SCALED SUCTION FOR MICRONEUROSURGERY: TECHNICAL NOTE

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OBJECTIVE: We have developed scaled suction to facilitate the measurement of aneurysm neck width and tumor size during operations.

METHODS: We constructed a new suction device scaled every 1 mm from the tip to 3 cm and every 5 mm from 3 to 5 cm. The scaled suction devices have been used in 50 aneurysm and brain tumor operations.

RESULTS: The new suction device permits easy measurement of aneurysm neck width, tumor size, the extent of internal decompression of tumor, and depth from the surface of the brain to the lesion.

CONCLUSION: Our scaled suction device is a simple and useful navigator for continuously measuring intraoperative variables such as lesion size and distance between the lesion and the surrounding vital structures.

KEY WORDS: Brain tumor, Cerebral aneurysm, Microsurgery, Scale, Suction

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Accurate determination of the size of a lesion and the distance between the lesion and surrounding structures is essential for safe and steady intracranial surgical procedures (1, 2). As the operative field is opened step by step to accomplish each operative exposure, these measurements should be easily repeatable during microsurgical procedures. Conventionally, these variables have been measured by placing a scale into the operative field. This method provides a rough estimate of lesion size but is difficult to use continuously in a hemorrhagic and/or deep operative field. To overcome these problems, we have designed scaled suction devices, which can be used as a continuous intraoperative monitor to estimate the size of lesions and the distances between the lesion and surrounding structures.

MATERIALS AND METHODS

A scaled suction device was constructed using pure titanium (Japanese Industrial Standard Grade 2, titanium ≥99.85%) (*Fig. 1B*). Each scale was marked entirely on the surface every 1 mm, from the tip to 3 cm, and every 5 mm from 3 to 5 cm (*Fig. 1A*). Five different sizes of scaled suction device of 1, 1.5, 2, 2.5, and 3 mm in internal diameter were made. The scaled suction instruments have already been used in 30 aneurysm and 20 brain tumor operations.

RESULTS

Figure 1, C and *D*, illustrates the application of the scaled suction device in an operative field. The patient was a 55-year-old woman who was found to have a right middle cerebral artery aneurysm during a brain examination. The aneurysm had a relatively wide neck (*Fig. 1C*), estimated to be approximately 6 mm by our scaled suction device. We selected a straight clip, 11 mm long, as the most suitable for clipping the aneurysm. During the aneurysm surgery, we were able to measure not only the aneurysm size but also the depth of the operative field and the distance between the lesion and the surrounding vital structures, such as the perforators. These intraoperative measurements facilitated selection of clips of suitable sizes and enhanced the accuracy of clip blade placement. During tumor surgery, the tumor size, depth of extension of the tumor, and depth from the surface of the brain to the lesion were easily estimated by our scaled suction instrument. The intraoperative measurements with our scaled suction device were performed without incident in 50 microneurosurgeries for cerebral aneurysms and brain tumors.

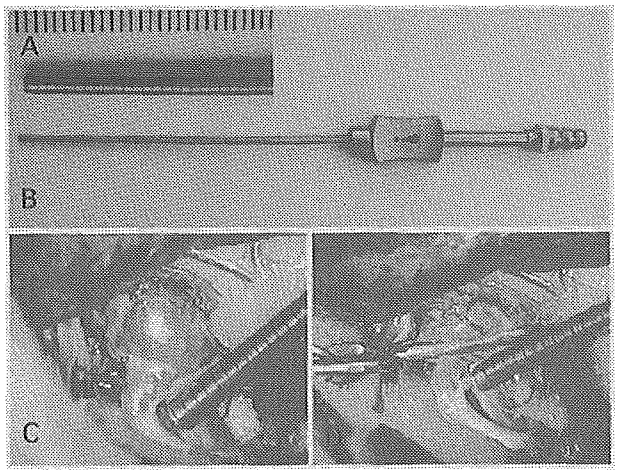

FIGURE 1. A *and* B, *pure titanium scaled suction (2-mm internal diameter). Each scale is sharply marked on the surface every 1 mm, from the tip to 3 cm.* C *and* D, *intraoperative photographs showing the scaled suction being used to assess the aneurysm size and neck width.*

DISCUSSION

Our newly developed scaled suction devices have permitted easy, rapid, and consistent measurements of the sizes of lesions as well as the distances between lesions and the surrounding vital structures during microsurgical manipulations. Various types of scaled instruments have been designed and are helpful for establishing orientation in neurosurgery (1, 2). Mizutani (2) developed scaled clips for aneurysm surgery, applied these clips in 40 aneurysms, and confirmed their usefulness. Suctions, however, are the most frequently used instruments in microneurosurgery. Constant intraoperative placement of a scaled instrument can facilitate intraoperative quantitative analyses of the size of the lesion and the distance between the lesion and corresponding structures, such as an aneurysm and its parent artery. From this point of view, scaled suction instruments serve as one of the most important navigation devices and as a constant detector of the width and depth of a lesion and the operative field. In conclusion, our newly developed scaled suction instrument is simple, safe, and useful for neurosurgical procedures.

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COMMENTS

The authors have described a scaled suction instrument. This is a logically and practically sounded add-on design. It allows a surgeon to measure the neck of an aneurysm directly to facilitate selection of a proper length of clip and is also useful for surgery for a deeply located tumor.

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Okada et al. have presented a set of tubes having marks every millimeter from the tip for 3 cm and every 5 mm thereafter from 3 to 5 cm. The suctions are available in a variety of sizes. It is common to use a suction tube for both suction and dissection around the neck of an aneurysm or in defining the margins of a tumor. It seems appropriate to place a scale on the suction as an aid to selecting the appropriate aneurysm clip or to measure how much of a tumor has been removed. The findings from this scale can also be correlated with results from image guidance. Other instruments typically used around an aneurysm or tumor include dissecting bayonets and various dissectors like the Rhoton Numbers 6, 7, and 8 (1). It seems reasonable to consider placing a scale on other instruments commonly used in dealing with tumors and aneurysms.

The authors list the inner diameters of their suction tubes in millimeters related. A more common practice in the United States is to describe suction tube diameter in terms of the French units. Each French unit is a third of a millimeter outer diameter, so a 3-French tube would have a 1 millimeter outer diameter and a 6-French would have a 2 millimeter outer diameter.

The most delicate neurosurgery is done with a suction tube held in a pencil grip with the hand rested on the margin of the wound. Some suction tubes, such as the Frazier tube, are designed to be held in a pistol grip with the hand floating in the air above the wound. We prefer the type of suction with the hand held in a pencil grip with the hand rested on the wound margin, because this allows for a more accurate and delicate dissection. Resting the hand on the margin of the wound and reaching the target sites requires that the length of the suction be sized to the depth of the target area.

Our tubes range in diameter from 3- to 12-French and are available in three lengths: superficial length for work at the surface, a deep length for work in areas such as the CP angle and the circle of Willis, and an extra deep set for use at sites such as in the front of the brainstem and in transsphenoidal surgery (1). We have also added atraumatic tips to the suction tubes.

Okada et al. have demonstrated an innovation, which could be applied to other instruments in addition to suction tubes. Most instrument makers will be willing to add marks like those presented in this article to a surgeon's favorite dissecting instruments.

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Okada et al. introduced a scaled suction for measuring during microsurgery. It is certainly handy and useful because suction is used almost continuously together with bipolar forceps during microsurgical procedures. This scaled suction is especially useful in measuring in a vertical dimension. We can measure in a horizontal dimension to some extent by tilting the suction or guessing from the scale on the suction under the microscope. The reason for using titanium is not explained well, but one would think that malleable materials would better serve the purpose of intraoperative measurement.

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CAROTID TISSUE LEVELS OF ARGATROBAN AFTER DIRECT LOCAL DELIVERY DURING CAROTID ENDARTERECTOMY TO PREVENT PERIOPERATIVE CEREBRAL EMBOLISM

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OBJECTIVE: Argatroban is a synthetic direct thrombin inhibitor. We applied argatroban locally during carotid endarterectomy to prevent local mural thrombus formation. Although local delivery of argatroban is expected to be effective for inhibition of mural clot formation, there is no report of the evaluation of its clinical effectiveness or local drug concentration in humans.

METHODS: Five mg of argatroban (0.5 mg/ml) was applied twice intraoperatively just after arteriotomy for measurement of intraplaque level of argatroban and during closure of the arteriotomy for preventing thrombus formation. After exposure of the carotid plaque to argatroban for a specified duration (0, 3, 5, or 10 min), argatroban was sufficiently washed with saline and the carotid plaque was removed for measurement of tissue concentration of argatroban. Intraplaque level of argatroban was determined by high-performance liquid chromatography. A second application was performed during closure of the arteriotomy. Argatroban was applied for 10 minutes, followed by washing with saline. Postoperative embolic cerebrovascular complications and carotid restenosis also were investigated to verify the efficacy of direct local application of argatroban.

RESULTS: Tissue levels of argatroban in the carotid plaque after 3, 5, and 10 minutes of direct application were 24.0 ± 13.7, 31.6 ± 20.0, and 44.0 ± 15.1 μg/g, respectively. The concentrations at all time points were significantly elevated compared with the control, and a significant difference in concentration was observed between 3 minutes and 10 minutes. In the present study, concentration at 3 minutes was much higher than the effective tissue levels of argatroban reported in experimental studies. No patient developed postoperative cerebrovascular complications.

CONCLUSION: The results suggest that direct local application of argatroban during carotid endarterectomy for at least 3 minutes may deliver high local tissue levels. Argatroban may be effective for prevention of perioperative embolic cerebral complications during carotid endarterectomy.

KEY WORDS: Argatroban, Carotid endarterectomy, Embolic, High-performance liquid chromatography, Restenosis, Thrombin, Tissue levels

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Thrombin is the central enzyme in hemostasis, possessing critical actions in coagulation, fibrinolysis, platelet activation, and vascular cell biology. Argatroban {(2R,4R)-4-methyl-1-[N(2)-(3-methyl-1,2,3,4-tetrahydro-8-quinolinesulfonyl-L-arginyl]-2-piperidine-carboxylic acid monohydrate} is a small molecular weight synthetic arginomimetic direct thrombin inhibitor derived from L-arginine, which reversibly inhibits the active site of thrombin (10, 17). Argatroban is active against clot-bound thrombin and is an effective inhibitor of thrombin-induced platelet activation and clotting (9, 10, 17). Argatroban has been approved for clinical use for both prophylaxis and treatment of thrombosis in patients with heparin-induced thrombocytopenia and treatment of various thrombotic disorders, including chronic arterial occlusion, acute cerebral thrombosis, and hemodialysis in antithrombin-deficient patients or in patients with decreased antithrombin (21).

