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Artifact Simulating Subarachnoid and Intraventricular Hemorrhage on Single-Shot, Fast Spin-Echo Fluid-Attenuated Inversion Recovery Images Caused by Head Movement: A Trap for the Unwary

ORIGINAL RESEARCH

A. Cianfoni
M.G.M. Martin
J. Du
J.R. Hesselink
S.G. Imbesi
W.G. Bradley
G.M. Bydder

BACKGROUND AND PURPOSE: Single-shot, fast spin-echo, fluid attenuated inversion recovery (SS-FSE-FLAIR) images are frequently used to detect disease in the brain and subarachnoid space in confused or uncooperative patients who may move during the examination. In some of these patients, high signal intensity areas are seen on good-quality images in the subarachnoid space and ventricular system in locations not associated with high CSF flow. These artifacts may simulate hemorrhage or leptomeningeal disease. The purpose of this article was to determine the cause of these artifacts, describe ways to recognize them, and find methods to reduce or eliminate them.

METHODS: Healthy volunteers were studied on 6 occasions with conventional multisection FSE-FLAIR images and SS-FSE-FLAIR images while at rest and while nodding and rotating their heads at different speeds. In addition, SS-FSE-FLAIR images with different section widths of the initial inverting pulse and a non–section-selective initial inversion pulse were performed with the subjects moving their heads in the same way. The scans of 30 successive patients with acute neurologic syndromes who had been studied with SS-FSE-FLAIR sequences were reviewed for evidence of high signal intensity in the CSF in regions not associated with high CSF flow.

RESULTS: Each of the volunteers showed areas of increased signal intensity in CSF at sites apart from those associated with rapid pulsatile CSF flow on SS-FSE-FLAIR images acquired during head motion. The images were otherwise virtually free of motion artifact. The use of a wider initial inversion pulse section and a non–section-selected initial inversion pulse reduced the extent of these artifacts. Nineteen of the 30 patients showed areas of high signal intensity in the CSF in regions not associated with highly pulsatile CSF flow. Six of these patients had negative lumbar punctures for blood and xanthochromia and normal CSF protein levels.

CONCLUSION: High signal intensity artifacts may be seen in CSF as a result of head movement on otherwise artifact-free images when imaging uncooperative patients with SS-FSE-FLAIR sequences. These artifacts have a different mechanism and distribution from those caused by CSF pulsation and may simulate subarachnoid and intraventricular hemorrhage. Artifact recognition is aided by signs of patient motion during the examination. The artifacts can be reduced by use of increased section width and non–section-selective initial inversion pulses. Recognition of these artifacts is important, because the circumstances in which the SS-FSE-FLAIR sequence is used and the particular properties of the sequence may conspire to produce a trap for the unwary.

Fast spin-echo fluid-attenuated inversion recovery (FSE-FLAIR) is a multisection MR imaging pulse sequence that can improve lesion detection in the brain, subarachnoid space, and meninges.[1-9] Because of this sensitivity to disease, FSE-FLAIR sequences are frequently included in the MR imaging work-up of patients with acute neurologic syndromes. These acutely ill patients may be confused and uncooperative and may move during the MR examination. As a result, conventional multisection FSE-FLAIR images are frequently degraded by motion artifact. To deal with this problem, a faster variant of the FLAIR sequence, single-shot FSE-FLAIR (SS-FSE-FLAIR) can be used.[10] This sequence employs an FSE acquisition with half-Fourier mapping of *k*-space to acquire the image data for each section in 0.1 to 0.2 seconds rather than the several minutes typically required for a simultaneous multisection FSE-FLAIR acquisition of the 20 to 30 sections necessary for examination of the whole brain.

Both of these variants of the FSE-FLAIR sequence tend to show high signals in the CSF within the posterior fossa, basal cisterns, lateral ventricles near the foramen of Monro, third and fourth ventricles, and the aqueduct.[1,9-12] These signals are caused by rapid oscillatory flow of CSF from pulsatile motion of the brain producing inflow of noninverted spins into the section of interest in the time between the initial inversion pulse and the later 90° pulse, which is typically approximately 2.0 to 2.5 seconds. It is necessary to differentiate these artifactual high signals from subarachnoid or intraventricular hemorrhage as well as leptomeningeal disease.[4,13,14]

In this article, we report the occurrence of high-signal-intensity CSF artifacts with use of the SS-FSE-FLAIR sequence,

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From the Department of Radiology (A.C., M.G.M.M., J.D., J.R.H., S.G.I., W.G.B., G.M.B.), University of California, San Diego, Calif; and the Department of Radiology (M.G.M.M.), Clinics Hospital of the University of Sao Paulo, Sao Paulo, Brazil.

