Permissible Arterial Occlusion Time in Aneurysm Surgery: Postoperative Hyperperfusion Caused by Temporary Clipping

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Abstract

The relationship between hyperperfusion and temporary clipping was evaluated to determine the safe limit for the duration of temporary clipping in aneurysm surgery. Twenty-one patients surgically treated for a ruptured aneurysm were examined using xenon-enhanced computed tomography on postoperative days 4 to 13. Eight of the 16 patients undergoing temporary clipping had focal hyperperfusion; whereas the five patients without temporary clipping had no hyperperfusion. Mean total temporary clipping time in patients with hyperperfusion was significantly longer than that in patients without (31.9 vs. 13.9 minutes, p = 0.0157) and mean maximum single temporary clipping time in patients with hyperperfusion was also significantly longer than in patients without (18.4 vs. 8.6 minutes, p = 0.0313). Moreover, cerebral infarction was related to hyperperfusion (p = 0.0027). These results support the hypothesis that temporary clipping during aneurysm surgery causes postoperative hyperperfusion and cerebral infarction. Temporary clipping may be harmful when performed for more than 20 minutes of total duration, since postoperative hyperperfusion was seen under this condition.

Key words: hyperperfusion, subarachnoid hemorrhage, temporary clipping, xenon computed tomography

Introduction

Abnormally high cerebral blood flow (CBF) caused by hyperperfusion,1,3,8,13) luxury perfusion,2,5,7,12) or hyperemia10,14) can occur in global or focal regions,14) and has been identified in patients with stroke,1,3,7,8,12) head injury,10,14) and epilepsy.13) Xenon-enhanced computed tomography (Xe-CT) showed that some patients suffer focal hyperperfusion in the postoperative subacute stage, following aneurysm surgery involving temporary clips.17,18) Hyperperfusion is often caused by vasomotor paralysis after ischemia, and the mechanism behind the hyperperfusion in such patients appears to be similar to that caused by postischemic reperfusion after prolonged temporary clipping. Temporary clipping of a parent artery is useful for preventing premature rupture during aneurysm surgery, but carries the risk of cerebral infarction. Studies of temporary arterial occlusion in the management of intracranial aneurysm indicates that the safe limit ranges from 14 to 20 minutes.6,11,15) This study investigated the relationship between temporary clipping and postoperative focal hyperperfusion to determine the safe limit for temporary clipping.

Subjects and Methods

I. Patient selection

Thirty-seven consecutive patients with cerebral aneurysms underwent surgery between December 13, 1994 and February 10, 1999. Temporary clipping was applied in 24 cases including four cases with premature rupture. Twenty-one patients were selected, in whom the aneurysms were clipped on days 0 to 2 after subarachnoid hemorrhage (SAH), and the first CBF examination was performed on postoperative days 4 to 13 after surgery (Table 1). Patients with Fisher group 4 on initial CT were not
Six aneurysms were located on the anterior communicating artery (AcomA), eight on the middle cerebral artery (MCA), and seven on the internal carotid artery (ICA). Sixteen patients, nine males and seven females aged 19 to 74 (mean 51.8) years, underwent temporary clipping at the parent artery of the aneurysm. The other five patients, two males and three females aged 41 to 70 (mean 58) years, did not undergo temporary clipping. On admission, 18 patients were classed as Hunt and Kosnik (HK) grades 1 or 2, and 14 patients had Fisher group 3 on initial CT. Cerebral angiography was conducted before and 3 weeks after surgery. General anesthesia was induced using intravenous thiopental and maintained with nitrous oxide and isoflurane. Before and during temporary clipping, agents such as mannitol, phenytoin, methylprednisolone, and vitamin E (Sendai Cocktail) were administered intravenously with induced systemic hypotension using prostaglandin E, or nicardipine at a systolic blood pressure of 90 to 100 mmHg. Nizofenone, sodium ozagrel, and fasudil hydrochloride were routinely injected as prophylaxis against cerebral vasospasm for 2 weeks after surgery. Induced hypertension, hypervolemia, and hemodilution therapy were attempted as required, and continuous cisternal drainage for 1 to 2 weeks was undertaken for all patients.

