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The Brain Aneurysm Institute

Multidisciplinary Care of Patients with Hemorrhagic and Ischemic Stroke

WINTER 2025

Neurovascular News

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The current role of surgery for intraparenchymal brain hemorrhage

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Spontaneous intraparenchymal hemorrhage (IPH)/hemorrhagic stroke, accounts for 6.5% to 19.6% of stroke cases[1, 2], but is associated with the majority of mortality and morbidity (1 year survival = 40%, 10 year survival = 24% and functional independence at follow-up varying between 12% to 39%). More than two thirds of IPH cases are "Primary IPH", typically attributed to hypertension or cerebral amyloid angiopathy (CAA). A smaller number of cases are due to "Secondary IPH" with an underlying cause such as coagulopathy, cerebral venous sinus thrombosis (CVST), moya moya, vasculitis, tumor, hemorrhagic conversion of ischemic stroke, mycotic aneurysm or arteriovenous malformation rupture.

Clinical presentation, assessment and diagnosis

IPH typically presents with sudden headache, seizures and focal neurological deficit. The presence of headache, nausea or vomiting and a depressed mental status should prompt suspicion for an IPH rather than an acute ischemic event[3]. Patients are prone to rapid deterioration, often during transport to a hospital or while waiting in the emergency department with the use of antiplatelet agents being one of the risk factors for deterioration.[4] Rapid CT or MR imaging should be obtained (Class I evidence).

Best medical management of primary IPH

Medical management of primary IPH involves admission to a dedicated stroke unit/intensive care unit, maintaining a systolic blood pressure (SBP) goal of less than 140 mmHg, aggressive reduction of systolic blood pressure if SBP is >220 mmHg, maintaining normoglycemia, seizure management with antiepileptic drugs (AEDs) without prophylactic treatment, early dysphagia screening, ECG and troponin, pneumatic compression stockings for DVT prophylaxis (subcutaneous heparin 1-4 days after hematoma stability is confirmed).[5]

Role of surgery in patients with IPH

1. External ventricular drain placement is recommended for hydrocephalus especially in patients with an altered level of consciousness. [Class IIa, Level B]
2. Decompressive craniectomy is recommended for supratentorial decompression in patients with a low GCS of 8 or less (with or without hematoma evacuation). These are also recommended in patients with very large hematomas (>30 ml) with significant midline shift (>10 mm) or refractory high ICP despite best medical management. [Class IIb, Level C]

3. Cerebellar hemorrhages with or without hydrocephalus, at least 3 cm in size can be managed by decompression and evacuation. [Class I, Level B]

The role of surgical evacuation of primary IPH has been studied extensively and there have been numerous trials to evaluate its benefit to patients in terms of survival and functional recovery. The original STICH trial (Surgical trial in intracerebral hemorrhage) was initiated to gain robust evidence after several prior trials showed conflicting results of efficacy of surgical intervention. The STICH trial showed a small, non-significant advantage of surgery over conservative management. This was considered good evidence of non-superiority of surgery for such patients, however, later studies and subsequent meta-analysis of sub-groups showed a significant benefit in terms of mortality for patients having lobar hemorrhages (superficially coming to the surface of the cortex). The STICH II trial was subsequently designed and powered to compare early surgical evacuation (within 12 hours) of lobar hematomas (without IVH) with initial medical management, in terms of functional outcomes. This trial confirmed that early surgery was non-inferior to medical management in terms of death and disability at 6 months. The option of minimally invasive evacuation of IPH was studied to reduce the morbidity associated with open surgical approaches. For superficial bleeds, minimally invasive approaches with small incisions, endoscopes and specialized tubular devices have shown a lower risk of death and disability when compared to craniotomy or conservative management. These approaches also have a lower rate of rebleeding and a higher rate of good recovery.[6, 7] The Minimally Invasive Surgery Plus rt-PA in Intracerebral Hemorrhage Evacuation (MISTIE) I, II and III trials utilized stereotactic evacuation of hematoma and subsequent thrombolysis by placement of a catheter and irrigation with alteplase. These trials did not show any significant difference in terms of mortality at 30 days or functional improvement at 1 year when compared with medical management. Subsequent post-hoc analyses of MISTIE III however, found better functional outcomes in patients with a residual hematoma volume <15 ml.[8]