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Address for correspondence: Graeme M. Bydder, Department of Radiology, University of California San Diego, 200 West Arbor Drive, San Diego, CA 92103-8756.

also known as half-Fourier acquisition, single-shot turbo spin-echo (HASTE-FLAIR; Siemens, Erlangen, Germany) and SS-turbo-FLAIR (Philips, Eindhoven, the Netherlands) in regions not associated with rapid pulsatile CSF flow such as the Sylvian fissures, cortical sulci over the convexities of the hemispheres, and the posterolateral regions of the lateral ventricles in patients with acute neurologic syndromes. These artifacts simulate subarachnoid or intraventricular hemorrhage. The purpose of the study was to determine their cause, describe ways to recognize them, and find methods to reduce or eliminate them.

Materials and Methods

After institutional review board approval, all studies were performed on a 1.5T MR scanner (Signa LX; General Electric, Waukesha, Wis) by using a multichannel head coil, field of view (FOV) 22 cm, matrix size 224 × 224, and bandwidth of 41.67 kHz.

Four versions of the FSE-FLAIR sequence were used: (1) Multisection FLAIR with repetition time (TR) = 8800 ms, echo time $(TE)_{eff}$ = 124 ms, and inversion time (TI) = 2200 ms. Twenty-six sections were acquired in 3 minutes and 32 seconds. The section thickness of the inversion pulse was 5 mm and that of the 90°(acquisition pulse) was 4 mm. These were the manufacturer's default settings. (2) Standard SS-FSE-FLAIR. The TR was long (fully relaxed), TE_{eff} = 122 ms, and TI = 2200 ms. The width of the initial inversion pulse was 5 mm and that of the 90° pulse was 4 mm. Partial FOV (0.75), half number of excitations (NEX), and a parallel imaging acceleration factor of 2 were used to reduce the number of acquired phase encodings from 224 to 42, with a data sampling time of 113 ms per section. Twenty-six sections were acquired in 61 seconds. (3) Wider initial inversion pulse width SS-FSE-FLAIR. This was the same as in (2), except the section width of the initial inversion pulse was 10 mm in one case and 30 mm in the other. The 90° pulse and acquisition mode were the same as in (2). (4) Non–section-selective SS-FSE-FLAIR. This used a non–section-selective inversion pulse and had TR = 10 seconds, TE_{eff} = 122 ms, and TI = 2550 ms. The 90° pulse and acquisition mode were the same as in (2).

Healthy Volunteers

Two adult volunteers were asked to hold their heads still during the acquisition of multisection FSE-FLAIR and standard SS-FSE-FLAIR images. They were then asked first to rotate from left to right in a horizontal plane and then to nod their heads as the same sequences were repeated. The volunteers were trained to repeatedly move their heads at approximately constant speed in each direction a specified distance and to do this at either a slow or moderate speed during the scan. By using the nasion as a reference point the distance the subject moved was measured relative to the head coil, and by counting the number of repetitions per minute the average speeds of head motion for slow and moderate movement were calculated as approximately 2 and 4 cm/s. Two adult volunteers repeated the motion with SS-FSE-FLAIR sequences having initial inversion pulse section widths of 5, 10, and 30 mm. Finally, 2 adult volunteers repeated the experiment with the non–slice-selective initial inversion pulse.

Patients

The images of 30 successive adult patients who had standard SS-FSE-FLAIR sequences as part of an acute neurologic syndrome ("stroke") protocol were reviewed for evidence of high signal intensity in the CSF at inappropriate locations (ie, areas apart from those associated with highly pulsatile CSF motion). Of these 30 patients, 19 showed high signal intensity in the CSF in such locations. Of these 19 patients, 6 subsequently had a negative lumbar puncture for blood and xanthochromia as well as a normal level of CSF protein. None of the 30 patients was treated with supplemental oxygen or propofol before or during the MR examination. The patients did not receive intravenous injections of either a gadolinium chelate or an iodinated contrast agent before performing the standard SS-FSE-FLAIR sequence. The remainder of the stroke protocol included T1-weighted FSE (TR/TE_{eff} = 550/12 ms), T2-weighted gradient-echo (TR/TE = 525/25 ms), and T2-weighted FSE (TR/TE_{eff} = 2800/102 ms) sequences as well as diffusion-weighted, echo-planar imaging (EPI).

