Sonic Detection of Intracranial Aneurysm and AVM

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SUMMARY This paper describes a method of detecting intracranial aneurysm and arteriovenous malformation (AVM) by analysing weak sounds produced by the blood circulation at the affected part. There is thought to be no turbulence in the normal cerebrovascular system, whereas abnormalities such as aneurysm and AVM sometimes cause turbulence in the blood flow. Thus, a small fraction of the flow energy might be converted into an acoustically detectable noise.

For the detection of the sound, sensitive detectors must be applied close to the head since the sound is very weak, and, as in cerebrovascular diseases, the origin of the sound is usually concealed deep inside the hard shelter of the skull. The detection system we used had a gain of 40 to 50 dB greater than that of an ordinary stethoscope. The detection points were the teeth or forehead. Usually the sound started about 160 msec after the ventricular contraction and lasted for 100 to 400 msec. Its frequency component mostly ranged from 400 to 2000 Hz, but the spectrum profile changed according to the position and degree of abnormalities. The uttered sound was very random, which facilitated detection of the position of the sound origin by means of cross-correlation methods using a pair of detectors.

This method is completely noninvasive, causes no pain to the patient, and might be used even in mass examinations.

THE ANEURYSM and the arteriovenous malformation (AVM) in the brain are potentially dangerous diseases because they often cause fatal hemorrhage without any symptoms. Several methods such as the ultrasound doppler method,1,2 the CT scanner method,3 and angiography4 have been tried for detection of the aneurysm and of AVM in some parts of the body before bleeding. However, these methods are less effective for intracranial applications; ultrasound cannot pass through the skull, the CT scanner method has low resolution, and angiography causes patients much pain.

Analysis of the weak sounds generated by the aneurysm and the AVM at the affected part is one of the more promising methods as it is noninvasive and causes no pain to the patient. Since Richardson and Kofman5 reported on the cranial bruits in 1951, the mechanism of the sound generation and its diagnostic significance have been widely discussed.6-9 Sounds generated by aneurysm and AVM are usually very weak and could easily attenuate in the process of propagation through the cranium. For example, though Ferguson6 did not detect any bruit at the surface of the head, he succeeded in recording the bruit in 10 cases out of 17 at the surface of the aneurysm exposed during surgery. For successful recording of the bruit noninvasively on the surface of the head, the choice of the recording position, a good transducer with high gain amplifier(s), and noise elimination technology are necessary. Olinger and Wasserman10 reported an apparatus for the recording of cranial bruit on the surface of the eyes and discovered a sharp spike in the frequency domain (250 to 800 Hz) corresponding to intracranial aneurysm.

Our bruit detection system has a pair of efficient detectors which can be applied anywhere on the head surface, and a special detector applicable to the tooth. The frequency range is wide enough (up to 2.5kHz) to detect bruits not only uttered by aneurysm, but also by AVM. To improve the signal to noise ratio, a signal gate inserted for the data processing procedure is controlled by electrocardiogram (ECG) signal and by excessive environmental noise signals. The cross-correlation technique gives a rough determination of the sound generation site. Our system is completely noninvasive and can be used even with children and in mass examination.

Method

The block diagram of the bruit recording system is depicted schematically in figure 1(a). In the system, three detectors are available; a pair of the detectors were usually placed on the surface of the head at symmetrical positions, and the third was used to detect the bruit from the tooth or to search for the best position for the recording, and sometimes was used to pick up the environmental noise when in noisy rooms. The microphone built into the detector case is a commercially available “cement wall microphone” (TWA-1-W Fujita Electric Mfg. Inc.) which was originally designed for picking up sound leaking through a wall. The probe rod, made of plastic and 3mm in diameter, is covered by a thin plastic sheet which efficiently derives the sounds from the skin while protecting the microphone from mechanical shocks. For recording sounds from a tooth, the cover sheet was removed and the microphone probe rob was directly attached to the tooth because the sonic impedance of the rod is matched for hard materials such as cement, bone, and teeth.

Prior to bruit recording, the patients lay on the bed and the blood pressure was measured at the arm. A pair of electrodes were placed on the right shoulder and on
the left belly for the ECG recording. The ECG signal was sent via an ECG telemeter (1422, San-ei Co.), and the Q-R rising edges were detected by a pulse indicator (1965A/2211B, San-ei Co). The timing of the Q-R edge, that is, the ventricular contraction, was indicated optically to allow discrimination of the bruit from other noise during the examination. When a stable ECG indication was attained, a pair of bruit detectors were applied to the patient's head like headphones. For perfect detector contact, a jelly for ultrasonic examination was used. The detected sounds were amplified by FET-top low noise amplifiers, and recorded in a data-recorder (SONY A-109) together with the timing pulses indicating the Q-R rising edges. The sounds were also brought into bandpass filters (from 400 Hz to 2K Hz) and monitored via stereo headphones. The recording time was 1 minute per position; during that period patients were asked to be silent, but breathing was allowed.