The Trial of Early Minimally Invasive Removal of Intracerebral Hemorrhage (ENRICH) evaluated the benefit of early (within 24 hours of last known well) minimally invasive surgery (Nico device) plus medical management versus medical management alone for anterior basal ganglia and lobar hemorrhages. ENRICH demonstrated the futility of surgery for anterior basal ganglia hemorrhages. There was a clear benefit of early minimally invasive surgery with medical management for lobar hemorrhages when assessed for safety end points as well as functional outcomes.[9] Figure 1 demonstrates a deep seated IPH (1a, b and c) that approaches the cortical surface at the lateral temporal lobe and is hence well suited for a minimally invasive endoscopic approach that was performed (1d, e and f). Looking into the future, the Dutch ICH Surgery Trial and the EVACUATE-RCT trial aim to explore the role of endoscopic minimally invasive hematoma evacuation within 8 hours (ultra early) of symptom onset. Both are currently enrolling patients.

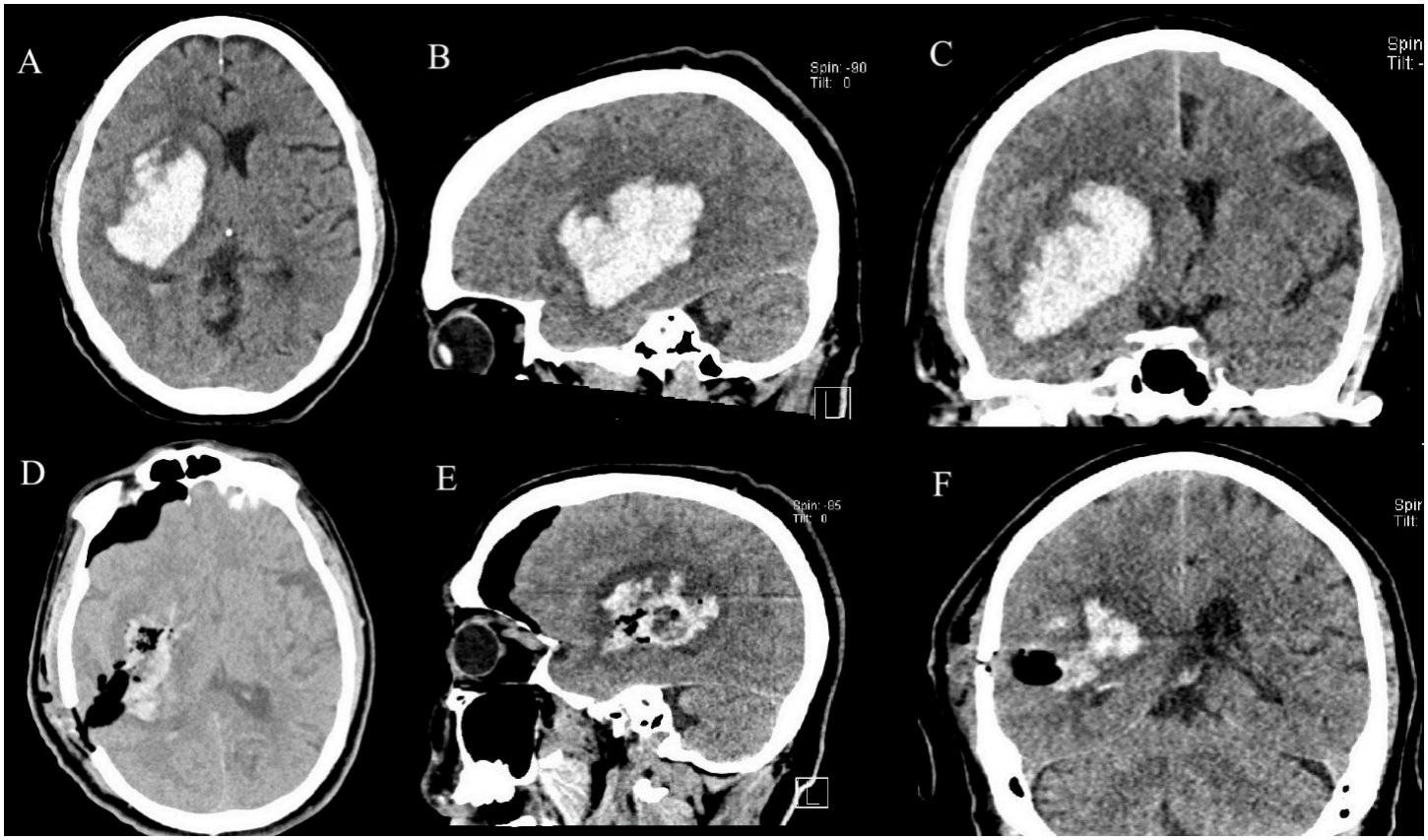