Results

Healthy Volunteers

In both subjects, the multisection FSE-FLAIR sequence acquired without head motion showed good visualization of brain structure (Fig 1*A*). The same sequence acquired during head motion was badly degraded by motion artifact (Fig 1*C*).

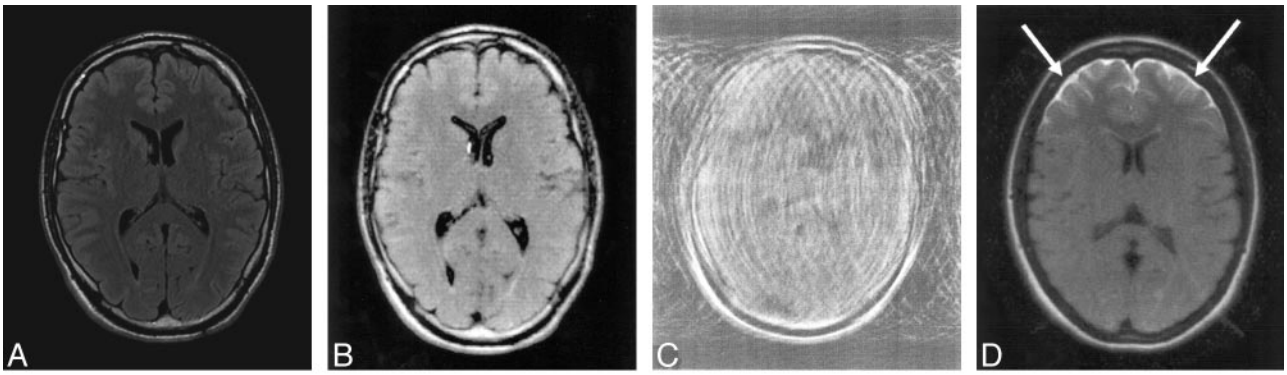
With the standard SS-FSE-FLAIR sequences acquired without head motion, pulsatile CSF artifacts were present in the usual locations. Images acquired during slow head motion showed these artifacts, but, in addition, high signals were seen in the CSF in the Sylvian fissures, subarachnoid space around the convexities and the lateral ventricles, though, as a whole, the images were not degraded by motion. Predominantly frontal artifacts are illustrated (Fig 1*D*). More widespread artifacts were seen with head movement at moderate speed (Fig 1*E*).

In both subjects during imaging in the transverse plane, with the standard SS-FSE-FLAIR sequence the pattern of artifacts was more marked in the through-plane direction (nodding for axial sections) than in the in-plane direction (rotating the head horizontally for axial sections) (Fig 2). The distribution of artifacts also differed with the direction of motion. At lower speed, images acquired during rotation of the head in-plane showed artifacts that were more diffusely located within the subarachnoid space and around the convexities with some greater prominence in the anterior frontal region. Only mild artifacts were seen in the lateral ventricles. In images acquired during nodding of the head (through-plane), artifacts showed a more specific location anteriorly and posteriorly with relative sparing of the frontoparietal region and central areas of the brain.

With the section-widened SS-FLAIR sequences, motion artifacts were progressively reduced as the width of the initial inverting pulse was increased from 5 to 10 and then 30 mm, though artifacts were still present at 30 mm, particularly with through-plane motion (Fig 3). The nonselective inversion pulse SS-FSE-FLAIR produced a marked reduction in artifact (Fig 4).

Patients

In 19 of the 30 patients with acute neurologic syndromes, standard SS-FSE-FLAIR images showed high signal intensity in CSF in regions not associated with pulsatile CSF artifacts such as within the Sylvian fissures and over the convexities of the cerebral hemispheres. Patient motion could be inferred from changes in the orientation of sections as well as by gaps in the