### Table 1 Clinical characteristics of 21 patients with ruptured cerebral aneurysm

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<th>Case No.</th>
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<th>Fisher group</th>
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<th>mCBF</th>
<th>HP-rCBF</th>
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II. Xe-CT CBF measurement

CBF studies were conducted using a Xe-CT CBF system (AZ-7000, AZ-725; Anzai, Tokyo) adapted to an Xpeed CT scanner (Toshiba, Tokyo). After the first Xe-CT study, follow-up CBF studies were performed in the patients with hyperperfusion. Patients inhaled 30% stable xenon gas in oxygen for 3 minutes, followed by a clearance period of 5 minutes, and CBF values were calculated. CBF was measured 44 times in the 21 patients. Hyperperfusion on Xe-CT was assessed visually compared to the regional CBF (rCBF) of the contralateral area in the unaffected hemisphere. Hyperperfusion was
III. Temporary clipping and neurological symptoms

Total duration, maximum single duration, and repetitions of temporary clipping applied to the parent artery were reviewed. The duration and repetitions of temporary clipping applied to dominant A1 were used in patients with AcomA aneurysm. Possible neurological symptoms induced by hyperperfusion were also analyzed: Symptomatic vasospasm based on the motor dysfunction, speech and mental disturbance in the subacute stage, outcome according to the Glasgow Outcome Scale (GOS), and occurrence of cerebral infarction in the vascular area applied by the clipped arteries.

IV. Statistical methods

The Mann-Whitney U test was applied to compare patients with and without hyperperfusion, and the chi-square test was used for individual factors related to hyperperfusion. Differences were judged significant at p < 0.05.

Results

I. Clinical outcome

A focal hyperperfusion area corresponding to the area supplied from the parent artery of the aneurysm was detected in eight of 16 patients on postoperative days 4 to 13 who underwent temporary clipping, and in none of the five patients without temporary clipping. Hyperperfusion was observed in three of eight patients with MCA aneurysms, four of six with AcomA aneurysms, and one of seven with ICA aneurysms. Hyperperfusion rCBF values ranged from 75 to 116 (mean 93) ml/100 g/min, whereas mean CBF values of the unaffected hemisphere were 29 to 48 (mean 37) ml/100 g/min. The interhemispherical ratio ranged from 1.3 to 2.31 (mean 1.7).

Cerebral infarction was seen in seven of eight patients with hyperperfusion and in two of eight without hyperperfusion of the 16 patients who underwent temporary clipping and in one of five without temporary clipping. Symptomatic vasospasm was seen in nine patients including six with hyperperfusion (Table 1). The GOS outcome for the eight patients with temporary clipping and hyperperfusion was good recovery (GR) in four, moderate disability (MD) in three, and severe disability (SD) in one. The eight patients with temporary clipping but without hyperperfusion had GR in six and MD in two. The patients without temporary clipping had GR in four and SD in one.

Hyperperfusion was significantly related to cerebral infarction ($\chi^2; p = 0.0027$) and to the occurrence of symptomatic vasospasm ($\chi^2; p = 0.0261$), but the GOS was not significantly related (Table 2).

II. Temporary clipping and hyperperfusion

The duration of single temporary clipping ranged from 4 to 34 minutes, total duration of temporary clipping from 4 to 60 minutes, and temporary clipping repetitions from 1 to 5. Total duration and maximum single duration of temporary clipping were significant factors in the occurrence of hyperperfusion. The total duration of temporary clipping in patients with hyperperfusion was 31.9 ± 14 (mean ± SD) minutes, compared to 13.9 ± 10.3 minutes in patients without (p = 0.0157). The maximum single duration of temporary clipping in patients with hyperperfusion was 18.4 ± 10.4 minutes, compared to 8.6 ± 3.4 minutes in patients without (p = 0.0313). Temporary clipping repetitions was not significant, 2.9 ± 1.2 in patients with hyperperfusion and 1.8 ± 0.9 in those without (p = 0.0742).