Thrombin has a stimulatory role in angiogenesis and restenosis after angioplasty and atherosclerosis. Although intracoronary stenting represents an important advancement in

percutaneous revascularization technology, limitations such as platelet-mediated clot formation lead to acute and subacute stent thrombosis. The mechanical manipulations in percutaneous coronary intervention, including angioplasty and coronary stent placement, result in additional plaque rupture and damage to the vessel wall, exposing subendothelial components to blood and resulting in the initiation of the clotting cascade and platelet activation (11). Platelet-thrombus deposition primarily mediated by thrombin occurs within minutes after injury, causing acute occlusion and contributing to late restenosis. Therefore, antithrombin agents have been tried to reduce platelet-thrombus formation after arterial injury and stent implantation (3, 5, 11). For this purpose, local delivery of thrombin inhibitors has been reported to have significant effects in reducing platelet deposition and mural thrombus formation after balloon angioplasty or stent implantation, with no effect on systemic coagulability compared with systemic administration of the drugs (11, 13, 20). In previous studies, investigators used catheter-based technology (13, 20) or newly developed stent technology (11) for local delivery.

In carotid endarterectomy (CEA), most of the early cerebrovascular complications have been reported to be embolic in origin (12, 15). Furthermore, signs of emboli during dissection, wound closure, and early postoperative periods have been associated with cerebrovascular complications in CEA (1, 4, 19). We have used argatroban locally during CEA to prevent local mural thrombus formation. After we remove the carotid plaque, we locally irrigate the lumen of the vessels with argatroban when we close the arteriotomy. Although local delivery of argatroban is expected to be effective in inhibiting mural clot formation, there is no report evaluating its clinical effectiveness or local drug concentration in humans (8). We investigated the intraplaque levels of argatroban after intraoperative local delivery during CEA and the rates of early cerebrovascular complications and restenosis to verify whether the present protocol is effective in inhibiting postoperative embolic events and chronic intimal thickening.

PATIENTS AND METHODS

CEA

General anesthesia was used for CEA in all patients. The internal carotid artery was exposed well beyond the carotid plaque. Before carotid cross clamping, heparin was administered intravenously. Protamine reversal was not used. A longitudinal arteriotomy was made over the carotid bifurcation, extending to a level beyond the carotid plaque. A T-shaped silicone shunt system was used routinely in all patients (*Fig. 1A*) (18).

Patient Population and Protocol

Between May 2000 and July 2002, 45 consecutive patients (43 men and 2 women) ranging in age from 42 to 76 years (mean, 66.1 yr), who underwent CEA for cervical carotid stenosis, were studied. Five mg of argatroban (0.5 mg/ml) was

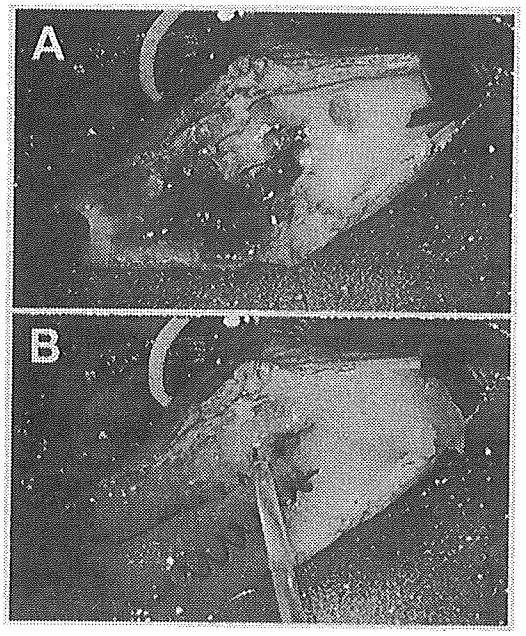

FIGURE 1. *Intraoperative photograph showing placement of the shunt tube (A) and local delivery of argatroban (B).*

applied twice intraoperatively: just after arteriotomy for measurement of intraplaque level of argatroban and during closure of the arteriotomy for preventing thrombus formation (*Fig. 1B*). In the first application, after exposure of the carotid plaque to argatroban for a specified duration (0 [control], 3, 5, or 10 min), argatroban was washed off sufficiently with saline and the carotid plaque was removed for measurement of tissue concentration of argatroban. A second application was performed during closure of the arteriotomy. Argatroban was applied for 10 minutes, followed by washing with saline. The excised carotid plaque was stored immediately at −80°C until measurement. The number of patients in each group was 12, 11, 9, and 13 in control, 3-minute exposure, 5-minute exposure, and 10-minute exposure, respectively. Informed consent for the study was obtained from all patients. The study protocol was approved by the institutional review committee.

Tissue Level Measurement

The cryopreserved carotid plaque segments without soft atheromatous changes were thawed at room temperature and cut cubes smaller than 2 mm. The cut tissues were weighed and homogenized ninefold by weight of water on an ice-water bath. The homogenized suspension samples were stored frozen at −20°C until analysis.

One hundred microliters of the homogenized sample, 100 μl of methanol, and internal standard (nitrazepam) solution were transferred to a test tube. Three hundred microliters of methanol was added to the mixture, shaken for a few seconds, and centrifuged at 10000 rpm for 5 minutes. One hundred microliters of supernatant was separated and mixed with 400 μl of water. The whole volume was filtered with 0.22-μm pore size

membrane filter. Next, 25 μl of the solution was subjected to high-performance liquid chromatography system at a temperature of 50°C.

The concentrations of argatroban were quantified by internal standard method. The range of the calibration curve was approximately 0.5 to 100 μg/g. The lower limit of detection of argatroban as measured by the high-performance liquid chromatography system was 0.5 μg/g.

Rates of Postoperative Cerebrovascular Complications and Carotid Restenosis

Postoperative embolic cerebrovascular complications and carotid restenosis were investigated to verify the efficacy of direct local application of argatroban. A cerebrovascular complication was defined as the occurrence of new signs or protracted aggravation of a preexisting neurological deficit. Postoperative carotid restenosis was defined as more than 50% stenosis on three-dimensional computed tomography angiography and B-mode ultrasonography. We assessed restenosis every 3 months during the first 6 months after CEA and every 6 months thereafter.

Data Analysis

Values presented in this study are expressed as mean ± standard deviation. One-way analysis of variance followed by post hoc Scheffé's test was used to determine the statistical significance of the differences among application durations. A P value of < 0.05 was considered statistically significant.

RESULTS

Tissue Level of Argatroban

Tissue levels of argatroban in the carotid plaque at 3, 5, and 10 minutes of direct application were 24.0 ± 13.7, 31.6 ± 20.0, and 44.0 ± 15.1 μg/g, respectively. Argatroban was undetectable (<0.5 μg/g) in all control tissues without argatroban application. The intraplaque concentration of argatroban increased proportional to the duration of application. The concentrations at all time points were significantly elevated compared with control ($P = 0.0022$, 0.0001, and <0.0001 at 3, 5, and 10 min, respectively). There was a significant difference in argatroban level between 3 minutes and 10 minutes ($P = 0.0112$). However, there was no significant difference in argatroban level between 3 minutes and 5 minutes. Furthermore, although a trend of increase was observed at 10 minutes of application compared with 5 minutes, the difference was not significant (*Fig. 2*).

Clinical Outcome

No patient developed postoperative cerebrovascular complications. However, postoperative carotid restenosis occurred in three patients (6.7%).

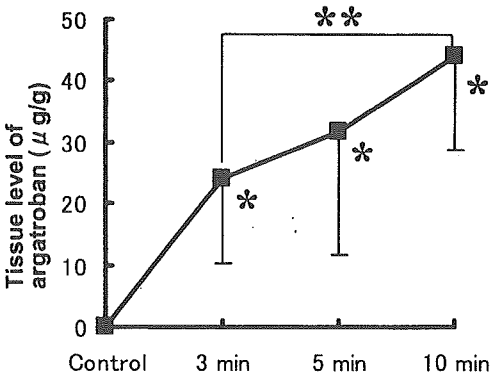

FIGURE 2. *Graph showing the tissue levels of argatroban (μg/g) by direct drug application for various durations during CEA. Data are mean ± standard deviation and were analyzed by one-way analysis of variance and then post hoc Scheffé's test. *, significant differences between control and each time point; **, significant difference between 3 minutes and 10 minutes.*

DISCUSSION

This is the first report of local tissue levels of argatroban after local application in humans. In the present study, the intraplaque concentrations of argatroban after direct application during CEA were elevated in a time dependent manner (*Fig. 2*). The levels at 3, 5, and 10 minutes of application were all significantly higher than the level in the control. Furthermore, the intraplaque concentration at 10 minutes was statistically elevated compared with that at 3 minutes. Even the concentration at 3 minutes was much higher than the effective tissue levels reported in a previous experimental study (8). These results suggest that direct application of argatroban delivers a high concentration of argatroban locally into vessel wall. The presence of drug within the plaque does not necessarily suggest that similar results will be observed in the residual artery wall after arteriotomy. However, we think that it is meaningful to assess tissue levels and clinical efficacy of argatroban simultaneously in the same patients. In humans, we cannot resect normal vascular wall after CEA to measure the tissue levels. Plaque tissue is the nearest resectable tissue that can be used to speculate regarding tissue level in the residual artery wall.

No patient developed postoperative cerebrovascular complications in the present series. Embolic ischemic complications have been reported to occur in approximately 2.6 to 6% of the patients after CEA (1, 12, 14, 19). Although the present investigation was not a controlled study, the results suggest that argatroban may be effective to prevent perioperative embolic cerebral complications. However, carotid restenosis occurred in three patients (6.7%). The reported incidence of carotid restenosis after CEA varies from 1.8 to 36%; this depends on the definition of restenosis (7, 16). A recent systematic review has demonstrated a 10% incidence of recurrent stenosis (≥50%) in the first year after CEA (6). Although locally delivered argatroban inhibited intimal thickening 20

days after balloon injury in animal models, as mentioned below (8), there was no long-term follow-up. In a comparison of the present results of recurrent carotid stenosis with those reported in the literature, single delivery of argatroban during surgery may not prevent restenosis in the chronic stage.

Two previous articles reported tissue argatroban levels after local delivery in experimental models (2, 8). In a newly developed catheter in porcine model, Anabuki et al. (2) demonstrated that the concentration of argatroban in the arterial wall at the coronary angioplasty site was significantly higher after local delivery. Furthermore, they demonstrated that high local concentrations (76.56 ± 30.74 μg/g) of argatroban did not damage vessel wall histologically (2). Imanishi et al. (8) evaluated the inhibitory effect of locally delivered argatroban on intimal proliferation after balloon injury of the carotid arteries in normal rabbits. In their study, the concentration of argatroban in the vessel wall was elevated sufficiently when a higher concentration (1 or 0.1 mg/ml) of argatroban solution was used (8). Furthermore, argatroban inhibited platelet aggregation, fibrin deposition, and intimal thickening 20 days after balloon injury (8).

