White et al., 1969a, b), most, if not all, of these multiple interfaces are separate from the interface from which the fluttering echo arises.

**CAUSE OF FLUTTERING ECHO** The fluttering fluctuation so readily apparent in the amplitude of the echo is accompanied by similar irregular fluctuations in the range of the echo (Fig. 2). It will be noted, however, that the form of the fluttering fluctuations are not necessarily similar in amplitude and range. While both show an irregular pattern, the flutter in amplitude displayed by the right hand transducer in Fig. 2 is quite different from the flutter in range. At other times, however, some similarity exists between them, as seen by the two types of flutter displayed by the left hand transducer.

If the echo were arising from a single planar inter-
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PM.31.  

ECG  

RESPIRATION  

AMPLITUDE from RIGHT  

AMPLITUDE from LEFT  

RANGE from LEFT  

RANGE from RIGHT  

1 second  

FIG. 2. Recording of the fluctuations in range and amplitude of the fluttering echo recorded simultaneously by transducers on the left and right parietal regions with simultaneous recording of ECG and respiration. The fluctuations in amplitude and range are different as recorded by the right-sided transducer. This difference is less for those recorded by the left sided transducer. The shapes of the fluctuations in amplitude are different as recorded by the two transducers but the fluctuations in range are similar but show a difference of 180° in phase. While the fluctuations both in range and amplitude are very irregular, they show a tendency to occur at approximately twice the heart rate and the shape of the fluctuations in range show a tendency to repeat themselves at the heart rate.

face of relatively large area, which was moving in range in a complex fashion, it would be expected that the amplitude of the echo would also fluctuate in an irregular fashion which need not be synchronous with the fluctuation in range. Such fluctuations, however, could be synchronous if the variations in range were accompanied by parallel variations in specular reflections back to the transducer. In just the same way, reflections of sunlight from the surface of a lake will not necessarily synchronize with the rise and fall of the ripples and waves disturbing the surface.

However, even if the fluctuations in range and amplitude would not necessarily be synchronous, it would be expected, if the reflecting interface were single and echoes from approximately the same area were being received by two transducers homologously placed on either side of the interface, that the fluctuations both in range and amplitude recorded simultaneously by the two transducers would be 180° out of phase. Such was usually the case as can again be appreciated in Fig. 2. That more exact phase reversals were not seen in amplitude was presumably due to the irregular scattering of the intensity of the insonating beam by the skull, despite the symmetrical placement of the two transducers (White, Clark, White, Campbell, Bahuleyan, Kraus, and Brinker, 1969c). Such an irregular scattering of the energy from the separate transducers would result in the same regions of the reflecting interface receiving different intensities of the insonating energy from either side at any one moment and
hence returning reflections of different amplitude. The phase inversion of the range fluctuation was usually more precise as would be expected if the reflecting interface were moving as a unit over a relatively wide area, albeit in an irregular fashion. While the fluctuation in both range and amplitude of the fluttering echo was always irregular, most of our recordings, especially in range, showed some tendency for a pattern to repeat itself with every cardiac systole (Fig. 3). Such an observation suggested that the prime mover of the movement was the cardiac contraction.

It was natural to consider if the movement in range resulted from the arrival of the systolic pressure pulse in the head. We concluded that this was not the case when repeated occlusion of one or other carotid artery failed, on even a single occasion, to modify the pattern of variation in either movement or amplitude that we recorded.

We also noted that the fluttering echo did not disappear when air was introduced into the ventricles for pneumoencephalography. In this procedure, however, only a small fraction of the ventricular fluid was replaced by air (Fig. 1).

We did note that not infrequently the irregular movements recorded were reminiscent of the irregular movements that used to be recorded by ballistocardiography. Indeed, a few of our recordings showed a striking similarity to the normal ballistocardiogram described by Starr and Schroeder (1940) and illustrated in the insert in Fig. 3.