The hardware configuration of the data analysing system is depicted in figure 1(b). The recorded data were carefully aurally examined with the aid of a graphic equalizer (YAMAHA Q1027), then the playback speed was reduced to 1/500 via tandem use of data recorders (SONY A-109 and FR-3215), and the chart recordings were obtained on a thermal pen-recorder (WX4404, Watanabe-Instruments Co.). In the spectrum analysis, noises such as breathing, foot steps, door shutting, etc. greatly affected the results. Some noises could not be eliminated through the filtering in the frequency domain, so a dual channel analog gate was used at the input of the processor which opens 200ms after the Q-R edge of the ECG, then closes 400ms after the Q-R edge. The gate can be also controlled by manual key tapping and automatic detection of excessive environmental noise. The gated signals were then put through a pair of low pass filters (cut-off 2.5kHz) to reduce hiss noise and to prevent the aliasing effect in the A/D conversion process. The signals were then converted to digital data at the sampling frequency of 5KHz, and spectrum analysis and cross-correlation analysis were carried out by 7T08 program packages #39 and #100A, where averaging was taken over 32 or 64 beats. The resultant data were displayed on a CRT screen and hard copies were plotted on a X-Y recorder.

Result

This system was used on 25 patients and 21 control subjects. The ages ranged from 11 months to 71 years. The diagnoses of the patients were mostly for intracranial aneurysm or AVM (aneurysm 9; AVM 13; carotid-cavernous fistula 1; brain tumor 2). For the control subjects, absence of intracranial circulation disorders was confirmed via angiography prior to the test. The bruit was detected for 15 out of 25 patients (aneurysm 5/9; AVM 8/13; CCF 1/1; brain tumor 1/2), whereas none of the control subjects produced sounds. In general for aneurysm cases, the lower frequency component of around 500 Hz was dominant and the apex of the spectrum was sharp; whereas for AVM cases, a wide spread of high frequency components 800 to 1500 Hz was significant. Typical examples of aneurysm and AVM cases follow:

Case 1

This 52 year old female patient had an aneurysm in the left middle cerebral artery. The patient complained of long persisting headache without any other neuro-
logical symptoms or signs. The CT scan revealed a round high density mass of 1.5 cm in diameter at the left Sylvian fissure. The angiography demonstrated the aneurysm at the left middle cerebral artery (fig. 2). No episode of subarachnoid hemorrhage has been noted.

The relationship between the waveforms of the bruit recorded from the anterior tooth and the Q-R edge of the ECG is shown in figure 3. According to this graph, the sound uttered by the affected part started about 160 msec after the Q-R edge. Breath noise was larger in amplitude when recorded at the tooth, compared to that recorded at the head surface. The breath noise was reduced by gating prior to the spectrum analysis. The spectrum of the bruits recorded at the anterior tooth and the temporal region are shown in figure 4 (a) and (b) respectively. The sharp spike at 470 Hz can be seen in both figures and is attributable to the bruit inherent to the aneurysm. The broad band component around 0–2.0 KHz in (a) corresponds to the breath noise.

Case 2

The 69 year old male patient had arteriovenous malformation. The patient had complained of head ache and neck pain for eight months. His visual acuity was slightly diminished. Bilateral papilledema was detected, but no other neurological signs and symptoms were noted. Although CT scan showed no abnormal findings, the angiography demonstrated the dural arteriovenous malformation fed from the left occipital artery (fig. 5). No bruit was audible by stethoscope at the time of admission.

With this system, clear bruit was recorded from the anterior tooth as shown in figure 6. We narrowed the aperture time of the signal intake to 75 msec by adjusting the gate controller, and shifted it along the bruit cycle as indicated by timing bars “a” through “e” in figure 6. The first observation period “a” opens at 150 msec and ends at 225 msec after the Q-R edge. The second observation “b” is for 225 to 300 msec after Q-R, and so on. The spectra of each observation are shown in figure 7 a–e, where the vertical axes are shifted to avoid overlapping of the curves.

Case 3

This 45 year old male patient had carotid-cavernous fistula. The patient complained of gradually increasing pain in his left orbital region. Diplopia appeared two months later but it had subsided day by day. Although slight conjunctival chemosis was noted in both eyes, no exophthalmos was noted. The faint bruit was auscultated only from his left preauricular region. The angiography revealed the carotid-cavernous fistula originated from both internal and external carotid arteries (fig. 8).

Clear bruit was recorded at the temporal region. The
chart recording and the power spectra are shown in figures 9 and 10 respectively. In this case, a rather high
toned and very random bruit was heard, but the domi­
nant frequencies seemed to change according to the
waxing and fading process of the bruit, synchronous to
the heart beat. In figure 10 it can be seen that the
significant frequencies (indicated by delta marks) are
shifting from the high frequency region (around 1.2
KHz) down to the low frequency region (around 800
Hz), as the fading process of the bruit progresses from
"a" to "e".