In a previous investigation, concentration of argatroban after local administration via a hydrogel-coated balloon catheter immersed three times in an argatroban/saline solution was 14.8 ± 10.9 nmol/g (mean ± standard deviation, 7.79 ± 5.74 μg/g) (8). Argatroban at this tissue level significantly inhibited platelet deposition and chronic intimal thickening after balloon injury. Furthermore, much lower tissue concentration, i.e., 5.5 ± 4.6 nmol/g (mean ± standard deviation, 2.89 ± 2.42 μg/g) obtained from use of 0.1 mg/ml argatroban solution also achieved significantly smaller intimal-medial area ratios in the chronic stage compared with control (8). These tissue argatroban concentrations were much lower than the levels in the present study. Even the concentration after 3 minutes of application (24.0 ± 13.7 μg/g) in the present study was much higher than the reported effective levels. Extrapolating the above data, local application of argatroban for at least 3 minutes in accordance with the present protocol should be effective in preventing local mural thrombus formation, although there might be discrepancies in the effective tissue concentrations for inhibiting clot formation between humans and animals.

In animal models, local delivery of thrombin inhibitors has been reported to have significant effects in reducing platelet deposition and mural thrombus formation after balloon angioplasty or stent implantation, without influencing systemic coagulability compared with systemic administration of the drugs (11, 13, 20). To obtain the same local concentration as local delivery, much higher doses are needed for systemic administration. During CEA, we must prevent local mural thrombus formation after removal of the carotid plaque. Local application of argatroban just before closing the arteriotomy, in addition to the use of intraoperative systemic administration of heparin, should be an effective strategy.

Direct local application of argatroban during CEA for at least 3 minutes may deliver high local tissue levels. Argatroban may be effective in the prevention of perioperative embolic cerebral complications in CEA.

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COMMENTS

Kawamata et al. have performed a unique study in which they tested the safety of intraoperative, intraluminal, topically applied argatroban in the setting of carotid endarterectomy. High local tissue levels of this low-molecular-weight synthetic direct thrombin inhibitor were obtained with as little as 3 minutes of exposure in all 45 patients. Although the authors are careful to avoid claiming that use of this drug reduced the incidence of either thromboembolic events or restenosis, their data do suggest that at least in their hands, neither adverse event seemed to be increased. The next step is clearly a larger Phase I/II study. Such a study would do well to include other safety outcomes, such as the incidence of neck hematoma formation. The authors would probably do well to examine the effect of this drug on other surrogate markers of thromboembolism, such as intraoperative and early postoperative high-intensity transient analysis with transcranial Doppler ultrasound. It would also be interesting to see whether the incidence of neuropsychological dysfunction would be reduced by adding this agent as well.

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The authors report a study in which the synthetic thrombin inhibitor argatroban was applied during carotid endarterectomy. The drug was applied twice during surgery, initially before excision of the atherosclerotic plaque and again after endarterectomy and before closure. Varying drug exposure times were used, and plaque tissue levels of the drug were measured. A time-dependent relationship was found, with longer exposure times associated with higher tissue concentrations. The tissue concentrations found in this study are comparable to those found to inhibit platelet activation and thrombosis in experimental models. Local application of argatroban during carotid endarterectomy has the potential to be a relatively low-risk method to further reduce the risk of thromboembolic complications. Unfortunately, it is difficult to conclude that local tissue concentrations of the drug will translate into a reduction in thromboembolic risk. Furthermore, because the overall incidence of thromboembolic complications with carotid endarterectomy is relatively low, a randomized trial to assess this method would require a prohibitively large number of patients. Therefore, we are uncertain about the usefulness of this approach.

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Kawamata et al. have examined the topical application of argatroban, a direct thrombin inhibitor, immediately after arteriotomy and during closure of the arteriotomy in 45 patients undergoing carotid endarterectomy. They examined carotid plaque tissue levels of argatroban after the initial application and examined perioperative embolic complications and restenosis as surrogates of clinical efficacy.

Carotid plaque tissue levels of argatroban demonstrated an exposure-dependent increase after initial application. Indeed, an exposure time of 3 minutes (the shortest duration assessed) resulted in tissue levels of argatroban that far exceeded the effective tissue levels reported in experimental studies. There was no instance of cerebrovascular perioperative complications, and the restenosis rate was 6.7% (three patients).

This is an interesting and potentially useful application of this agent. The authors have convincingly demonstrated the ability to increase tissue levels in carotid plaques after topical application. However, the tissue levels in the residual vessel wall remain unknown. It is not unreasonable to assume that, particularly after the second application, tissue levels in the vessel wall will be increased.

The clinical effectiveness of this agent remains to be determined. The authors' preliminary perioperative complication rate is commendable; however, without a control group, these results cannot be ascribed to the use of argatroban. This will require further study. The authors do not report any complications of argatroban use, but do not specifically address whether local application of argatroban will influence systemic coagulation status. This is a difficult issue to address, because it is confounded by the use of systemic heparin, which is not reversed at the end of the procedure (thus prohibiting the use of early partial thromboplastin time as one coagulation parameter to assess). The chronic effects on restenosis of local application of argatroban after carotid endarterectomy also cannot be determined from this study. The duration of action after this type of application is unknown, and there is no control group to assess for a potential influence on restenosis rates.

The above notwithstanding, the authors have provided a meaningful contribution that documents preliminary experience with a potentially useful pharmacological adjunct during carotid endarterectomy that merits further study.

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In this article, the authors studied 45 patients undergoing carotid endarterectomy who received 5 mg of argatroban twice during carotid endarterectomy, once after arteriotomy, and they then measured intraplaque levels of the drug after application and then repeated the dose once during closure of the arteriotomy. The authors report that the carotid plaque revealed significant levels of the drug, which increased with exposure to the drug. They noted no clinical postoperative cerebrovascular complications and noted a 6.7% postoperative carotid restenosis rate of greater than 50%.

The authors' study actually raises as many questions as it seems to answer. The presence of drug within the plaque does not necessarily suggest that similar results will be seen in the residual artery wall after arteriotomy. The primary end point of

lack of embolic complications is followed only by clinical presentation rather than any measurement of emboli or studies for that. The restenosis rates are not well defined as to what period of time or what uniform studies were used to determine stenosis. It is not clear in the human what dose of drug one is trying to achieve in the residual wall, and this study does not answer that question any further. The duration of action of the drug over the entire period of reendothelialization of the endarterectomy site is also not clear. From this study, it is very difficult to imply efficacy of the agent. One would think that animal studies may be more pertinent for developing ideas of tissue levels with various dosing regimens. In the present protocol, one may not be certain that the presence of the agent within the plaque is also evidence for the presence of drug within the residual, presumably normal, wall.

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脳卒中の外科 33: 335～341, 2005

特集　頸部頸動脈狭窄症の治療方針

頸部頸動脈狭窄病変に対する外科的治療指針
—現時点でのCEAの問題点と対策—

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Guidelines for Surgical Treatment of Cervical Carotid Stenotic Lesions

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Summary: We focused on complicated carotid lesions in 324 of our carotid endarterectomies (CEAs) to clarify controversies in carotid surgeries. Carotid lesions extented to the C_2 level in over 20% of lesions. Bilateral stenotic lesions were operated in 22 cases without problems. Nine of 15 contralateral occlusion cases were supported with STA-MCA anastomosis indicated by the CBF. In near-occlusion cases, distal sites of lesions were detected by IVUS. Restenosis was observed in 9 cases. Only 1 restenotic case was symptomatic and 4 restenotic cases were reoperated with patch graft. Hemashield patch grafts were used in 18 cases and no restenotic changes were observed. Intracranial aneurysm was seen in 12 cases and 7 cases were clipped before CEA. Hyperperfusion syndrome was seen in 6 cases. Two cases showed intracerebral hemorrhage resulting in postoperative neurological deficits. Symptomatic occlusive coronary lesions were seen in 62 cases and surgical or intravascular treatment or both were performed in 30 cases.

Guidelines for CEA have been established by randomized controlled trails, but some cases have very complicated clinical features such as multiple lesions. For these cases, safer and more effective strategies should be established by collaborative studies.

Key words:
- carotid occlusive lesion
- carotid endarterectomy
- multiple lesions

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はじめに

欧米における頸部頸動脈狭窄病変は，脳虚血発作，脳梗塞の主要な原因疾患として注目され，治療法として頸部狭窄病変を摘出する頸動脈血栓内膜剥離術(Carotid Endarterectomy: CEA)が多くの症例に施行されてきた．このCEAの有効性に関して国際的な共同研究が進められ，症候性と無症候性頸動脈狭窄病変の頸動脈写所見から内科的治療と外科的治療のRCT (randomized control trial)が行われ，外科治療選択のEBM (evidence based medicine)が

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確立されてきた[7)8)15)17)]．しかしこの国際共同研究のみですべての頸動脈病変に対処できるものではなくいまだ問題点が残されている．たとえば両側頸動脈病変，パッチグラフトの選択，脳動脈瘤を伴う頸動脈狭窄病変，閉塞性冠動脈病変を合併している症例などに対する指針があげられる．本邦においても脳虚血発作の原因として頸部頸動脈狭窄病変が注目されるようになり，CEA例も増加している．しかし本邦例では高位病変が多いことや頸部頸動脈が細いことなど欧米での症例と異なる点も知られている[21)]．これらの問題に関して324側の自験CEA症例からCEAの現状での問題点を検討した．

対象，方法

324側のCEA症例を対象とした．臨床像は，無症候性狭窄，transient ischemic attack (TIA)，reversible ischemic neurologic deficits (RIND)，minor completed stroke (MCS)で分類した．狭窄度は，脳血管写や3D-CTAからECST法で求めた．血流測定は，定量的なCold Xe-CT法，Xe^{133}-inhalation methodで施行した．