Figure 1: A deep seated right sided IPH with midline shift (a, b and c). Surgical management involved a minimally invasive endoscopic approach utilizing a single burr hole (1d) with significant volume reduction of the IPH and decreased mass effect (d, e and f)

In summary;

a. Anterior basal ganglia hemorrhages without the need for life saving decompressive craniectomy and evacuation, do not benefit from hematoma evacuation (craniotomy) or minimally invasive evacuation of hematoma in terms of mortality or functional outcome improvement.

b. Lobar hemorrhages (within 1 cm of cerebral convexity surface) benefit from early (within 24 hours) minimally invasive surgical evacuation of hematoma. This benefit is valid for both an improvement in mortality and functional outcomes provided the volume of residual hematoma is under 15 ml.

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Reduced Flow, Not Pressure, Account For Aneurysm Obliteration

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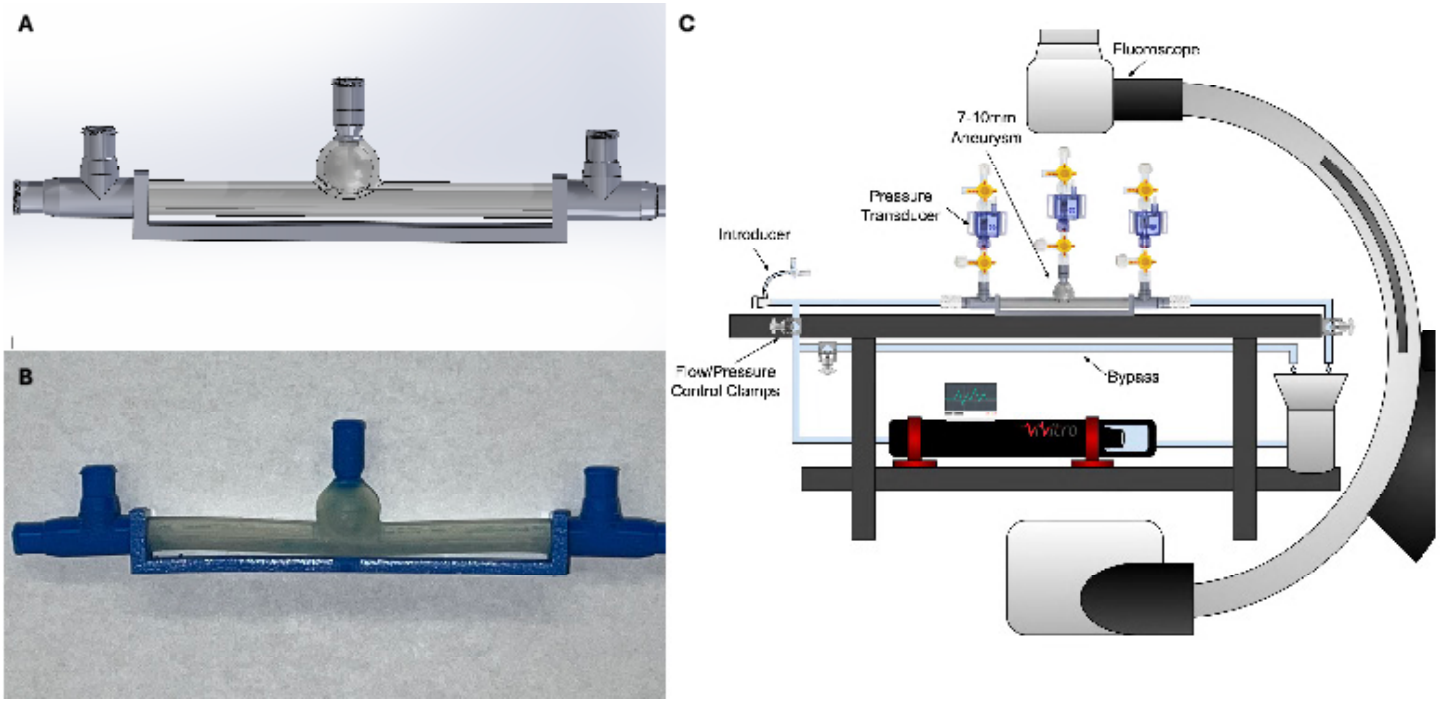