That the movement we record is indeed the result of ballistic forces is suggested by the further observation that the fluttering movement can be largely damped when the patient lies prone on a mattress and pillow. In these circumstances the movement of the fluttering interface assumes a more regular relationship with the cardiac pulse (Fig. 4). If the bed or stretcher on which the patient is lying is then lightly tapped a marked oscillatory movement of the fluttering interface is recorded and this is accompanied by a similar oscillation of the amplitude of the echo (Fig. 4). These oscillations diminish in amplitude in the fashion typical of a damped resonant system before reverting to their pre-percussive pattern. That such oscillations are not due to movement artefact of the transducers can be appreciated by the fact that the through transmission pulse continuously measuring changes in the distance between the two transducers shows no accompanying movement. It is also shown by the fact that recording by the same transducer of the echo amplitude and range from another cerebral interface away from the midline and presumably in the cerebral mantle also shows no such oscillation after percussion while writing out a trace typical of the amplitude and range pulsations recorded from intracerebral interfaces (Clark et al., 1971).

The same observations can also be made on the fluttering midline echo when the patient is sitting upright and its fluttering movement is more obvious.

A light tap on the patient’s body produces a very marked change in the amplitude and range of the fluttering echo, which then ‘rings down’ as would be expected in an oscillatory system (Fig. 5). No similar effects were seen in echoes from interfaces in the cerebral mantle.

**DISCUSSION**

We believe therefore that the striking echoencephalograhic curiosity of the fluttering M-echo arises in our case from the falx cerebri and that it resulted from ballistocardiac movement.

It is interesting to speculate why it is seen only in cases of hydrocephalus with large heads. The increased size of the head will enlarge the region in the sagittal median plane from which echoes can be received by a laterally placed transducer applied tangentially to the skull (White, 1970). In persons with normal sized skulls their curvature prevents echoes that originate from the falx being received at high amplitude by an extracranial receiver. In hydrocephalus there is a larger area over the side of the skull from which the transducer can be applied tangentially to the surface and its axis still remain normal to interfaces in the sagittal plane.

**FIG. 3. Recording of the fluctuations in range of the fluttering echo with a transducer placement that emphasizes the tendency for these variations to repeat themselves at the heart rate. The inset is a diagram of the normal ballistocardiogram (Starr and Schroeder, 1940) which closely resembles the range fluctuations recorded here.**
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FIG. 4. Recording of the amplitude and range variations of the fluttering echo in the midline and another echo internal to the near side of the skull and thought to originate from an interface in the left cerebral mantle. This interface moves away from the transducer towards the centre of the head after systole as is the case with all cerebral interfaces. In order to monitor for movement artefact the recordings from both echoes are made by a single transducer placed on the left of the head and, at the same time, the range of this transducer from a second transducer placed on the homologous region on the right of the head is continuously measured by means of through transmission pulses (Clark et al., 1971). The recording was made with the patient prone and the variations in range of the midline echo should be contrasted with those shown in Figs. 2, 3, and 5, where the patient was upright. The range variations in this Figure show a much closer relationship to the cardiac systole. (ECG is not included in this Figure but systole coincides with a downward movement of the range recording from the left hemisphere echo.) The two sets of arrows on the traces from the midline echo represent two light taps given to the stretcher on which the patient lay. A marked oscillation of the range of the fluttering midline echo resulted of more than 0.25 mm and lasting 1 sec. No change is seen in the amplitude or range of the echo from the cerebral mantle recorded by the same transducer and only minimal movement artefact is recorded by the through transmission pulses (arrows).

Thus it should be possible in hydrocephalics to receive echoes from the falx cerebri to a greater degree than in normal persons. If the falx cerebri were normally moved by ballistocardiac forces, it would be easier to display the effects of such movement in hydrocephalics. It seems unlikely that this explanation accounts for the phenomenon, since the fluttering echo is so striking in appearance that glimpses of it would have been expected to have been observed in persons without hydrocephalus, especially with dolicocephalic skulls, if the falx cerebri were normally in ballistocardiac movement.

It seems more probable that the movement observed is due to a change in the resonant properties
of the intracranial contents resulting both from an increase in size of the calvarium and also from an increase in the quantity of the lateral ventricular CSF. That both factors are necessary is suggested by our inability to find a fluttering echo in cases of cerebral atrophy with large ventricles unless the skull were also enlarged. Such changes could result in ballistic movement of the ventricular fluid with each heart beat and consequent fluttering of the falx cerebri. It is perhaps significant that cases showing the fluttering echo do not show many other echoes from the region of the cerebral midline, so that the cerebral mantle, on either side of the falx cerebri and which might be expected to damp its oscillations, may well be very thin in such cases. Such an explanation would also readily account for the fact that ballistic forces applied to the patient appear to be the only easy method of modifying the movement and that such percussion causes an oscillation that decreases just as does the oscillation of a damped resonator.