Discussion

Among the 25 patients, no bruit was recorded in 10
cases. Is utterance of the bruit dependent on blood
pressure? To answer this question, we examined the 20
cases for which the systolic pressure had been record­
ed, and halved the data into two groups; one of clear
bruit cases (mean 130.5 mmHg with standard devi­
ation 14.9 mmHg) and the other for none or faint bruit
cases (mean 124.1 mmHg, sd = 15.1 mmHg). The
clear bruit cases showed slightly higher pressures,
however the difference was not statistically significant
when we performed a t-test; probably because cranial
blood circulation is pertinently maintained unless the
systolic pressure is exceedingly lowered (less than 50
mmHg), as discussed by Ferguson 6 for Arfonad hypo­
tension cases.

There are alternative interpretations for the bruit
utterance mechanism of aneurysms: Ferguson 6 postu­
lated that turbulence may exist in human aneurysm and
that the bruit must be generated by it, whereas Olinger
and Wasserman 7 suggested the possibility of a Helm­
holtz generator as the energy conversion mechanism
(which explains the invariability of the bruit frequency
against the blood flow speed). In our cases, bruits of
AVM and CCF were significantly different from those
of aneurysms in their spectrum profile; which suggests
different sound utterance mechanisms as pointed out
by Simkins et al. 8 When the waxing-fading process of
the bruit was analyzed via time shifting spectra, as
shown in figures 7 and 10, it is clear that the significant
frequencies are time dependent.

Therefore, a number of possibilities might be con­
sidered. The apparently random noise with wide range
frequency components is a compound of artery wall
resonances, whose resonant frequencies vary from ar­
tery to artery as suggested by Foreman and Hutchison 10
with their experimental results on isolated arteries. If
Figure 8. The angiography of Case 3. The carotid-cavernous fistula originated from both internal and external carotid arteries is demonstrated.

Figure 9. Bruit recorded from the anterior tooth for Case 3.

Figure 10. Spectra of the bruit recording for Case 3.

So far neither of these possibilities can be rejected. In any event, the bruit is a result of arterial wall vibration which could be a hazardous mechanical shock leading to rupture. Perhaps those patients who do not generate a detectable bruit are safer than those with a detectable bruit. We need more of a case follow up to find the answer.

The interval between the Q-R edge and the starting point of the bruit varied from subject to subject, and seemed to depend on the position of the affected part and on the individual circulation conditions. The spectrum of sounds also differed from patient to patient which suggests that the spectrum pattern reflects the position and the degree of cerebrovascular disorder.

It is important to improve the transducer sensitivity and the S/N ratio of this system for successful sound detection. However there may be a threshold point in the bruit generation mechanism; in that case the detection rate would not be improved simply by the sensitivity increment of the system. In our present system, only audio-frequencies higher than 400 Hz were considered. Whereas, according to the experiments on animal arteries, the dominant frequencies of artery wall vibration exist at less than several decade Hz; at
Evaluation of the Risk of Immediate Anticoagulant Treatment in Patients with Embolic Stroke of Cardiac Origin

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SUMMARY We evaluated immediate anticoagulation of embolic stroke. Of 39 patients suffering a focal cerebral ischemia caused by a cardiac embolus, 38 were submitted to CT within 24 hours after onset. Twenty-one patients received direct full anticoagulation and, in 10 patients, treatment was delayed because of hemorrhagic infarction on initial CT (3 cases), cardiac cause 24 hours or more after stroke onset, or treatment delay without specific reason (6 cases). Eight patients with severe deficit were not anticoagulated because of hemorrhagic signs on initial CT, impaired consciousness, or general contra-indications to such treatment. Twenty-one follow-up CT-scans were performed under full anticoagulation, and in only 2 cases hemorrhagic infarction was noticed without clinical deterioration. No clinical worsening attributable to anticoagulant treatment was observed during the three week observation period. It is concluded that direct anticoagulation therapy does neither induce hemorrhagic infarction nor cerebral hemorrhage in patients with embolic stroke nor does it cause clinical deterioration.

Anticoagulation Treatment in Patients with Cerebral Ischemic Events Caused by Cardiac Emboli is No Longer a Matter of Debate. The problem is when to start. Because of a recurrence rate of 15 to 20% in the first few weeks after the initial event, direct anticoagulation by means of intravenous heparin followed by oral anticoagulants seems to be indicated. However, in pathological studies, a high incidence of hemorrhagic infarction was seen in cases with embolic stroke, and in experimental studies hemorrhagic infarction increased in animals under anticoagulant treatment and led to clinical worsening. Therefore, some clinicians feared aggravation of the neurological deficit by inducing hemorrhage into ischemic brain areas in patients with embolic stroke, although...
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