Figure 1. (A) Computer Aided Design (CAD) model of 7 mm inner diameter (ID) aneurysm dome, 3 mm ID parent vessel, with 3 pressure taps. (B) 3D-print model using soft Agilus30-Clear® for the aneurysm and parent vessel and hard Vero-Cyan® for the pressure taps and support frame. (C) Comprehensive benchtop flow system pumping BDL blood analog through the aneurysm model and bypass, with physiological flow and pressure control, under fluoroscopy

Introduction

Endovascular coiling and flow diversion (FD) are popular treatment options for intracranial aneurysms. Coiling can also be performed with stent or balloon assistance depending on the features of the aneurysm.[1] These treatments have been shown to provide safe and effective aneurysm occlusion, but questions remain regarding their impact on the hemodynamics within the aneurysm and parent vessel. There has been a lack of consensus on the effect of aneurysm coil and flow diverter placement on the pressure within the aneurysm, and whether a drop in pressure is responsible for durable aneurysm occlusion. Understanding intra-aneurysmal pressure and flow changes that occur with treatment may aid in clinical decision-making, predicting outcomes, and guidance of future research for improvement in long-term aneurysm treatment stability.

At the BIDMC Brain Aneurysm Institute we set out to investigate intra-aneurysmal pressure changes throughout treatment with endovascular devices using a novel 3D-printed, physiologic benchtop aneurysm model with continuous real-time monitoring of inflow, outflow, and aneurysm dome pressures in collaboration with Dr. Timothy Becker and his Bioengineering Devices Lab (BDL) at Northern Arizona University. The objective was to determine if the packing of the aneurysm with coils or flow disruption from FD had an influence on intra-aneurysmal pressure.

Model and Flow System Setup

The aneurysm models were designed in SolidWorks based on physiologic aneurysm measurements (medium: 7mm dome, 3mm neck, large: 10mm dome, 4mm neck) with pressure taps built directly into the aneurysm dome and parent artery from which the aneurysm arises (Figure 1A). The model was then 3D-printed with two different UV-cured materials using PolyJet printing. The vessel and aneurysm were printed with soft Agilus30-Clear polymer, which was previously validated against human tissue to ensure the model maintained tissue-like mechanical properties.[2] The pressure taps and support frame were printed with rigid Vero-Cyan material (Figure 1B). The