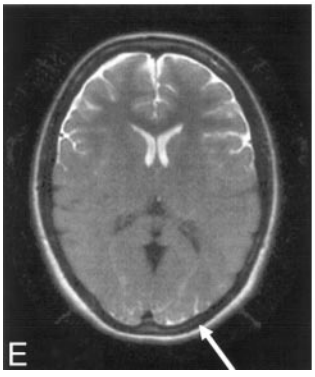


Fig 1. Healthy volunteer. Transverse multisection single-shot, fast spin-echo, fluid-attenuated inversion recovery (SS-FSE-FLAIR) (repetition time [TR]/echo time $[TE]_{eff}$/inversion time [TI] = 8800/124/2200 ms) (*A*) and standard SS-FSE-FLAIR (*B*) images at rest, corresponding images with slow nodding (*C* and *D*), and a standard SS-FSE-FLAIR image with moderate nodding (*E*). Pulsatile CSF flow artifacts are seen in the region of the foramen of Monro, but no high-signal-intensity artifacts are seen elsewhere in the CSF (*A* and *B*). In *C*, the multisection image is badly degraded by motion. The corresponding standard SS-FSE-FLAIR image (*D*) is not degraded by motion artifact, but high signal intensity is seen in the CSF around the frontal lobes (*arrows*). With moderate nodding additional high-signal-intensity artifacts are seen on the SS-FSE-FLAIR image in the frontal horns of the lateral ventricles and in sulci of the occipital lobes (*E, arrow*). There is still no overall motion degradation. Slightly higher gray-white matter contrast is seen in the frontal lobes in *C* and *E* compared with *B*.

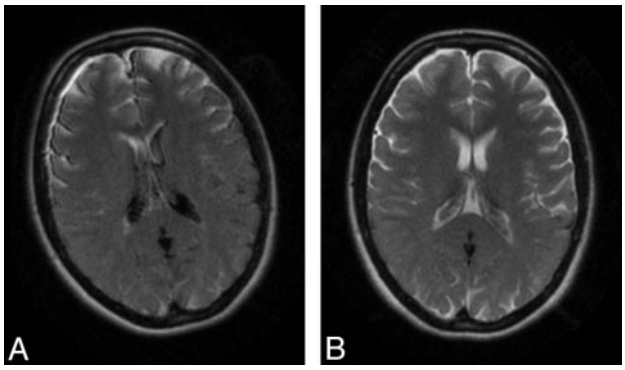


Fig 2. Healthy volunteer. Transverse standard single-shot, fast spin-echo, fluid-attenuated inversion recovery (SS-FSE-FLAIR) images with moderate rotation (*A*) and moderate nodding (*B*). The high signal intensity in the subarachnoid space and ventricular system is more extensive in panel *B*.

section pattern and overlap of sections on most sets of standard SS-FSE-FLAIR images. In each case, conventional T1- and/or T2-weighted images showed motion artifact. In patients who had not had lumbar punctures, it was not possible to be certain that subarachnoid blood or elevated CSF protein levels were not present and had caused the high signal intensity in the CSF. For that reason, attention was focused on the 6 patients with a negative lumbar puncture for blood and xanthochromia and without elevated CSF protein. Images from one of these patients are shown in Fig 5.

Discussion

SS-FSE-FLAIR images of the brain in confused patients often show artifactual high signals in the subarachnoid space and ventricular system in locations not associated with rapid pulsatile CSF flow. These may mimic hemorrhage as well as infectious, inflammatory, or neoplastic processes. The most likely cause of these artifacts is motion of the subject's head between the initial section-selective inversion pulse, and the subsequent 90° pulse and data acquisition of SS-FSE-FLAIR sequences. As a result of this motion, noninverted or incompletely inverted spins enter the section that is subsequently imaged. In this section, the magnetization in CSF is not nulled and high signal intensity is seen in the subarachnoid space and ventricular system. The belief that the artifacts described in this study are caused by head motion was based on the results in healthy volunteers that showed absence of artifact when the head was still, increase in artifact with increase in speed of motion, and a change in pattern of artifacts with change in type of motion from nodding to rotating. In addition, the artifacts could be reduced by using techniques designed to decrease the effects of head motion between the initial inversion pulse and the subsequent 90° pulse.

The common CSF flow artifact, often seen with multisection and standard SS-FSE-FLAIR sequences, is due to pulsatile CSF flow displacing inverted spins and replacing them with noninverted spins from outside the inverted section. This motion is due to cardiac pulsation of the brain and has been well characterized with velocity mapping techniques.[15] Regions of high signal intensity are seen at the foramen magnum, in the basal cisterns and third and fourth ventricles, as well as in or near, the foramen of Monro. These are at or near constrictions, or relative constrictions to flow. The head as a whole is in the same position between the initial (section-selective) inversion pulse and the subsequent 90° pulse. Only the pulsatile CSF significantly changes its location between the 2 pulses of the FLAIR sequence.