CEAは，以下のようにシャントシステムを用いて行った[19)]．症例は右頸部頸動脈に高度狭窄を認めた68歳男性である(**Fig. 1A**)．麻酔は，脳神経機能モニタリング(体性感覚誘発電位と運動誘発電位)を行うために筋弛緩剤を使用しないでpropofolとfentanylによる全身麻酔法を用いた．体位は，頭部を対側に20-30度回転し，胸鎖乳突筋の前縁を最も高くし，布テープで下顎先端部を挙上するように固定した．皮切は乳様突起から胸鎖乳突筋の前縁に沿って弧状に約7-8 cm行った．皮切に沿って広頸筋を切開し，carotid triangle内で頸動脈鞘に達し，狭窄病変のない位置で総頸動脈を確保した．この総頸動脈から末梢に向かって頸動脈の露出を進め，外頸動脈を確保し，次に内頸動脈を狭窄病変より末梢1-2 cmの位置で確保した．ヘパリン(3000-5000単位)を全身投与し，ACT(activated coagulation time)が200 sec以上としたのちに総頸，内頸，外頸動脈を遮断した．総頸動脈側から正常な内頸動脈部まで動脈を切開し，シャントチューブを装着した．総頸動脈側より内頸動脈側に向かって中膜と内膜の間で病変の剝離を進めた(**Fig. 1C**)．病変は，原則として一塊として摘出し，残ったdebrisを丁寧に除去した(**Fig. 1D**)．動脈切開部は，5-0プロリーン糸で連続縫合した．術後3D-CTアンギオグラフィーで狭窄病変の摘出を確認した(**Fig. 1B**)．

324側(298例)のCEAで以下のような点；(1) 病変の広がり，(2) 多発性病変(両側狭窄病変と対側閉塞病変)，(3) near-occlusion病変，(4) 脳動脈瘤を伴った病変，(5)

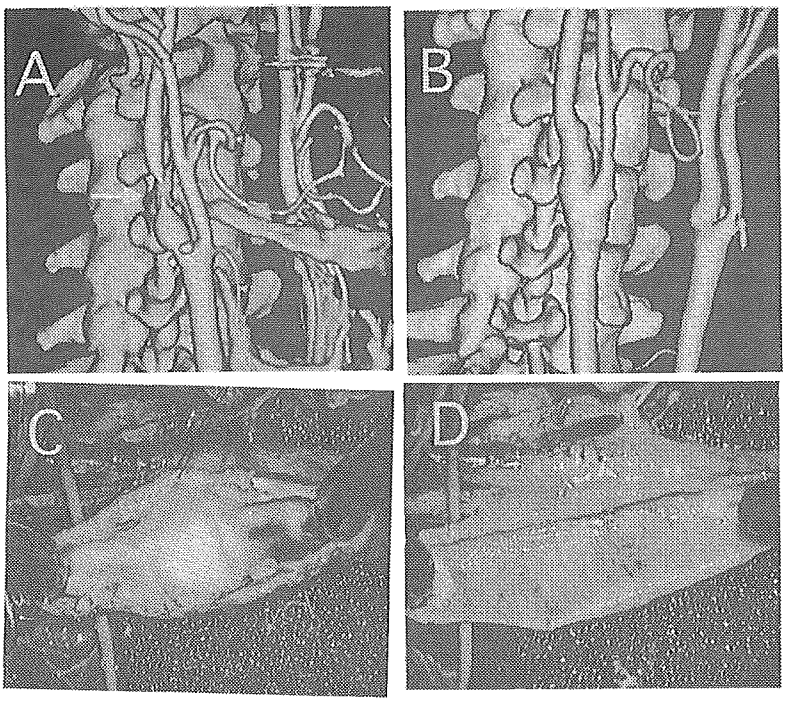


Fig. 1 Preoperative 3D-CT angiography demonstrates severe ICA stenosis (A). Postoperative 3D-CT angiography shows complete removal of the stenotic lesion (B). Intraoperative photographies show severe ICA stenosis with atheromatous plaque (C) and complete removal of the lesion (D).
3D-CT: 3 dimensional computed tomography, ICA: internal carotid artery

パッチグラフを要した病変，(6) 再狭窄病変，(7) 過還流症候群を呈した病変，(8) 心疾患について検討した．

結　果

298症例の性別は男性260例，女性38例で年齢は36-81(平均64)歳であった．324側頸動脈病変の臨床像は，無症候性狭窄102側，transient ischemic attack (TIA) 107側，reversible ischemic neurologic deficits (RIND) 42側，minor completed stroke (MCS) 73側であった．

病変の広がり：病変の広がりは脳血管写上での病変部の末梢側の位置で評価した．画像上末梢端の確認が困難であったnear-occlusion例を除いた317側の位置は，C2，C2/3，C3，C3/4，C4がそれぞれ51，37，168，26，35側であった．

多発性病変(両側狭窄病変と対側閉塞病変)：CEA症例に合併していた脳血管病変を**Table 1**にまとめた．多発性病変としては両側CEA施行例が22例で，対側に50%以上の狭窄病変を有しながらもCEAを施行しないで経過観察を行っていた症例が27例であった．対側内頸動脈閉塞例が12例，中大脳動脈閉塞例3例であった．これらのうち症候性で安静時血流量が20%以上低下し，ダイアモックス負荷テストで反応性が低下していた9症例ではSTA-MCA吻合術を施行したのちにCEAを行った．

near-occlusion病変：near-occlusionと判断した7症例にCEAを施行した．IVUS (intravascular ultrasound)カテーテルを血管外から使用する方法で病変の広がりを把握した．シャントチューブは全例問題なく装着できた．全例合併症なく十分な血行再建が得られた．

Table 1 Summary of complicated CEA cases with multiple lesions and restenotic changes

Bilateral ICA stenoses & CEAs	22 cases
Contralateral ICA occlusion	15 cases
(STA-MCA anastomosis	9 cases)
Contralateral stenosis>50%	27 cases
Near occlusion	7 cases
ICA stenosis & Cerebral aneurysm	10 cases
(Clipping	8 cases)
Restenosis	9 cases
(Reoperation	4 cases)

脳動脈瘤を伴った病変：CEA症例の12例に脳動脈瘤の合併を認め，8例にクリッピング術を施行した．CEA後に動脈瘤のクリッピング術を施行した症例は1例のみで，7例はクリッピング後にCEAを施行していた．

パッチグラフトを要した病変：パッチグラフトは，グラフトとしては足関節近傍から採取した静脈片かHemashieldを用いた．またパッチグラフトの適応は，繊維性狭窄病変例，内頸動脈の狭窄病変が3cm以上にわたる例，再狭窄例とした．パッチグラフトは，シャントチューブをステントとして利用し連続縫合で行った(**Fig. 2**)．

静脈片を用いたパッチグラフトを15側，Hemashieldを用いたパッチグラフトを18側のCEAで施行していた．術後1年間の観察で静脈片を用いた1例で再狭窄を認めたが，Hemashieldを用いた症例では再狭窄は認められなかった．

再狭窄(1年以内での超音波検査で50%以上の狭窄)病

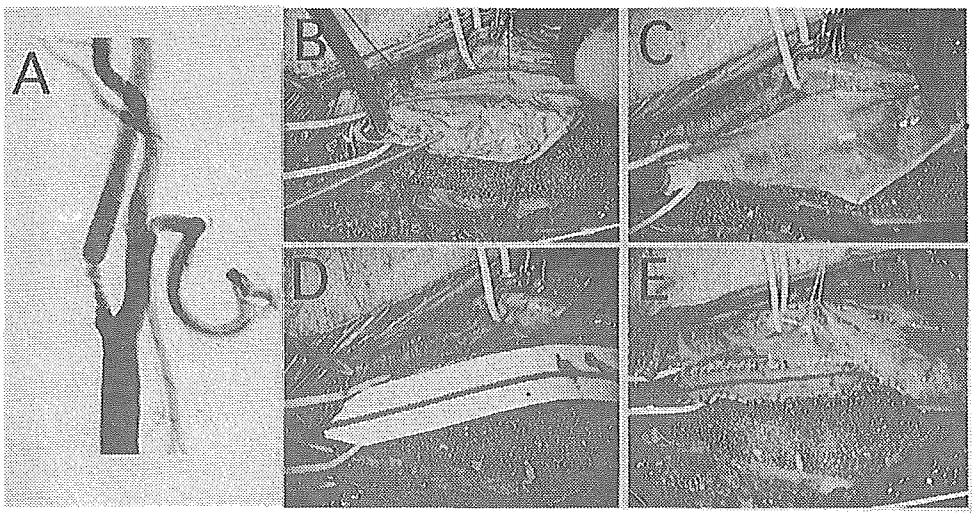


Fig. 2 Carotid angiography shows long segment severe stenosis of the ICA (A). The stenotic lesion is dissected using our shunt system (B). The stenotic lesion is removed completely (C). The size and shape of Hemashield patch graft is adjusted to the arteriotomy. (D). Arteriotomy is closed with Hemashield patch graft by running sutures (E).

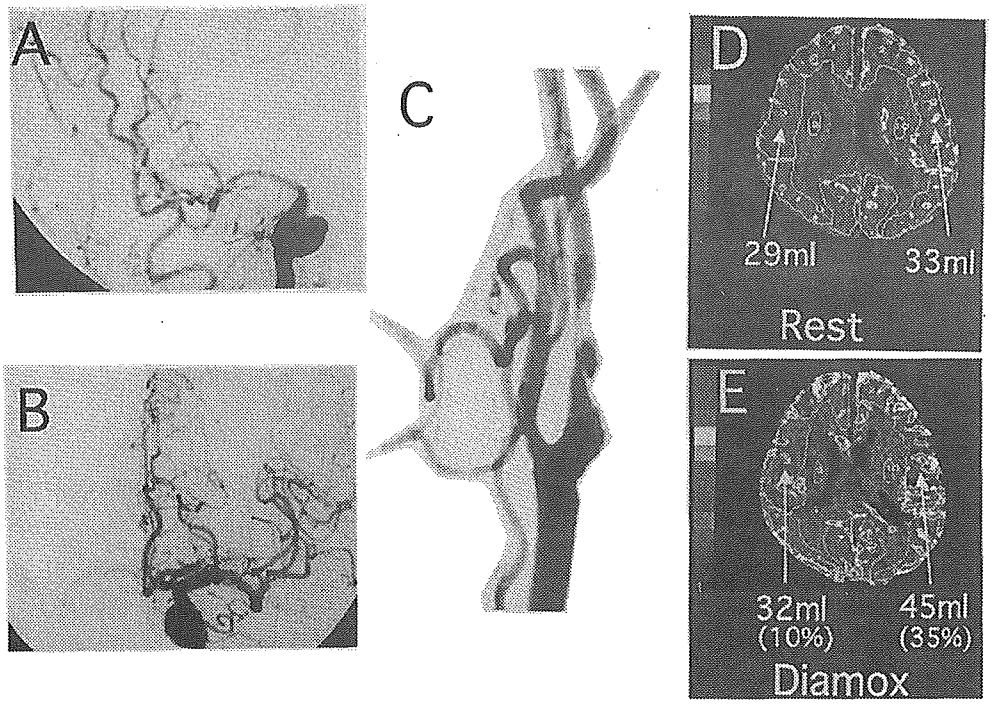


Fig. 3 Right carotid angiography shows poor visualization of the right A1 (A). Left carotid angiography demonstrates poor anterior cross circulation (B). Right cervical carotid angiography shows the severe ICA stenosis with deep ulcer (C). Cold Xe-CT's reveal significant decrease in resting cerebral blood flow (D) and poor vasoreactivity (E) on the right hemisphere.