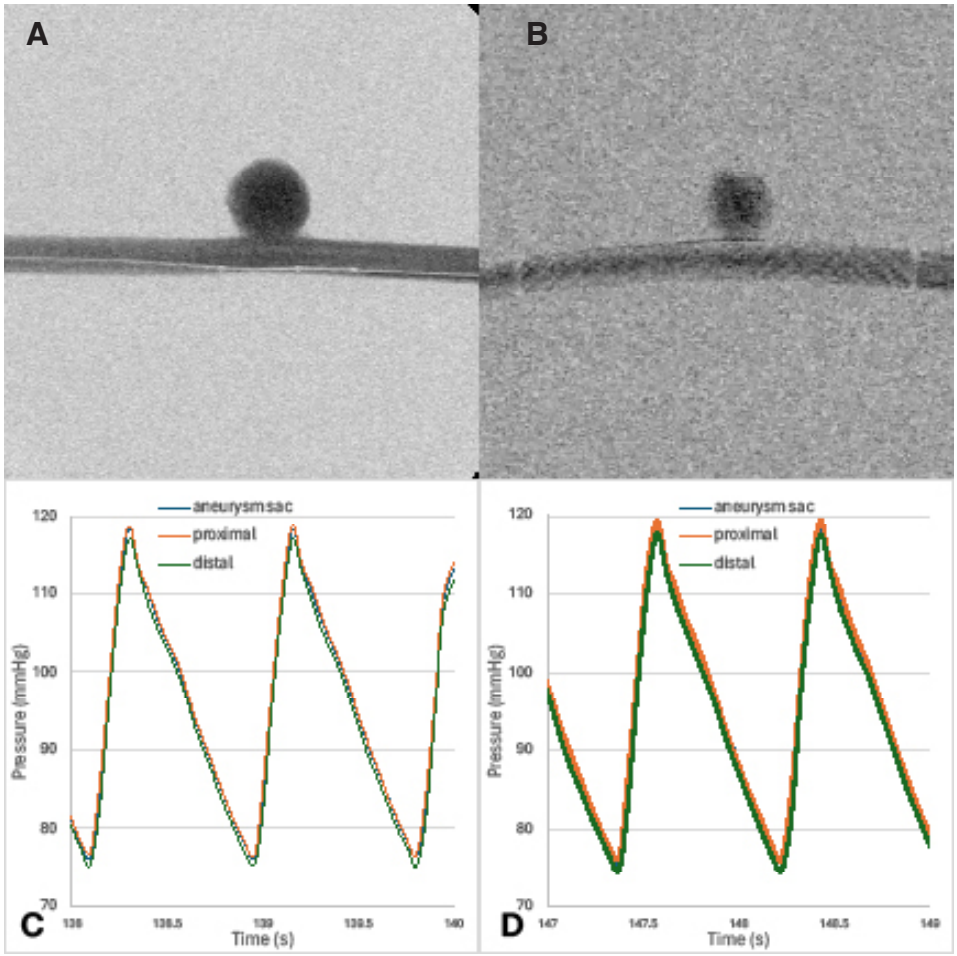

Figure 2. Test 7, (A) DSA of 7 mm aneurysm pre-treatment, (B) post-flow diverter placement DSA with slight reduction in intra-aneurysmal flow, (C) two (2) second sample of pre-treatment pressures, (D) no significant change in post-treatment pressures

aneurysm models were connected with the BDL benchtop flow system (Figure 1C) which consisted of a pulsatile pump capable of maintaining physiologic flow rate and both systolic and diastolic pressure (110 ± 5 ml/min and 118/78 ± 10 mmHg) using a viscosity-matched blood analog fluid (hydroxypropyl-methyl cellulose, ethanol, DI water). Pressure transducers were connected directly to the taps on the proximal and distal parent artery, and aneurysm dome. Real-time data was recorded in LabVIEW continuously before, during, and immediately after coiling (Axium Prime, Medtronic), stenting (Neuroform Atlas, Stryker), FD (Pipeline Flex with Shield Technology, Medtronic), and temporary balloon occlusion (Scepter C/XC, Microvention).

Test Scenarios and Results

To adequately assess aneurysm pressure, and based on the devices available, we ran a total of nine different test scenarios with real-time pressure monitoring. The summary of results is shown in Table 1. Pressure transducers were calibrated and zeroed prior to running each test. Tests 1 and 2 were negative controls to represent untreated aneurysms with the 7mm and 10mm models, respectively. The pressure within the aneurysm in both negative controls remained constant throughout the two tests, as expected. Tests 3 and 4 were positive controls with sealed aneurysms to represent a “healed” neck. Both positive controls resulted in arrest of transmission of parent artery pressure to the aneurysm dome, with the aneurysm transducer measuring approximately 0 mmHg.

In Test 5, coiling of the medium sized aneurysm was performed with stent assistance. After stent placement, coiling was performed to achieve physiologic packing density of over 20%.

No significant changes in aneurysm pressure were observed after placement of the stent or final packing with coils. Digital subtraction angiography (DSA) post-treatment showed cessation of intra-aneurysmal flow. Test 6 consisted of coiling in the large aneurysm, with coils alone placed up to a packing density of greater than 20%. DSA confirmed full aneurysm occlusion with the coils, and again there were no significant pressure changes observed during and after treatment.

Test 7 had the medium aneurysm treated with a single flow diverting stent deployed across the neck (Figure 2). Post-treatment DSA revealed slight reduction in intra-aneurysmal flow, but with residual flow stagnation (Figure 2B). There was no significant change in post-treatment aneurysm pressure (Figure 2D). In Test 8, three consecutive flow diverters were placed concentrically across the neck of the large aneurysm. DSA showed significant reduction in intra-aneurysmal flow after the second flow diverter, and near-complete flow disruption after placement of the third. Continuous monitoring again revealed no significant change in aneurysm pressure after placement of the first, second, and third flow diverter. Finally, in Test 9 we tested temporary balloon occlusion across the neck of the large aneurysm. Two balloons were consecutively inflated and then deflated. Both balloons exhibited dampening and reduction of intra-aneurysmal pressure. The pressure became static and approached the average distal pressure of the parent vessel which was reduced by 14%. This reduction was due to collateral flow bypass maintaining some distal blood pressure. DSA confirmed complete obstruction of flow in the parent artery, and pressure data confirmed the restoration of pre-treatment intra-aneurysmal pressure and distal pressure when the balloon was deflated.