In the situation we are discussing here, the head as a whole moves between the initial section selective inversion pulse and the subsequent 90° pulse. If the movement is sufficient to shift the acquisition to areas of the head where the CSF has not

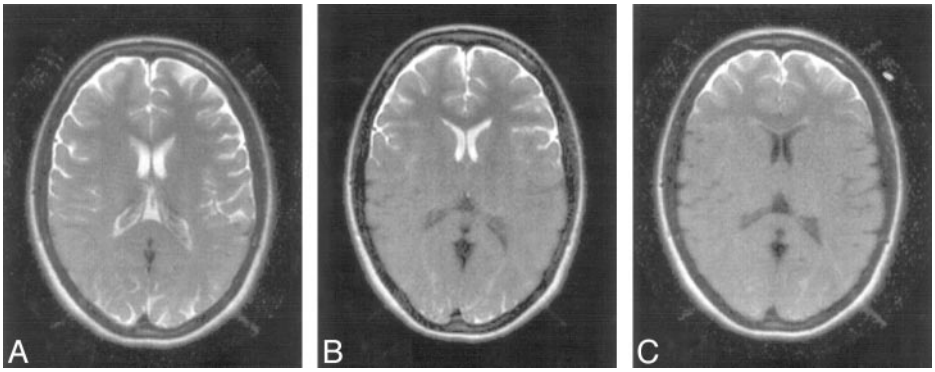


Fig 3. Healthy volunteer. Transverse standard single-shot, fast spin-echo, fluid-attenuated inversion recovery (SS-FSE-FLAIR) with 5-mm initial inversion pulse section width (*A*), a section widened (10 mm) SS-FSE-FLAIR (*B*), and another section-widened (30 mm) SS-FSE-FLAIR sequence with moderate nodding. With increasing section width there is a progressive reduction in both subarachnoid and intraventricular artifact.

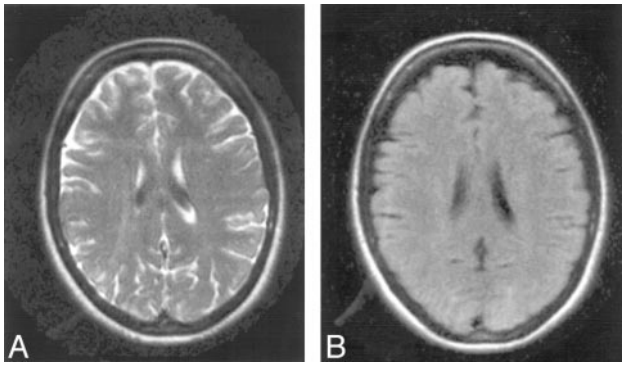


Fig 4. Healthy volunteer. Transverse standard single-shot, fast spin-echo, fluid-attenuated inversion recovery (SS-FSE-FLAIR) (*A*) and image obtained with a non–section-selective inversion pulse (*B*). Both were acquired with moderate nodding. There is much better control of motion artifact in *B*.

experienced the initial inversion pulse and is therefore not nulled, high signal intensity is likely to appear in the CSF (Fig 6). Displacement of CSF between the 2 pulses can be related to the section thickness of the initial inversion pulse and to that of the 90° pulse as well as a calculation of the speed of the head necessary to displace the region covered by the initial inversion pulse from that covered by the subsequent 90° pulse and acquisition, assuming an ideal section profile and no significant flow of CSF. The speeds of 2 cm/s and 4 cm/s used in this study with a 2.2-second interval between the initial inversion pulse and the subsequent 90° pulse, and a section width of the initial inversion pulse of 5 mm, are sufficient to do this, as was confirmed experimentally. Even with an initial section width of 30 mm, it is still possible for the region covered by the inversion pulse to move away from that covered by the 90° pulse and acquisition. More obvious effects were seen when the direction of head movement was through-plane (eg, nodding with an axial section). If the motion is in-plane, much of the CSF within the section may still be nulled even if the head has moved. With head movement a later section may be acquired over a region where the CSF has been recently inverted and has only partly recovered in the time between single-shot acquisitions. This may also result in an increase in the signal intensity from CSF in the subsequent excitation and acquisition.

In addition to the movement of the brain and CSF as a whole to a different location between the initial inversion pulse and the subsequent 90° pulse as described above, the situation may be complicated by the fact that the movement itself induces CSF flow. This is related to the inertia of the CSF. This effect is similar to the pattern seen in a bucket of water that is displaced and then brought to a standstill. The motion of the water may be delayed initially relative to that of the bucket and subsequently continue after the motion of the bucket has stopped. The pattern of flow is modulated by the container and any fixed or relatively fixed material that may be in the bucket. This type of motion has been observed in human volunteers with serial single-shot cine EPI studies of the inside of the cranial cavity[16] (V. Weeden, personal communication, 1991, 2005). The motion is complex with acceleration and deceleration phases and has a swirling or rotatory character. It may continue for a few seconds after motion of the head as a whole has stopped. The pattern in an enclosed volume such as a ventricular cavity is usually more constrained than that outside of the brain. It may be affected by differences in physical density between the lighter brain and heavier CSF.[17] It differs from the oscillatory motion of CSF because of pulsation of the brain of cardiac origin. Diseases such as atrophy and cerebral edema may affect the size and shape of CSF spaces and thus the flow pattern.