The relationship between hyperperfusion and temporary clipping total duration and repetitions is shown in Fig. 1. The occurrence of hyperperfusion increased markedly in patients with total duration of temporary clipping exceeding 20 minutes.

Postoperative serial Xe-CT study of the eight patients with hyperperfusion showed high rCBF

| Table 2 Statistical analysis of clinical features and hyperperfusion |
|-----------------------------|---------------|-----------------|-----------|-----------|-----------|
| No. of cases | HK grade (average) | Cerebral infarction | SVS | Severe disability on GOS |
|----------------|-----------------|-----------------|-------|-----------|-----------|
| TC (+), HP (+) | 8 | 2.1 | 7* | 6** | 1 |
| TC (+), HP (-) | 8 | 1.9 | 2 | 2 | 0 |
| TC (-), HP (-) | 5 | 2.4 | 1 | 1 | 1 |
| Total | 21 | 2.1 | 10 | 9 | 2 |

Significance was determined according to the chi-square test: *p = 0.0027, **p = 0.0261. GOS: Glasgow Outcome Scale, HK: Hunt and Kosnik, HP: hyperperfusion, SVS: symptomatic vasospasm, TC: temporary clipping.
values in the subacute stage after surgery (Fig. 2), and the rCBF values of all patients gradually decreased to normal or low values during postoperative days 15 to 305.

III. Representative case

A 44-year-old male (Case 1) with HK grade 2 and a ruptured left MCA aneurysm required temporary arterial occlusion twice during operation for a total of 21 minutes. Xe-CT CBF study showed hyperperfusion in the left MCA area, with the rCBF value decreasing from 104 ml/100 g/min on day 5 to 18 ml/100 g/min on day 52, and the interhemispherical ratio from 1.56 to 0.38 (Fig. 3). The outcome was GOS MD due to mild motor weakness and motor aphasia.

Fig. 1 Relationship between hyperperfusion and the total duration and repetitions of temporary clipping. AcomA: anterior communicating artery, IC-ACHO: internal carotid artery-anterior choroidal artery, IC-PC: internal carotid artery-posterior communicating artery, IC-top: top of internal carotid artery bifurcation, MCA: middle cerebral artery. ●: hyperperfusion (+), ○: hyperperfusion (−).

Discussion

The CBF value defining hyperperfusion or hypereemia has not been determined, but 21% and 72% increases of CBF measured by single photon emission CT (SPECT) have been described as hypereemia during the subacute phase of stroke. The present study indicated an interhemispherical ratio of 1.7, or a 70% increase of CBF.

Two types of hyperperfusion are observed in patients with SAH: Global luxury perfusion is common among patients with severe SAH and is caused by dysautoregulation in vessels of the whole brain or reduced intracranial pressure after surgery; and focal hyperperfusion is observed in a patient with mild SAH and epileptic seizure, probably caused by the activation of the sensitive trigeminovascular system by SAH.

The mechanism of hyperperfusion in our patients differs, except a case of postischemic hyperperfusion and 5 and 6 days after aneurysm surgery with temporary clipping measured by SPECT. Eight of our 16 patients undergoing temporary clipping had focal hyperperfusion at the postoperative subacute stage, and did not suffer severe SAH and external decompression, or epileptic seizure. Three important findings indicate the relationship between temporary clipping and hyperperfusion: Temporary clipping...
was performed in all patients with hyperperfusion; the hyperperfusion area was consistent with the ischemic area caused by temporary clipping; the significant relationship between hyperperfusion and the total and maximum duration of temporary clipping. Consequently, we considered that localized increases of CBF in our series were postischemic hyperperfusion \(^7,8\) secondary to temporary occlusion of a parent artery.

Our study used Xe-CT to verify the limit for temporary clipping in aneurysm surgery. The mechanism of hyperperfusion after temporary clipping may be similar to that of postischemic reperfusion in cerebral embolism, \(^9\) which causes metabolic acidosis and vasoparalysis and results in hyperperfusion. Temporary clipping is apparently a factor in the occurrence of ischemia. The repetition of temporary clipping was not the significant factor related with hyperperfusion in our study, as repeated short episodes of focal ischemia are relatively less damaging than a single equivalent long episode of focal ischemia in a cat model. \(^9\) Hyperperfusion was less common in patients with ICA aneurysm than in those with MCA and AcomA aneurysm, which may indicate that collateral flow prevents territory ischemia during temporary clipping.