In summary, the treatment conditions (Tests 5-7) consisting of coiling (with and without stent assistance) and FD (single and multiple) in both medium and large aneurysm models displayed no significant changes in aneurysmal pressure post-treatment despite visible alterations in intra-aneurysmal flow on DSA. The only treatment condition (Test 9) that reduced aneurysm and distal artery pressure was the balloon occlusion, which acted in a temporary fashion similarly to the positive control condition.

Alterations in Flow Versus Pressure

Our results suggest that post-treatment aneurysm occlusion may be driven by changes in flow, rather than pressure, inside the aneurysm. Coiling and flow diversion reduced aneurysmal filling, but parent artery pressure was still transmitted to the dome of the aneurysm. Balloon occlusion resulted in a significant pressure drop, which is useful when acutely stabilizing a ruptured aneurysm, however the balloon itself does not provide permanent aneurysm occlusion. Balloon occlusion is typically followed by coil placement, which as we have shown results in alteration of intra-aneurysmal flow, not pressure.

Flow is an important parameter in aneurysm occlusion, as flow stagnation promotes thrombosis within the aneurysm which isolates it from the parent artery. As flow within the aneurysm decreases, the risk of rupture is reduced secondary to reduced shearing forces within the aneurysm.[3] Coil packing causes mechanical obstruction of aneurysmal flow, scaling with increasing packing density. On the other hand, FD diminishes flow through changes in impedance, which is dependent on the number of devices, porosity, and ingrowth of endothelial cells for long-term occlusion.[4,5]

A few previous experimental reports have also indicated that pressure within the aneurysm remains unchanged during and after coiling, with one utilizing a microcatheter tip at the fundus of the aneurysm sac rather than continuous monitoring from the transducer incorporated in the dome as in our experiment.[6,7] Few studies have measured intra-aneurysmal pressure in patients during FD treatment, and these studies have found no significant difference in pressure before and after stent deployment.[8,9] However, challenges remain with patient sample size and pressure measurement reliability in the in vivo setting. Computational fluid dynamic (CFD) analysis can provide insight into post-treatment intra-aneurysmal hemodynamics but lacks physical measurement validation and relies on virtual simulations.[10] The use of carefully controlled realistic in vitro models as in our experiment may provide strong complementary data that addresses an unmet need.

Limitations and Future Directions

While engineering efforts were made to ensure physiologic accuracy of our model and flow system, the biomechanical properties of living tissue may not be fully replicated. This is particularly important when considering pathological states and anatomic variation due to atherosclerosis, tortuosity, calcification, and blood coagulation. This work lays the initial groundwork for future analyses, including Particle Image Velocimetry (PIV) and further experiments that incorporate residence time and the use of vorticity-induced activation of blood components. Future experiments will be directed to quantify flow, shearing forces and oscillatory changes within the aneurysm and downstream vasculature. These variables are essential for understanding the clinical significance of long-term aneurysm occlusion, extending beyond the absence of initial pressure drop post-treatment that was demonstrated in this study.

Treatment modalities tested, including pre- and post-treatment pressures					
Test	**Aneurysm Size (mm)**	**Aneurysm Treatment**	**Packing density %**	**Pre-Pressure Aneurysm (mmHg)**	**Post-Pressure Aneurysm (mmHg)**
1	7	Negative control	NA	119/78	119/78
2	10	Negative control	NA	112/73	112/73
3	7	Positive Control: sealed neck	NA	125/84	-2
4	10	Positive Control: Filled dome, sealed neck	100%	121/80	1
5	7	Aneurysm coiling + Stent placement	30%	118/79	118/79
6	10	Aneurysm coiling	24%	117/77	117/77
7	7	Flow Diverter	NA	111/73	111/73
8	10	3 Flow Diverters	NA	112/73	108/72
9	10	Temporary balloon occlusion	NA	118/78	83/83