Patient motion in the acute situation is affected by the patient's anatomy and the physical constraints associated with the machine. It is likely to have both in-plane and through-plane components as well as a spectrum of different velocities and may be episodic. The inertial CSF flow induced by this motion is also likely to be complex and difficult to characterize and have section-to-section differences depending on patient motion during the preparation and acquisition phases of the sequence as well as motion relative to previous preparations and acquisitions. As a consequence the distribution and intensity of head motion induced artifacts are likely to be more variable and less predictable than those of pulsatile CSF flow artifacts. Common locations of artifacts were anterior to the frontal lobes, over the cerebral hemispheres, and in the ventricular system.

Unlike CSF, the brain signal intensity itself is only slightly affected by displacement between the initial inversion pulse and the later 90° pulse, because its magnetization largely recovers from inversion by the time the 90° pulse is applied.[1] It therefore makes only a relatively small difference to the brain signal intensity whether it was in a section which was subject to an inversion pulse, though this difference may be significant for artifact recognition (see below). The difference may be manifest as higher gray-white matter contrast (more complete longitudinal recovery of magnetization similar to that seen with a heavily T2-weighted FSE sequence) in brain that has not experienced an initial inversion pulse, compared with brain that has experienced such a pulse with a typical FLAIR sequence, when TIs in the shorter range are used (eg, Figs 1 and 5).

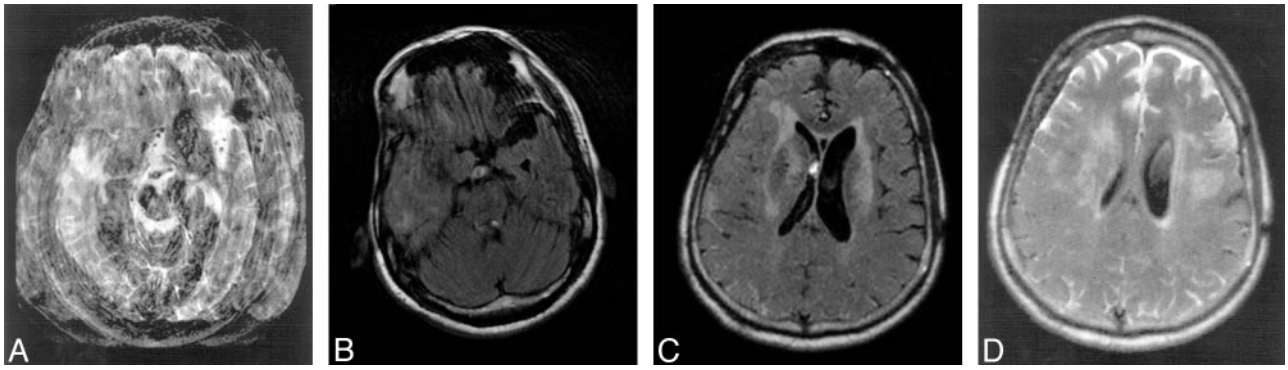


Fig 5. Images from a 53-year-old man with chronic hypertensive encephalopathy and acute mental status change. The patient was agitated and confused. Multisection T2-weighted FSE (repetition time [TR]/echo time $[TE]_{eff}$ = 2800/120 ms) image (*A*) and standard single-shot, fast spin-echo, fluid-attenuated inversion recovery (SS-FSE-FLAIR) images at different levels (*B–D*). The T2-weighted FSE image is severely degraded by motion artifact. Images in *B* to *D* show no obvious motion artifact, though the patient moved during the acquisition of the images as shown by the change in angulation of the head between sections. In *D,* obvious high-signal-intensity areas are seen anteriorly in the subarachnoid space. More subtle changes are seen posteriorly. These mimic subarachnoid hemorrhage. A slight increase in gray-white matter contrast is seen in the frontal region in *D,* consistent with the brain in this region not having experienced the initial inversion pulse. The brain shows extensive white matter change.