Global \(^10\) and focal \(^4\) hyperemia has been observed in patients with head injuries. \(^14\) Hyperemia is theoretically not the same as hyperperfusion, but the term has been used to describe abnormally high CBF in many studies. \(^1,5,9,10,14\) Global hyperemia is malignant and associated with increased intracranial pressure and poor outcome, whereas focal hyperemia is benign and associated with minimal effect on intracranial pressure and level of consciousness, and with better outcome. \(^14\)

Just as the prognosis for patients with head trauma depends on the extent of hyperemia, the extent of hyperperfusion in patients with SAH seems to affect outcome, although vasospasm and severity of initial SAH may also be involved. Almost all of our patients with focal hyperperfusion, except one with HK grade 3 (Case 6), attained better outcomes. Hyperperfusion or hyperemia is transient, and observed in the acute and subacute stages on day 2 after mild SAH, \(^3\) days 0 to 13 after postischemic reperfusion, \(^8\) days 9 to 15 after stroke, \(^7\) day 1 after arteriovenous malformation surgery, \(^9\) days 3 to 6 after episodes of coma, \(^2\) days 1 to 8 after severe head injury, \(^10\) within 3 days after severe SAH, \(^12\) and within 2 weeks after head trauma. \(^14\) In our study, hyperperfusion was observed in the period of vasospasm after SAH on days 4 to 13 and was significantly related with symptomatic vasospasm. However, vasospasm is generally considered to cause decreased rCBF in patients with SAH in the subacute stage. Study of CBF and response to acetazolamide after aneurysmal SAH \(^17\) during the subacute stage showed CBF values fell considerably below control values in patients with symptomatic vasospasm, and the response to acetazolamide fell transiently even in patients without symptomatic vasospasm. Accordingly, if vasospasm causes hyperperfusion, this high rCBF area should be, but is not, observed after the peak of vasospasm because the hyperperfusion occurs in postischemic vessels. Therefore, whether vessels in the area of focal hyperperfusion after temporary clipping show vasospasm in the subacute stage remains unclear.

The combination therapy of fasudil hydrochloride and ozagrel sodium is superior to only ozagrel sodium for treating patients at risk of vasospasm after aneurysmal SAH. \(^9\) However, these agents are not known to cause CBF increase of 70% or more, so the focal effects on CBF cannot be explained by administration of these drugs.

Temporary vessel occlusion, which facilitates aneurysm clipping and reduces the risk of premature rupture, is a valuable technique in cerebral aneurysm surgery. However, there is a permissible time limit to avoid stroke during aneurysm surgery. On the other hand, there are many factors which may be related to the occurrence of brain ischemia in surgery, such as compression by a spatula, external decompression, collateral blood supply, SAH grade, timing of surgery after SAH, aneurysm location, patient age, operative procedure, and influence of anesthesia and other drugs. Therefore, the duration of temporary clipping may not be the only factor in the occurrence of hyperperfusion and cerebral infarction. In our study, all patients were treated in the acute stage by the same surgeon and the same approach, without external decompression, and the hyperperfusion area did not coincide with the area compressed by the spatula. Furthermore, there were significant correlations between hyperperfusion and the occurrence of cerebral infarction and between hyperperfusion and temporary clipping. Therefore, temporary clipping must be important in the occurrence of hyperperfusion.

Thus, temporary clipping should not exceed the tolerable duration to avoid hyperperfusion and cerebral infarction.

The present and previous studies \(^5,11,13\) indicate that the safe limits for temporary clipping range from 14 to 20 minutes, because the occurrence of hyperperfusion markedly increased in patients with total temporary clipping duration exceeding 20 minutes. Mannitol prolongs the tolerance time for temporary occlusion of cerebral arteries, \(^16\) so tem-
porary clipping without mannitol injection will make the occurrence of hyperperfusion more likely. We emphasize the importance of safe temporary clipping in aneurysm surgery if postoperative hyperperfusion is to be avoided.