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Atrial Fibrillation And Ischemic Stroke

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Atrial fibrillation(AF) is a common cause of acute ischemic stroke and accounts for approximately 25% of such strokes in the United States and other developed countries (1). Strokes caused by AF have a less favorable outcome as compared to ischemic strokes from other etiologies. In ischemic stroke patients with a history of AF and an infarct pattern on imaging consistent with an embolic stroke mechanism, the stroke can very likely be attributed to AF. However, if there is stenosis in a neck or intracranial vessel supplying the region of infarction, then the cause of the stroke is less certain. In patients with a lacunar infarction clinically and on imaging with a history of AF, the likely mechanism of the stroke is small vessel disease and not an embolus from the heart related to AF. Determining that AF was the likely cause for the stroke is important because for secondary stroke prevention anticoagulation with one of the newer direct oral anticoagulants (DOACS), apixaban, dabigatran or rivaroxaban should be initiated unless the risk of major bleeding or frequent falls precludes their use (2). Warfarin is used infrequently because of its higher risk for major bleeding side effects.

AF can be persistent or intermittent. The risk of stroke with persistent AF is significantly greater than with intermittent AF. With the availability of external cardiac monitors, implantable loop recorders (ILRs) or other cardiac devices such implanted defibrillators prolonged monitoring for AF can be performed in stroke patients in whom the cause of their stroke remains uncertain after the initial workup (3). For patients with intermittent AF, the percentage of time they have AF divided by the overall monitoring time can be used to ascertain the burden of AF. Increasing evidence suggests that the higher the AF burden, the greater the stroke risk (4). For patients with very brief AF episodes (<6 minutes) and a low burden the likelihood that AF caused their stroke is low. Typically, in ischemic stroke patients in whom there is no obvious vascular explanation for their stroke a cardiac source is looked for. A structural cardiac source such as a valvular abnormality, cardiomyopathy or dyskinetic cardiac wall segment is identified by transthoracic echocardiography, supplemented in some cases by transesophageal echocardiography. A cardiac rhythm abnormality, AF is the most common cardiac source for ischemic stroke. In ischemic stroke patients without a prior history of AF, the search for it commences in the hospital with prolonged ECG monitoring. If AF is not detected during the hospitalization, an external cardiac monitor can be ordered for up to 30-days at discharge or when the patient returns for follow up. If AF is still not detected and an embolic stroke without an identified source remains a concern, an ILR can be placed that will continue to monitor the patient for several years. Intermittent AF may then be detected in approximately 30% of patients over 3 years of monitoring (5). The AF burden will however be quite variable during that time period among individual patients.

AF detected after an ischemic stroke, termed AFDAS, has a lower risk of recurrent stroke than AF that was known prior to the stroke (6). Trying to detect AF after stroke is still appropriate because in many cases the stroke can be attributed to AF and anticoagulation initiated. However, in cases where the episodes of intermittent AF are brief and the overall burden of AF is low, the cause of the stroke cannot clearly be attributed to AF and the need for anticoagulation is unclear.

The best approach to reduce the risk of recurrent stroke when AF is the presumed cause is clearly anticoagulation with one of the DOACs (1). In patients with brief AF episodes and a low AF burden, the results of randomized clinical trials comparing anticoagulation and antiplatelet therapy are needed to make treatment decisions. In patients with concomitant AF-related stroke and underlying coronary artery disease (CAD) who typically are taking an antiplatelet drug, the combination is problematic because it is associated with a significantly higher risk of major bleeding side effects than either therapy alone. Several recent clinical trials demonstrated that in patients with stable CAD such as patients with the remote insertion of a coronary stent, a DOAC alone is as effective as the combination in preventing recurrent coronary ischemic events and has a significantly lower risk of major bleeding side effects (7). Based on this data, such patients should probably only receive a DOAC for prevention of both a recurrent stroke and coronary ischemia.

In conclusion, AF is a common source for acute ischemic stroke and when this association is determined, patients should be treated with a DOAC to reduce the risk of recurrent stroke. However, when the relationship of AF to the ischemic stroke is unclear such as in patients with brief intermittent AF detected after a stroke or a low AF burden, the need for a DOAC is less certain and the results of clinical trials are needed to determine if anticoagulation is the best approach to secondary stroke prevention.

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