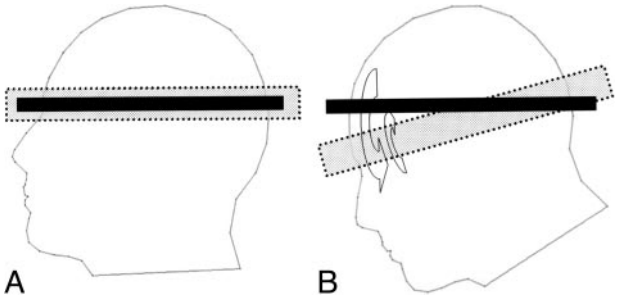


Fig 6. Sagittal drawing of the head showing the region covered by an initial section selective inversion pulse (*shaded area*) and that covered by the subsequent 90° pulse and acquisition without head movement (*A*) and with head movement downward in the superior-inferior direction between the initial inversion pulse and the 90° pulse (*B*). Possible directions of head motion–induced CSF flow at different times are also shown (*curved arrows*). With movement (*B*), the excitation and acquisition includes posterior areas of the subarachnoid space, which have experienced the inversion pulse as well as anterior areas that have not experienced it. These latter areas are likely to display high signal intensity, though the final result may be affected by CSF flow induced by the head motion. Initially, some CSF may move with the head while other portions of the CSF may move in the same direction later. When the head stops moving, there may be overshoot of the CSF moving with the head, followed by a later reversal of its direction of flow.

The head displacement and the CSF motion induced by it result in artifacts at sites not associated with rapid oscillatory CSF flow, and these may simulate subarachnoid and intraventricular hemorrhage as well as leptomeningeal disease. This problem is compounded by the fact that the SS-FSE-FLAIR images may not appear to be degraded by motion artifact as a whole because of the rapid data acquisition, and so there may be no typical motion artifacts present on the images to alert the radiologist to the fact that the patient may have moved during the SS-FSE-FLAIR sequence and that motion-induced CSF flow artifacts could be present. Excessive emphasis may be placed on the SS-FSE-FLAIR images because they may be the only T1- or T2-weighted images acquired during the examination that do not appear degraded by motion artifact. In addition, motion is more likely in the clinical situation in which SS-FSE-FLAIR sequences are preferred—namely, confused and uncooperative patients with acute neurologic syndromes—and it is just these patients whose differential diagnosis frequently includes subarachnoid hemorrhage and leptomeningeal disease. This combination of factors may result in a trap for the unwary.

Features on SS-FSE-FLAIR images that may alert a radiologist to the fact that the patient has moved during the examination (and that this motion may have caused high-signal-intensity artifacts in the CSF) include a change in the orientation of successive single-shot images, apparent jumps or overlaps between adjacent sections, and marked high CSF signal intensity at a single level with a normal appearance at another adjacent level. A relative increase in gray-white matter contrast in the brain adjacent to high signal intensity in the CSF may be a sign of the presence of artifact and provide a direct indication that the brain and the adjacent CSF have not experienced the initial inversion pulse. Motion degradation seen on other pulse sequences acquired at the same examination may mean that the patient is likely to have moved during the SS-FSE-FLAIR sequence. Unusual locations and intensities of apparent subarachnoid or intraventricular hemorrhage and their occurrence in an inappropriate clinical context may also help in recognition of this type of artifact.

There are causes of high signal intensity in the CSF with FLAIR sequences that may result in high signal intensity either alone or in concert with head motion. These include increased protein as a result of infective, neoplastic, and other pathologic processes. These cause T1 shortening of CSF.[18-20] High signal intensity in the CSF can also be seen in patients breathing increased inspired oxygen.[21-24] This is due to dissolved paramagnetic molecular oxygen shortening the T1 of CSF. There are also other less well defined causes of increased signal intensity in CSF such as leakage of contrast agents into the subarachnoid space[25] and propofol administration.[26]

Confused patients often tend to move toward their feet and thus outside of the head coil, so regions of the brain move away from the area of highest radio-frequency field uniformity, which is located centrally within the transmitter coil, toward the entrance of the coil and beyond it. As a result, the CSF around some central and inferior regions of the brain may not be fully inverted by a radio-frequency pulse that fully inverts the magnetization at the center of the coil. This can cause spurious high signal intensity in the CSF. The effect may be synergistic with those caused by head motion.