変：9例に再狭窄を認め，1例のみ症候性であった．9例の再狭窄例のうち1例で静脈片を用いたパッチグラフトを施行されていた．9例のうち症候性の1例を含めた4例にHemashieldを用いたパッチグラフトを用いた再手術を施行した．

過還流症候群：6症例で術後過還流状態(電磁血流計で200 ml/min以上の増加，transcranial Doppler (TCD)で中大脳動脈本幹部の平均血流速度が100 cm/secを超える)を認め，2例に脳内出血を認めた．脳内出血をきたした2例に術後神経症状をきたした．代表例をFig. 3, 4に示した．症例は，72歳男性で，2000年9月に一過性左片麻痺をきたし内科的治療を受けていたが，2001年2月再度左片麻痺をきたし入院となった．画像診断で右頸動脈に高度狭窄病変を認め，anterior cross circulationはきわめて乏しい状況であった．Cold Xe-CTでは安静時血流が40%低下し，ダイアモックス負荷テストで反応性は約10%と低下していた．TCDによる中大脳動脈血流動態の検索で術前の平均血流速度30 cm/secが術後100 cm/secに増加した．血圧を厳重にコントロールするためにバルビタール療法を48時間行ったが，24時間後のCTスキャンにて脳内出血を認めた．バルビタール療法後軽度の左片麻痺の悪化を認めたが，1カ月後に独歩退院となった．

心疾患：CEA症例に狭心症や心筋梗塞を合併していた症例をTable 2にまとめた．狭心症，心筋梗塞の既往歴は，62例に認められ，16例でCABG，14例でPTCAやステントの治療を受けていた．術前に冠動脈造影が施行された症例は48例で，この結果からCEAの前に予防的にPTCA，ステント留置術を3例に施行していた．また不整脈は18例に認められ，5例にペースメーカーが装着されていた．

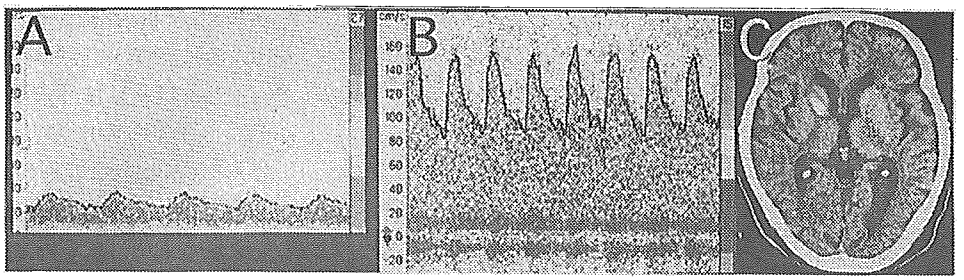
CEAの成績は，mortalityはゼロ，morbidityは6例(2%)で内訳は創部出血が2例，塞栓症が2例，過還流症候群が2例であった．

術後経過観察で死亡例は19例あり，その内訳は癌11例，虚血性心疾患5例，肺塞栓症2例，筋萎縮性側索硬化症1例であった．

考　察

頸動脈狭窄病変の治療には国際的な共同研究からEBMに基づいた指針が示されている[7)8)15)17)]．しかし個々の症例や手術術式などにはさまざまな問題点が残されている．今回（1）病変の広がり，（2）多発性病変（両側狭窄病変と対側閉塞病変），（3）near-occlusion病変，（4）脳動脈瘤を伴った病変，（5）パッチグラフを要した病変，（6）再狭窄病変，（7）過還流症候群を呈した病変，（8）心疾患について検討した．

病変の広がり：豊田ら[21)]は頸動脈写を用いて本邦例と



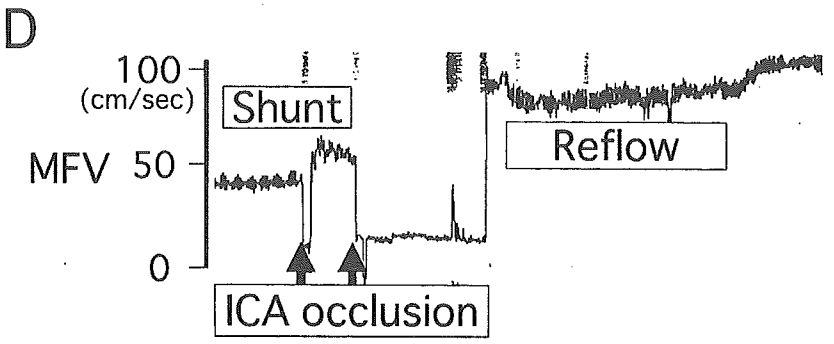


Fig. 4 Intraoperative TCD demonstrate mild decrease in flow velocity in the right MCA (A), which is normalized by a shunt placement (D). The mean flow velocity of the middle cerebral artery prominently increases over 100 cm/sec (B, D). Postoperative CT shows intracerebral hemorrhage at the right caudate putamen (C).

ハンガリー人の頸部頸動脈の分岐部の位置について検討している．本邦例の頸動脈分岐部の位置は，第3頸椎の下部1/3が27%，中部が1/3が17%，上部1/3が12%で，半数以上で第3頸椎に位置していた．一方ハンガリー人の頸動脈分岐部は，約60%が第4頸椎領域に位置していた．自験例324側のCEA症例での分岐部の位置は，約55%が第3頸椎より高位であった．また病変の末梢部は約1/3の症例で第2頸椎のレベルであった．以上から本邦の頸動脈狭窄病変は高位病変が多数を占めていると考えられ，CEAにおいては留意すべきである．

多発性病変(両側狭窄病変と対側閉塞病変)：多発性病変に対する治療に関してはいくつかの問題がある．すなわち両側頸動脈狭窄病変の場合両側同時手術か一側ずつの手術か，後者の場合ではどちらの手術を先に行うか．また片側内頸動脈閉塞を伴う例においては閉塞側のバイパス術と狭窄側のCEAの治療選択などがある．両側頸動脈狭窄に対するCEAでは同時手術も安全に行える可能性を示した報告もあるが，片側障害による無症候性の上喉頭神経麻痺の問題など大きなリスクは否めない．したがって現時点では著者らは最近の症候側のCEAを第一とし，6週間以上の間をあけて対側のCEAを基本としている．しかし血管内ステント留置術では上喉頭神経の問題はないため，今後血管内手術を含めた新たな治療戦略の確立が求められる．

Table 2 Summary of CEA cases with is cardiac complications

History of angina	62 cases
Coronary angiography	48 cases
CABG	16 cases
Stenting	14 cases
Stenting & CABG	3 cases

対側内頸動脈閉塞例では症候側が閉塞側か狭窄側かが大きな問題である．対側閉塞例での狭窄側のCEAの周術期のmortalityやmorbidityに関しては危険因子となるという報告と問題がないという報告がある．NASCETのデータ分析やOntarioのCEAデータでは対側閉塞を危険因子として報告している[9)22)]．一方ACASのデータ分析やAbuRahmaらの報告では対側閉塞は問題とならないと結論している[1)4)]．これらの結果には脳血流検査がなく，症候の原因が閉塞側と狭窄側か，また血行動態かartery-to-arteryの塞栓によるものかなどさらに検討が必要と考えられる．著者らは閉塞側の血流低下(正常の80%以下)，血管反応性低下(10%未満)の場合には閉塞側のバイパス術を先に行うことを原則としている．

near-occlusion病変：本検討ではMorgensternら[16)]が定義したnear-occlusionの中でより狭窄度の高いnear-occlusion with a string-like lumenの症例を対象とした．この

ような病変に対しては頭蓋内—外バイパス術とCEAが考えられたが，著者らはより本幹部の病変を治療する方針でこのような病変に対してCEAを採用してきた．問題点は病変の末梢端の位置，虚脱化した末梢側内頸動脈の正常化，術後の過還流などがあげられている．川俣ら[12]は末梢側の把握に術中IVUSを用い，Yasakaら[24]は術前B-mode Dopplerを経口的に用いる方法で有用な所見を得ている．今後より簡便で確実な病変部の末梢端の把握方法の導入が求められる．またnear-occlusion症例においてのCEA適応は狭窄度の評価などの問題や内科治療群の良好な結果など今後検討されるべきである．

脳動脈瘤を伴った病変：脳動脈瘤症例は，309例中12例(3.8%)であった．この未破裂脳動脈瘤の治療選択は，動脈瘤のサイズ，形状など動脈瘤の問題と頸動脈狭窄に伴う虚血脳に対する侵襲を考慮すべきである．動脈瘤を未処置のままにして頸動脈狭窄病変の加療を行った場合には破裂の危険性が増すか否かが問題である．Ladowskiら[13]の19例のreviewではCEA中や周術期での動脈瘤の破裂の危険性は低く，CEAにより未破裂動脈瘤の破裂を誘発することはないと結論している．しかしOrecchiaら[18]は，術後経過中に破裂例もあり，動脈瘤の大きさ，形状による動脈瘤の処置も考慮すべきことが示唆されている．

パッチグラフト：欧米ではCEA後の再狭窄を予防する目的でパッチグラフトをルーチンに施行している施設もある．パッチグラフトとしては静脈片と人工血管材料(Hemashield)が用いられている[3][14]．自験例では静脈片とHemashieldをほぼ同数に用いていた．選択の基準としては良い静脈片が利用できる症例では自家静脈片を用い，両側例や静脈瘤などがある場合にはHemashieldを用いた．静脈片で1例再狭窄を認めたが，Hemashieldでは1例の再狭窄例もなく，今後の検討が必要である．またパッチグラフトの適応に関しても本邦例での検討が求められる．

再狭窄：再狭窄は周術期を乗り切ったのちの大きな問題である．文献的には5-10%程度の再狭窄率が報告されているが，自験例では2.8%であった．再狭窄病変は，myointimal hyperplasiaとされており，内膜剥離部の平滑筋の異常増殖によるものである．病理組織所見などからmyointimal hyperplasiaをきたしやすい症例の推察もされているが，術前，術中に判断することは困難である．また再狭窄例のCEAはきわめて困難であり，神経損傷などの合併症も高くなっている．したがってHemashieldなどを用いたパッチグラフトや再狭窄の早期診断，ステント留置術などによる新しい治療法の確立が求められる．

過還流症候群：Hyperperfusion Syndromeは，Sundtら[20]によれば術後早期の血流測定で200%以上の脳血流増加をきたし，頭痛，痙攣，脳内出血を呈するものである．この合併症は3-5日以内に発症することが多く，術後脳血流の著明な増加が伺われる場合には厳重な血圧管理が求められている．しかし術直後からの連続的なTCDなどによる脳血流の検討では過還流状態は術直後から生じており，3-5日目に発症する機序に関してより詳細な検討が今後求められる．また血圧管理の方法にもプロポフォールのような新しい鎮静方法の検討も早急な課題である．