References


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Commentary

The authors have utilized xenon-enhanced computed tomography to study the relationship between hyperperfusion and temporary clipping during intracranial aneurysm surgery. Although the study is small, it strongly suggests a relationship between temporary clipping and hyperperfusion. Such a relationship is suggested in this study by the facts that temporary clipping was performed on all patients with hyperperfusion, the hyperperfused region is similar to the ischemic area caused by temporary clipping and the significant relationship between hyperperfusion and the total duration of temporary clipping. Based on
these observations, the authors have proposed that increases in cerebral blood flow are due to post-ischemic hyperperfusion secondary to temporary occlusion. I believe this case is well supported by the evidence.

Although the authors utilized their data to suggest that repetition of temporary clipping does not increase the risk of hyperperfusion and subsequent ischemia, the small size of this study precludes any definitive statement regarding the issue of repeated short versus single long episodes of temporary clipping.

The findings from this study are in keeping with other clinical and experimental studies that suggest the safe limit for temporary occlusion ranges from 14 to 20 minutes. Clearly, a number of factors influence the tolerance of a particular vessel to temporary occlusion. These factors include the collateral circulation to the temporarily occluded vessel, the presence or absence of perforating end arteries, the use of proximal occlusion as opposed to temporary trapping, pharmacologic agents utilized to prolong ischemic thresholds, and the circumstances under which temporary occlusion is utilized (premature rupture versus planned temporary occlusion to decrease this risk).

It is interesting to note that the authors routinely used systemic hypotension prior to and during temporary clipping. In my experience, this is highly unusual and will theoretically increase the risk of infarction associated with temporary clipping. More commonly, induced hypertension is utilized during temporary clipping to augment and optimize collateral circulation.

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Generally temporary clipping is used in the overall aneurysmal surgery so that the safe use of this technique is required for better outcome. The risk of infarction related to this technique has been reduced with the introduction of brain protective methods: mannitol, hypertension, hyperthermia, and barbiturate. However, further study for identification of the technique and patient specific factors is still required to use it safely. Some studies reported previously approached these factors, focusing on the duration of vessel occlusion. In these studies, the clinical stroke and/or radiological infarction were parameters of assessing the effects of temporary vessel occlusion.

In this study, the authors applied hyperperfusion, using Xe-enhanced CT scans, as this parameter and analyzed temporary vessel occlusion time to avoid the risk of stroke. I think this is a more sensitive and objective way to approach these points with the aspect of hyperperfusion. However, twenty minutes of temporary occlusion time provided here seems to be somewhat weak evidence because other various factors were not taken into the analysis. The other point is that the proportion of patients with infarction was too high, compared to others, with the relatively small numbers of patients enrolled in the study. Nevertheless, this study provides a very helpful information and guideline for research associated with temporary clipping in aneurysmal surgery.

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This study was conducted to retrospectively examine the effect of temporary clipping in aneurysm surgery on postoperative hyperperfusion, and to determine the safe time limit for temporary clipping. The results indicated that temporal vessel occlusion causes postoperative hyperperfusion and cerebral infarction. The authors concluded that temporary clipping longer than 20 minutes may be harmful. Although temporary clipping is apparently a factor in the occurrence of ischemia, there are many other factors, including compression by a spatula, external decompression, collateral blood supply, SAH grade, timing of surgery, aneurysm location, patient's age, operative procedure, and influence of anesthesia and other drugs, as the authors mentioned. The brain is damaged after SAH due to acute and severe elevation of intracranial pressure. In such a situation, temporary arterial occlusion may be very stressful to the damaged brain, in relation to the various degrees of collateral blood circulation. Nobody can exclude the effects of these factors. Therefore, I would like to recommend that the authors to perform the same study in patients with unruptured cerebral aneurysms. As the authors suggested, we should keep in mind that temporary artery clipping should not exceed 20 minutes during aneurysm surgery. It would be better to use repeated short-time temporary clipping rather than single long-time temporary clipping.

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