If this should be the explanation of the fluttering echo observed in our case, then the fluttering movement of the falx cerebri is a movement imposed upon it by resonant oscillations of the ventricular CSF. This oscillatory movement of the intraventricular fluid will, of course, not cause movement only in the falx cerebri between the two hemispheres. Similar movements would also be expected to be imposed upon other periventricular structures such
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as the septum pellucidum and the walls of the lateral ventricles. This would explain the observation of McKinney et al. (1966) who saw a fluttering echo arising from the region of the walls of the lateral ventricles. The same explanation would also account for the belief that the fluttering echo may arise from the septum pellucidum. Some observers have felt that its fluttering movement might be due to perforation of the septum pellucidum, which is commonly present in cases of hydrocephalus. If our explanation is correct, then the intact septum pellucidum would be expected to show similar irregular ballistic movements to the falx cerebri and would be capable of giving rise to an irregularly fluttering echo. That we could not record the fluttering echo well from the anterior regions where the axis of the transducer would be perpendicular to the septum pellucidum, we suggest was due to the perforation and absence of the septum pellucidum in our case. It is common in cases of longstanding hydrocephalus for the septum pellucidum to perforate and be largely destroyed. It was unfortunate that the air study did not display the septum pellucidum in our case so that this hypothesis could be tested. We do not believe that the fluttering echo can arise from the fragments of a perforated septum pellucidum as has sometimes been suggested. While such fragments might be expected to move irregularly in the ventricular cavities their torn surfaces would, at any one moment, lie perpendicular to the insonating beam only over a small area and thus would not be capable of giving rise to an echo of very large amplitude nor would the amplitude remain consistently large, as is always the case with the fluttering echo. Moreover, the pattern of the irregular movements of the interface would not be expected to be similar over such a wide area as we found to be the case, and especially would not be expected to be of highest amplitude over the parietal area. Finally, it seems unlikely that the pattern of movement of such fragments would be likely to be as repetitive as we found to be the case.

It is reasonable to suggest that these oscillatory movements of the ventricular CSF might be a contributory factor in causing rupture of the septum pellucidum which is so commonly found in cases of hydrocephalus. Perforations of the septum pellucidum in hydrocephalus have usually been ascribed to stretching of the septum pellucidum (Davidoff and Epstein, 1950). While the symmetrical pressure changes between the two ventricles resulting from ballistocardiographic forces would subject the septum pellucidum to physical stress which does not occur in normal persons, it seems more likely that it is the much greater forces that accompany ballistic or acceleratory forces to the head that could rupture the septum between the lateral ventricles. Presumably, it is the lowering of the resonant frequency of the ventricular fluid that allows these ballistic and acceleratory forces to cause such asymmetrical pressure changes between the two ventricles that the midline structures separating them, at times, move laterally through some millimetres. It is easy to conceive the thin septum pellucidum rupturing in these circumstances. In persons with normal sized heads and ventricles the natural frequency of the resonant oscillation of the fluid in the lateral ventricles would be expected to be higher and more highly damped so that displacement of the structures separating the two ventricles after blows or acceleratory forces would be less marked. For this reason, presumably the much greater acceleratory forces that accompany head injuries only infrequently cause rupture of the septum pellucidum.

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REFERENCES


ADDENDUM

Since this paper was accepted for publication, the patient on whom these studies were made died of inanition. At necropsy, the cause of her disease was found to have been a basal arachnoiditis which completely obliterated the foramen of Magendie. The whole ventricular system was greatly dilated including the foramina of Monro, the third ventricle, the aqueduct and fourth ventricle as well as one
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foramen of Lushka. Our prediction that the septum pellucidum was perforated and absent, was confirmed (Fig. 6). Indeed the whole medial walls of both lateral ventricles were absent over an area $9 \times 6$ cm. No free fragments of the septum were present though anteriorly its eroded margin showed perforations.

It would appear that, for such a huge perforation to have such smooth and regular margins, erosive forces must have been present in the lateral ventricles over a very prolonged period. The same forces presumably account for the fact that the corpus callosum was very thin especially anteriorly where a perforation was present. Similarly, the floor of the third ventricle had perforated into the cisterna interpeduncularis. It would seem that ballistocardiac forces could cause such effects, though larger acceleratory movements may have been necessary to produce the ruptures found.
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