心疾患：頸動脈狭窄病変と冠動脈閉塞性病変の合併に関してNASCETの登録例では約40%に心筋虚血の既往歴が報告され，心筋虚血のある群とない群で心筋梗塞による死亡率では前者が有意に高いことが報告されている[11]．また高齢(75歳以上)，高血圧，糖尿病，喫煙歴，腎不全，心電図での左室肥大，心電図での心筋梗塞のうち4つ以上有している症例では積極的な冠動脈撮影の必要性を示唆している．本邦でも宇野ら[23]は頸動脈狭窄症例の半数近くで有意な冠動脈狭窄を認め，CEA症例での冠動脈精査の必要性を示唆している．冠動脈狭窄病変を合併する症例に対する治療は，血管内治療の導入，発展で大きく変化し始めている．冠動脈閉塞性病変を有している症例のCEAに関しては心筋虚血症状がなく，心機能が安定している場合は心筋保護(大量モルフィン麻酔，ニトロ製剤)を図りながらCEAが施行できる．冠動脈狭窄病変が重症で負荷心筋シンチが陽性を示す症例では冠動脈の血管内治療(PTCAやステント留置術)を施行したのちにCEAを検討する．血管内治療が困難，禁忌の症例ではCABGが選択される．この場合頸動脈が無症候性病変であればCABGを先に行う．頸動脈病変が症候性であれば頸動脈の血管内治療(ステント)を行い，CABGを施行する[2]．しかし頸動脈，冠動脈ともに症候性でかつ血管内治療に不適切な症例ではCEAとCABGを同時に施行することも考慮される．同時手術の成績としてYanakaらは6例でmortality，morbidityが0%と報告している[25]．海外の多数例での報告ではmortality 4%，morbidity 3.3%という良好な結果からcombined stroke & deathが13.8%というけっして受け入れられないような結果まであり，総じて良好な成績ではない[5][6]．近年off-pump coronary artery bypass (OPCAB)が導入され，頸動脈狭窄病変による血行不全からの脳虚血を防止できる血圧80 mmHgを維持しながら冠動脈血行再建が可能となってきた．著者の施設でも冠動脈病変精査中にcritical levelの狭窄を示している頸動脈病変を合併している症例ではOPCABを選択している[10]．

CEAの欧米の手術成績では1-5%のmortality & morbidityが報告されている．著者の成績は，mortality 0例，morbidity 6例(1.9%)で，本邦主要施設でのmorbidity & mortalityも4-6%である．しかし症例数の少ない本邦で

は手術適応を厳格にし，多様な頸動脈病変に対するより安全確実な治療法の確立が求められる．

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狭窄性脳血管病変に対する外科治療

——頸部内頸動脈以外の頭蓋外血管狭窄——

Surgical treatments for extracranial occlusive cerebrovascular diseases

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◆key words：脳動脈閉塞性病変，血行再建術，人工血管

はじめに

頭蓋外脳主幹動脈の狭窄・閉塞性病変は，頸部内頸動脈閉塞・狭窄症が代表的な疾患である。この頸部頸動脈狭窄病変に対してはもっともRCT（randomized controlled study）が進められ，内科的治療と外科的治療（CEA；carotid endarterectomy）の比較検討により病変の程度と術者の技量からCEA選択のガイドラインが作成され，もっともEBM（evidence based medicine）の確立した外科治療分野となっている[1)2)]。さらにこの分野では血管内治療も長足の進歩を遂げ，CEAとのRCTで優位な結果まで報告されるようになっている[3)]。一方この頸部頸動脈狭窄病変以外にもさまざまな頭蓋外の頸動脈，椎骨動脈，腕頭動脈，鎖骨下動脈狭窄・閉塞などが知られている[4)5)]。しかしいずれもRCTによるデータはなく，各施設でのさまざまなアプローチから外科的治療が進められている[6)-8)]。この外科的治療に関しては診断・適応とともに手術方法にもさまざまな工夫や改善がなされている。診断に関しては脳血管撮影はgolden standardではあるが，合併症などの危険性からMRA，超音波ドップラー，3D-CTAなどの応用が進んでいる。一方，外科的治療に関しては，血行再建に人工血管や静脈片を用いるさまざまな方法が導入されている[9)10)]。

本稿では筆者が進めてきた頸部頸動脈狭窄病変に対するCEA以外の頭蓋外脳主幹動脈の狭窄・閉塞病変に対する外科的治療法について述べる。

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頭蓋外脳主幹動脈の狭窄・閉塞病変と外科的治療

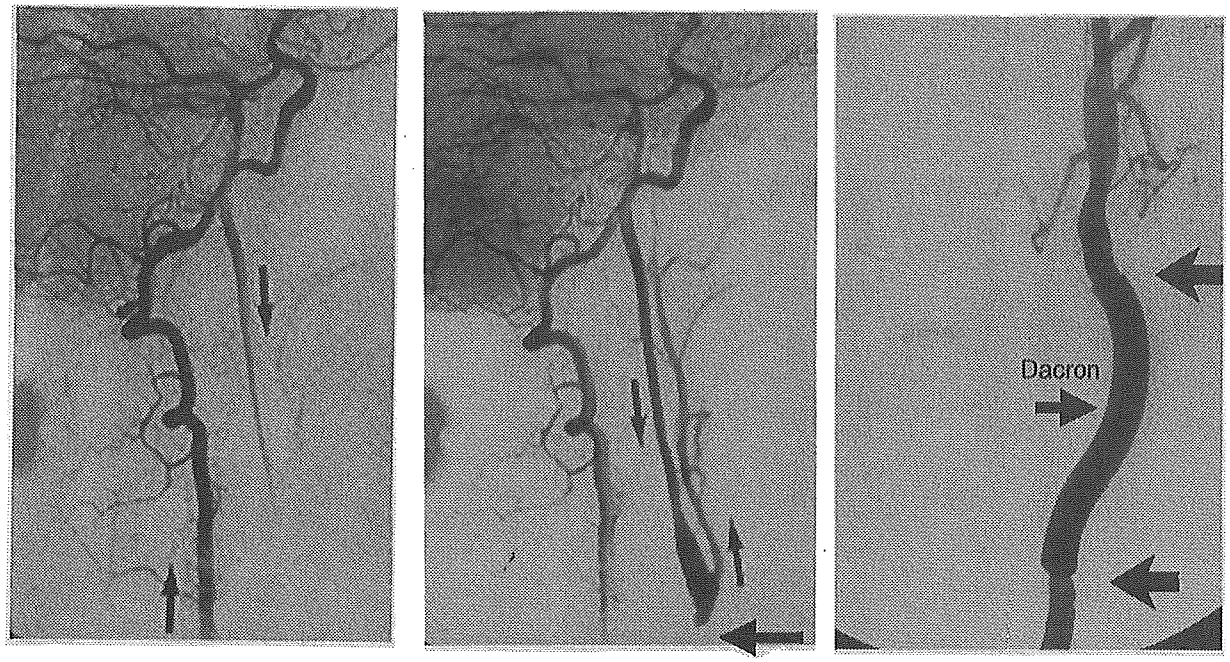
頭蓋外脳主幹動脈の狭窄・閉塞病変は，さまざまな部位に生じることが知られている。代表的な頭蓋外狭窄・閉塞病変としては動脈硬化性病変で粥腫形成による頸部内頸動脈狭窄症があげられる。本稿では粥腫による頸部頸動脈狭窄病変を除いた，大動脈弓から頭蓋に達するまでの脳主幹動脈の狭窄・閉塞病変を対象とする。このような疾患としては外傷や放射線治療などに伴う頸動脈狭窄・閉塞病変，大動脈炎症候群，動脈硬化性病変に伴う椎骨動脈閉塞症，腕頭動脈・鎖骨下動脈狭窄・閉塞に対する外科的治療について述べる[4)-8)11)]。

代表例

1. 外傷性総頸動脈閉塞

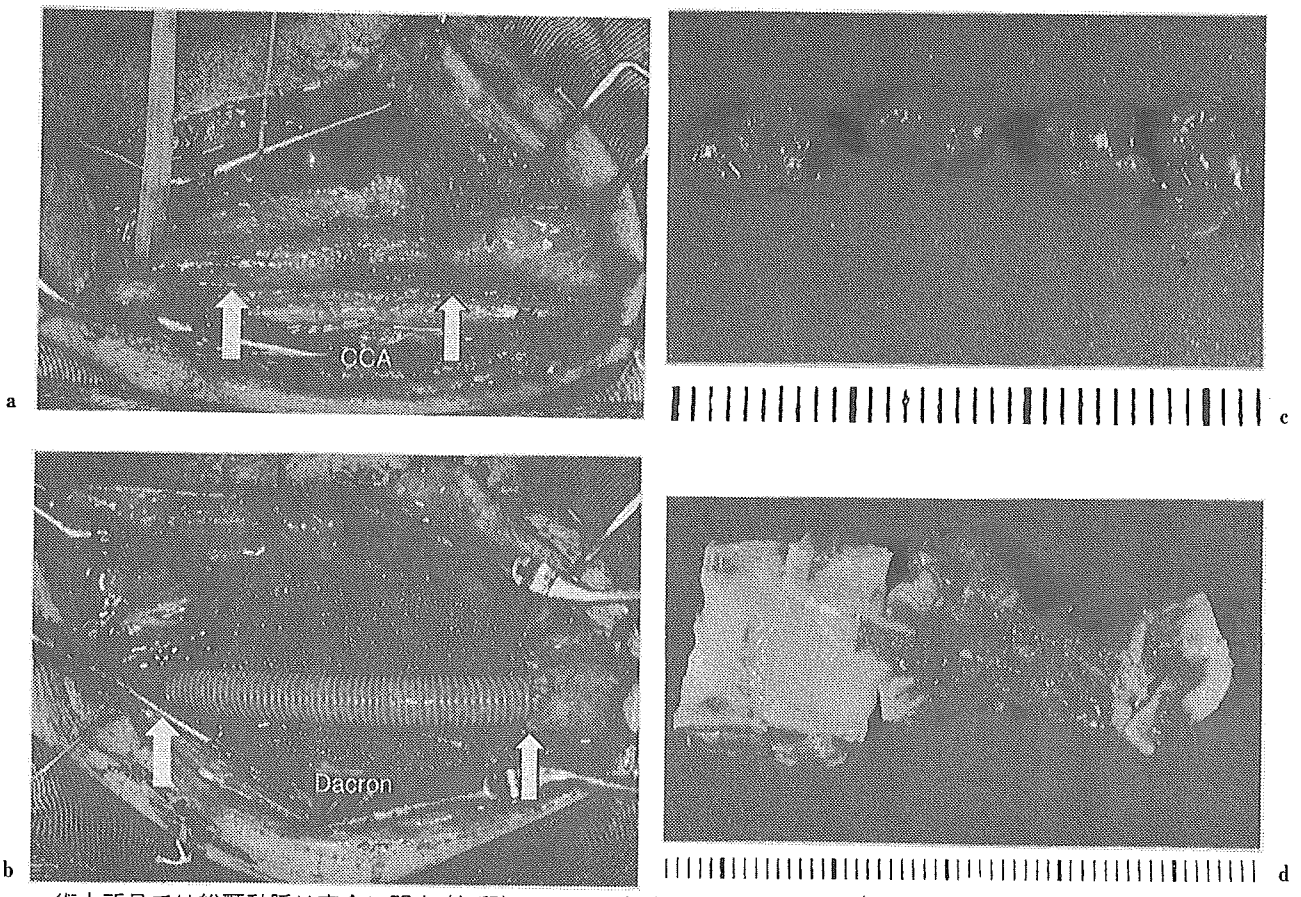
症例：患者は46歳，男性。作業中にプロペラにて左頸部を強打し裂傷を負った。大量出血に対し創部の結紮にて救急処置を受けた。幸い片麻痺などの神経脱落症状はみられなかったが，受傷3カ月目頃より起立動作時に眩暈が生じるようになった。CTスキャンなどの検査では頭蓋内には異常所見はみられなかった。血管撮影にて左頸動脈の閉塞と椎骨動脈から後交通動脈，左内頸動脈，頸動脈分岐部を介して外頸動脈に向かう血流を認めた（図1a，b）。