Since the introduction of the FLAIR sequence, various techniques have been used to reduce high signal intensity ar-

tifacts produced by CSF pulsation. These have included the use of a non–section-selective initial inversion pulse, which nulls all the CSF within the effective imaging volume of the head coil and so reduces the effects of inflow of CSF between the inversion and acquisition pulses.[27-29] Within limits, all CSF is equally inverted and flow in and/or out of the section has no effect on the net magnetization excited by the subsequent 90° pulse. Even the CSF in the section that is subsequently excited has a similar signal intensity to the non–section-selected CSF, because its longitudinal magnetization is zero or near zero after the 90° pulse and subsequent section-selective inversion pulses used in the data acquisition. The net effect on the in-section longitudinal magnetization may be only slightly different from that of the surrounding CSF which also has zero or close to zero longitudinal magnetization at this time. In multisection acquisitions with a nonselective inversion pulse, only a limited number of sections can be acquired with the CSF nulled or nearly nulled.

It has usually been preferable to increase the section width of the initial inversion pulse relative to the acquisition pulse to reduce the effect of inflowing CSF.[1-3] Although this is generally helpful, too great an increase in section width leads to inefficient interleaving of sections. Another method of reducing CSF pulsation artifacts is to use a non–section-selective initial inversion pulse and reorder *k*-space for each section so that the central lines of *k*-space are acquired with a TI at or near the null point for all sections in a multisection acquisition.[30] 3D acquisitions may also be performed with a non–section-selective or slab-selective initial inversion pulse[31,32] and so show improved control of CSF pulsation artifact. *k*-space reordering requires the same time as multisection techniques and is likely to be unsuitable for confused patients. 3D FSE-FLAIR is also likely to be too slow for this group of patients. Multishot FSE and EPI techniques have the disadvantage that patient motion between acquisitions may introduce artifacts.

In this study, it was shown that increase in the section width of the inversion pulse reduced head motion induced CSF artifacts. It may be possible to effectively increase the section width with an acceptable impact on total imaging time for a whole-brain acquisition. If the initial inversion pulse is too wide, there is a risk that with patient motion a recently inverted region of CSF may be included in the section and produce high-signal-intensity artifacts. The use of a non–section-selective inversion pulse with a single-section acquisition can effectively suppress the CSF flow artifact, though this may not be completely successful near the apex of the skull and in the region of the foramen of magnum where the CSF may not be fully inverted by conventional inversion pulses. More complete inversion can be achieved by use of adiabatic fast-passage inversion pulses.[33] It is possible to acquire multiple sections at or around the null point to make the use of a non–section-selective inversion pulse variant of the FLAIR sequence time efficient.[27,28]

The reason that this artifact has not been recognized as a specific problem with multisection FSE-FLAIR pulse sequences is probably because scans from patients who have moved significantly display obvious motion degradation (eg, Fig 1) and spurious high signals in the CSF that are readily recognized as part of this pattern. Although degradation of image quality on HASTE-FLAIR images due to artifacts from inflow of blood from outside of the section has been described previously in one study,[9] to the best of our knowledge, high signal intensity artifacts in CSF due to head motion and associated CSF motion have not been described. EPI-FLAIR is mostly used in multishot form where patient movement between acquisitions is likely to lead to obvious image degradation and alert the radiologist to the possibility that head motion has induced high-signal-intensity CSF artifacts.

There are ongoing issues regarding the diagnostic value of both conventional multisection FSE-FLAIR sequences and CT in subarachnoid hemorrhage in general, as well as at different stages in the evolution of this condition.[34-42] It is possible that the performance of the SS-FSE-FLAIR sequence in cases of subarachnoid hemorrhage may be improved by use of the widened and non–section-selective inversion pulse variants of the SS-FSE-FLAIR pulse sequence we have described. These techniques may also be applicable to SS-EPI-FLAIR sequences.

Conclusions

SS-FSE-FLAIR is used in confused patients who are likely to move and where the disadvantages compared with multisection FSE-FLAIR (such as lower resolution and loss of edge definition) are outweighed by the need to obtain interpretable images without significant motion artifact. Patient motion during the examination can induce high signal intensity artifacts in the subarachnoid space and ventricular system and these may simulate hemorrhage. The artifacts are due to head motion and have a different cause and distribution than those due to CSF pulsation. The high signals in CSF may not be recognized as motion artifacts because, as a whole, the single-shot images are not degraded by motion. The artifacts can be reduced or eliminated by widening the initial inversion pulse and by using a non–section-selective inversion pulse. This may become more important with the more general use of the single-shot techniques made possible by the improved signal-intensity-to-noise ratio achievable at higher fields and the greater availability of parallel imaging. Recognizing and understanding these artifacts is important, because the context in which SS-FSE-FLAIR sequences are used and the presence of high signal intensity in the absence of other signs of patient motion may readily lead to the misdiagnosis of hemorrhage and other intracranial disease.

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