手術：全身麻酔下に頸動脈を露出した。損傷部は周囲組織と強固に癒着しており，完全閉塞の状況であったが，末梢側ではほぼ正常な頸動脈分岐部，内頸動脈，外頸動脈を認めた（図2a）。損傷部位を切除しDacron（径6mm）を用いて血行再建を行った（図2b）。摘出した損傷動脈には繊維性肥厚と血栓



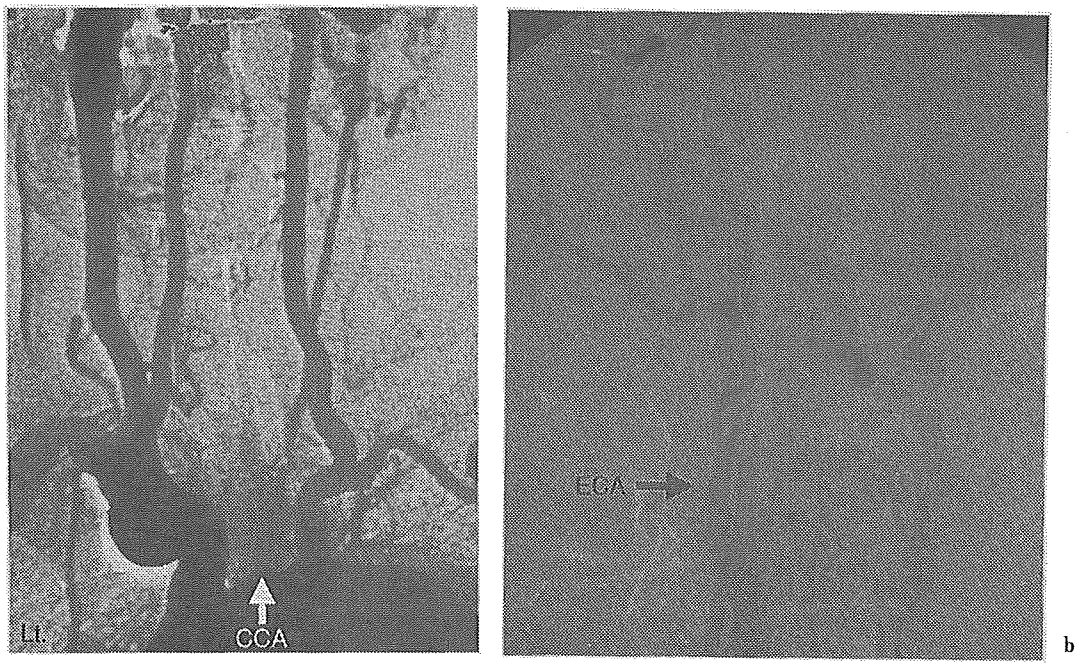
左椎骨動脈写で後交通動脈を介して左内頸動脈-外頸動脈が造影され，総頸動脈は完全に閉塞している（矢印）（a，b）。術後 Dacron を用いた血行再建（矢印）により良好な血流を認める（c）

図 1　外傷性総頸動脈閉塞症例(1)



術中所見では総頸動脈は完全に閉塞（矢印）している（a）。総頸動脈の損傷部位は Dacron にて置換している（b）。摘出した損傷部位には血栓が充満し（c），動脈壁は器質化している（d）

図 2　外傷性総頸動脈閉塞症例(2)



panaortogram で左総頸動脈（矢印）の高度狭窄を認め（a），左頸動脈写では筋肉枝を介した左内頸動脈系へ側副路の発達を認める（b）

図3　大動脈炎症候群症例(1)

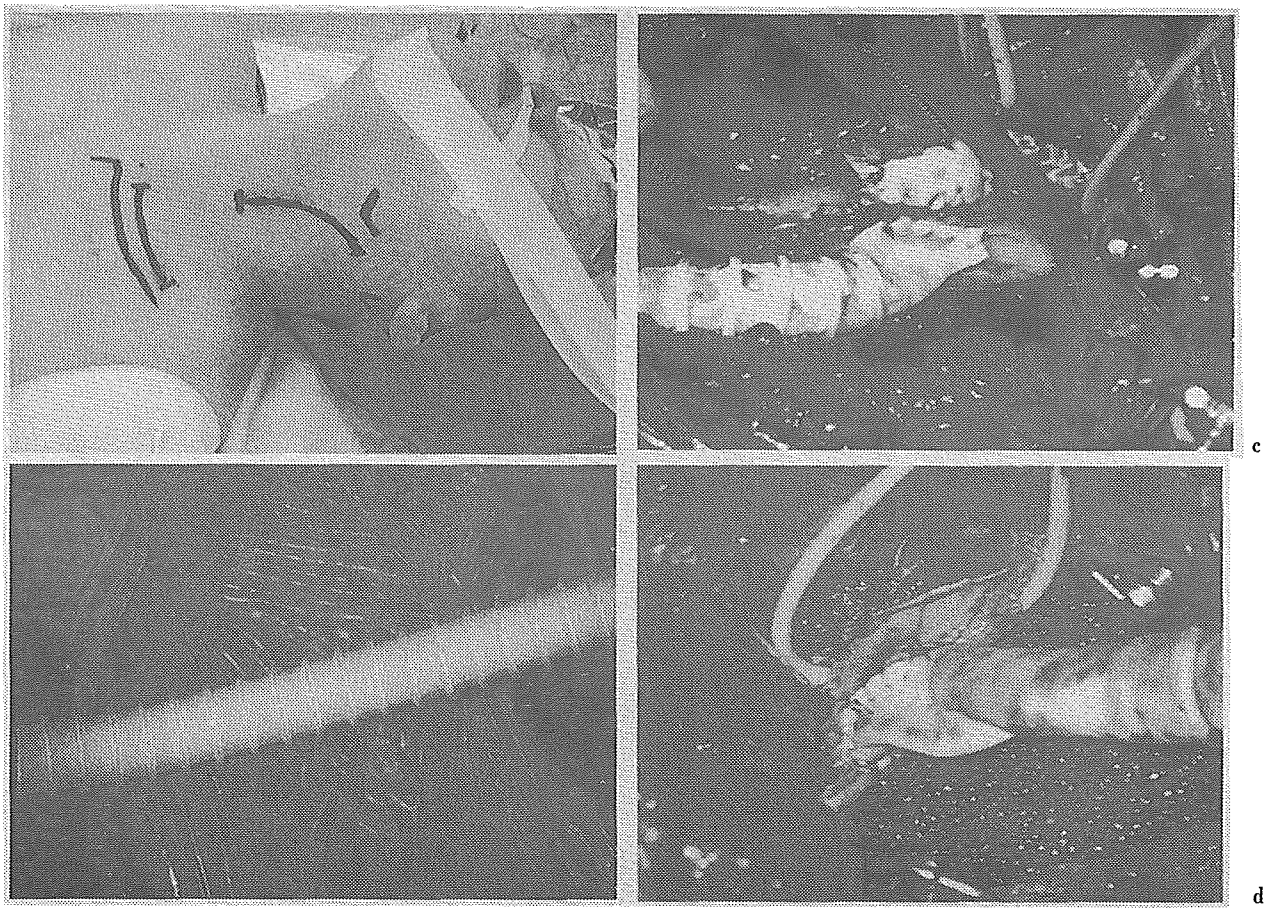
形成を認めた（図**2 c**，**d**）。術後血管撮影（図**1 c**）にて良好な血流を認め，眩暈などの愁訴も消失した。

2.　大動脈炎症候群

症例：患者は37歳，女性。胸部大動脈瘤，大動脈炎症候群の診断を受け加療中であった。約1年前より眩暈発作が生じ，日常の活動にも支障をきたすようになり精査を受けた。脳血管撮影で左総頸動脈の高度狭窄，左外頸動脈の閉塞，左外頸動脈系と椎骨動脈系の間に側副路の著しい発達を認めた（図**3 a**，**b**）。cold-Xe CT による脳血流測定では明らかな低灌流域はみられず，Diamox 負荷テストでもほぼ正常の反応を認めた。内科的な治療にもかかわらず体位変換時などの眩暈発作の改善が得られないため，左鎖骨下動脈と左頸部内頸動脈間の人工血管を用いたバイパス術を計画した。全身麻酔下に仰臥位にて図**4 a** のような皮膚切開を設けた。鎖骨下動脈を thyrocervical trunk 分岐部で確保した。頸部頸動脈は分岐部を中心に剝離・露出したが，分岐部から外頸動脈に至る約2 cm が閉塞していた。総頸動脈は紐状の hard な組織であったが，内頸動脈はほぼ正常の動脈構造であった。鎖骨下動脈と内頸動脈間の皮下に人工血管（Gore-Tex の径5 mm のリング付き ePTFE）を通すために長さや方向を調節した（図**4 b**）。内頸動脈に径5 mm の vascular punch で側孔を穿ち，人工血管と end-to-side の吻合を行った。吻合部からの出血はフィブリン糊の塗布でコントロールは容易であった（図**4 c**）。次に鎖骨下動脈に5 mm 径の側孔を設け，人工血管の長さを調節し，吻合を行った（図**4 d**）。術後の MRA ではバイパスを介した良好な血流を確認した（図**5 a**，**b**）。また臨床症状は，後頸部の拍動感の消失やめまい感の著明な軽減が得られた。

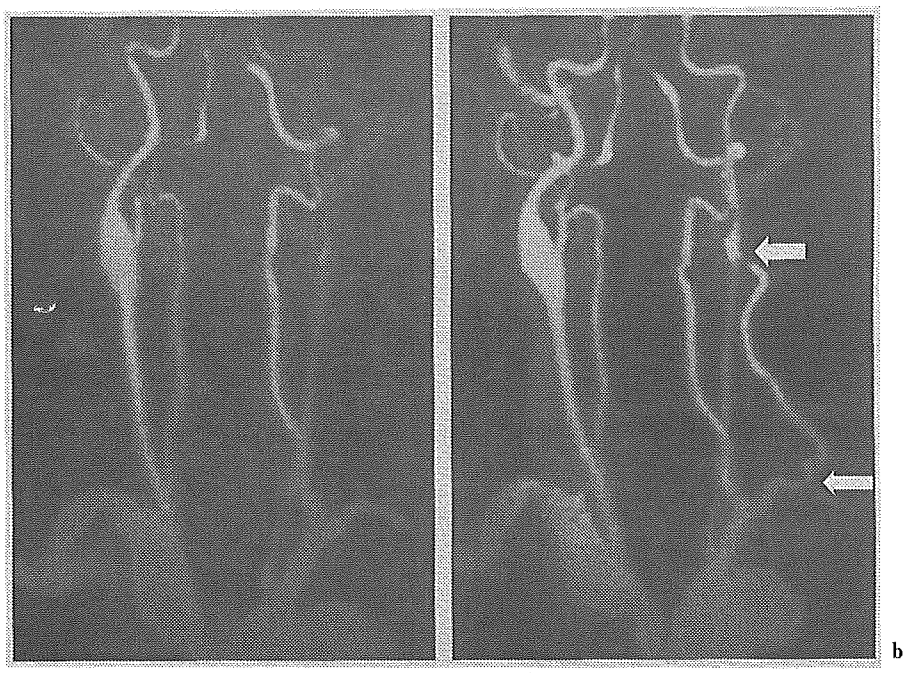
3.　腕頭動脈，鎖骨下動脈閉塞病変

症例：患者は69歳，男性。血圧の左右差が20～30 mmHg あり，体位変換や運動にて眩暈発作が生じ，後頸部熱感，頭痛もしばしば経験していた。突然左片麻痺が生じ，精査の結果腕頭動脈閉塞と脳梗塞が指摘された（図**6 a**，**b**）。右頸動脈系の血流改善のため axillary-axillary bypass を予定した。全身麻酔下に両側の鎖骨下約2 cm に6 cm の横切開の皮膚切開を設け，大胸筋を筋束に沿って分け，さらに小胸筋を外側に圧迫して両側の腋窩動脈を露出した（図**7 a**，**b**）。人工血管（径5 mm のリング付き ePTFE）を両側の創部間の皮下に通した。右側の腋窩動脈を約3 cm にわたり Sugita clip にて trapping し，径5 mm の vascular punch にて側孔を設け，人工血管と end-to-side anastomosis を行った。吻合は6-0プロリンを用い，2点固定をおいて連続縫合で行った（図**7 b**，**c**，**d**）。次に人工血管の長さを十分に調整した後に対側の腋窩動脈と人工血管の



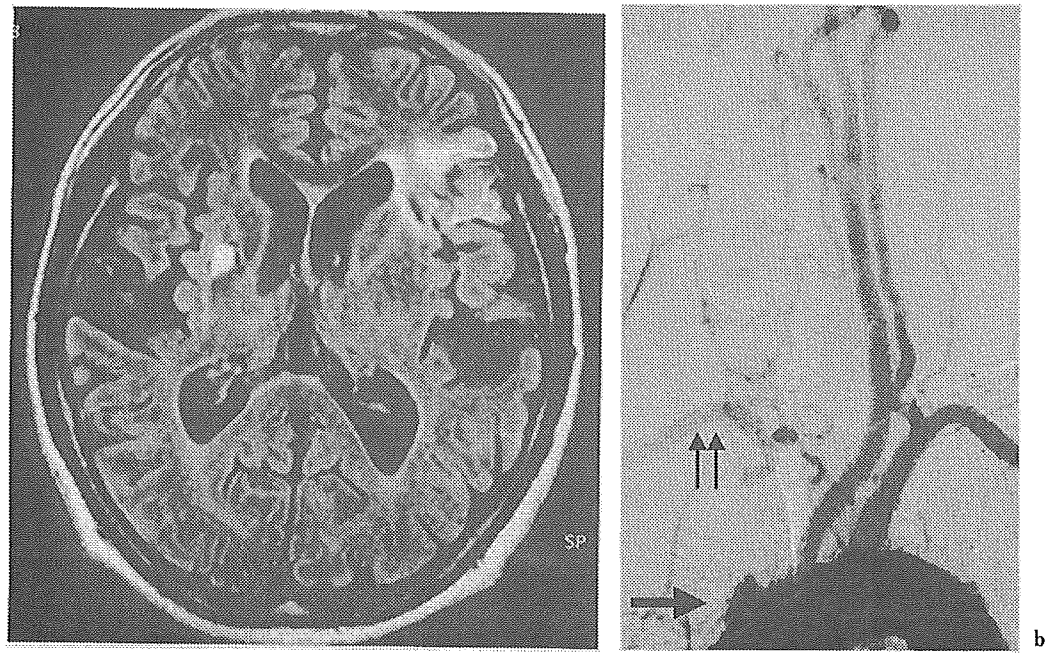
手術体位と皮膚切開（a）。鎖骨下動脈と内頸動脈間のバイパス人工血管（径5 mmのリング付きePTFE）調整（b）。内頸動脈と人工血管と吻合（c）。人工血管と鎖骨下動脈の吻合（d）

図4　大動脈炎症候群症例(2)



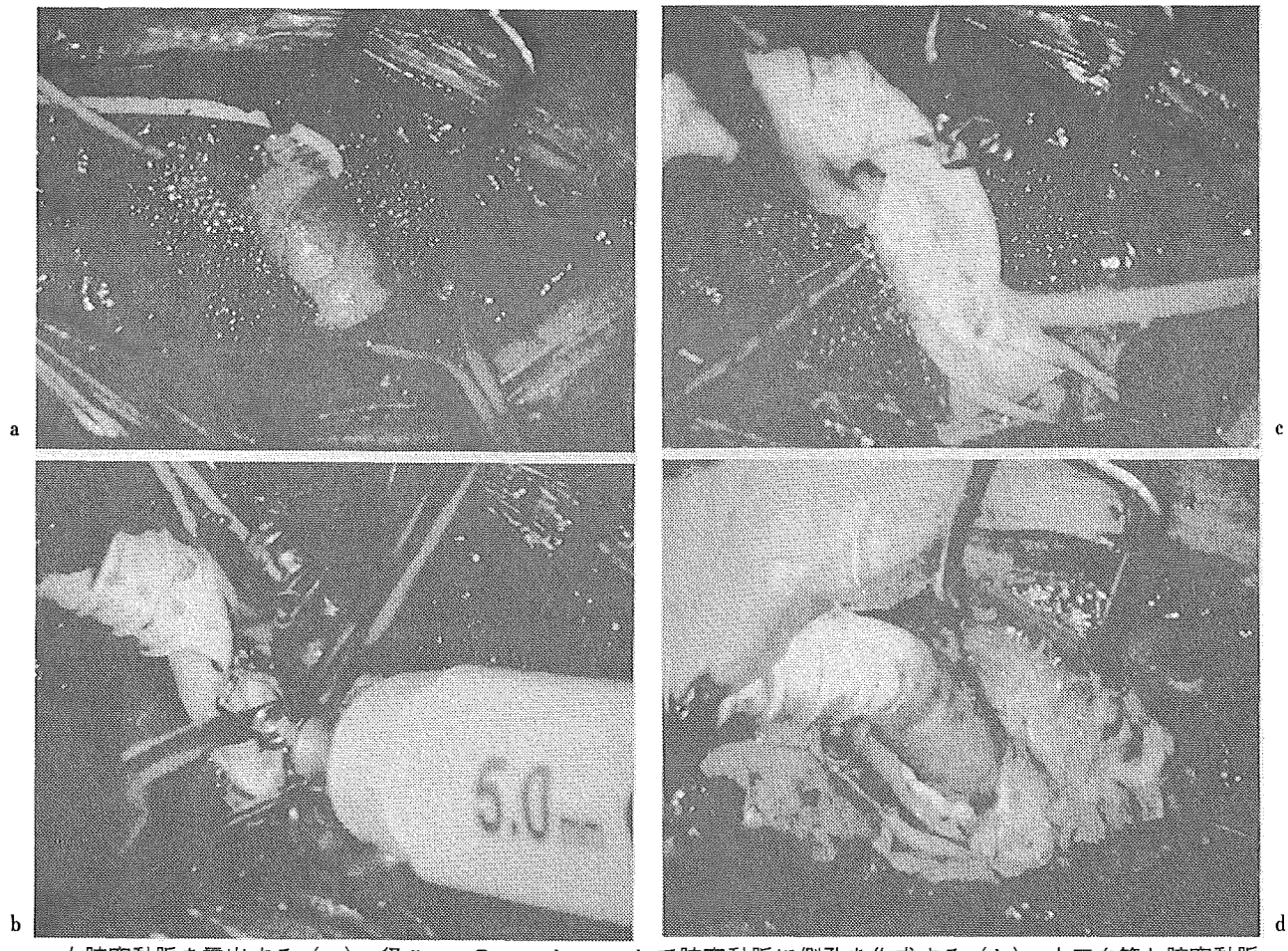
術前のMRAで総頸動脈の高度狭窄を認める（a）。術後のMRAでは人工血管（矢印）による血行再建が確認できる（b）

図5　大動脈炎症候群症例(3)



MRI で右基底核，左前頭葉に梗塞像を認める（a）。panaortogram で腕頭動脈の閉塞（大矢印）を認め，筋肉枝を介した側副路により右鎖骨下動脈（2本矢印）の淡い造影を認める（b）

図 6　腕頭動脈，鎖骨下動脈閉塞病変症例(1)



右腋窩動脈を露出する（a）。径 5 mm の vascular punch で腋窩動脈に側孔を作成する（b）。人工血管と腋窩動脈を吻合する（c，d）

図 7　腕頭動脈，鎖骨下動脈閉塞病変症例(2)