Red cerebral veins and the cerebral steal syndrome

Evidence from fluorescein angiography and microregional blood flow by radioisotopes during excision of an angioma

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The circulation through an arteriovenous malformation and the microcirculation of the surrounding brain were examined during craniotomy by fluorescein angiography and by microregional blood flow measurements using xenon\(^{133}\) and Neohydrin-Hg\(^{197}\). The evidence indicates that occluding the arterial supply to the angioma obliterated the cerebral steal by reducing the shunt or non-nutritional flow and improving the perfusion flow through the cortical microcirculation. The features characterizing the cerebral steal syndrome are described.

**KEY WORDS**
cerebral microcirculation · fluorescein angiography · arteriovenous malformation · microregional blood flow · xenon · Neohydrin-Hg · cerebral steal · red cerebral veins

**T**his report concerns the changes in speed, pattern, and volume of blood flow in the epicerebral vessels and cortical microcirculation noted before and after closure of the arterial supply to an arteriovenous malformation during its surgical removal. The redistribution of flow was displayed by fluorescein angiography of the brain.\(^{17,18}\) The conversion of shunt flow to perfusion flow was quantitated from transit times and clearance curves of intracarotid radioactive tracers which were monitored from the brain surface by miniature gamma-sensitive probes.\(^{11,12,20}\)

The evidence from this examination of the circulatory changes under direct vision during surgery supports the concept suggested by previous authors\(^{1,2,5,21,38,40,46,47,51,53,64}\) that excessive blood flow is diverted through an arteriovenous malformation at some expense to the circulation of the surrounding brain. We have referred to this feature of intracranial arteriovenous shunts as "cerebral steal,"\(^{10,13,16,19,20}\) in analogy to the term "subclavian steal" applied earlier\(^8\) to the vertebral-subclavian artery flow diversion demonstrated by Reivich and others.\(^{15}\)

In the case reported below obliteration of the shunt flow of the cerebral steal has been documented as one of the beneficial effects offered by satisfactory surgical excision of these malformations.
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Case Report

A 47-year-old man had had generalized seizures for 8 years, which were poorly controlled by medication. He complained also of impaired memory.

Examination

Neurological examination and x-ray examination of the skull were normal. The electroencephalogram showed bilateral epileptiform abnormalities from the mesial temporal regions, maximal on the right side. The contour brain scan soon after injection of Neohydrin-Hg\textsuperscript{197} showed over the right temporal region an uptake of 12\% that was absent 6 hours later.\textsuperscript{11} Angiography showed an arteriovenous malformation in the right temporal region supplied almost entirely by two large temporal branches of the middle cerebral artery (Fig. 1). The vertebral circulation gave no supply to the angioma. A large T-shaped temporal vein and many tortuous surface veins over the right hemisphere filled rapidly (Fig. 2). The arteries of the right hemisphere filled poorly (Fig. 3 left). The anterior cerebral arteries on the right were in fact filled better from a left carotid injection, which also displayed filling of the angioma by cross-over flow.

Operative Studies

On March 21, 1969, right temporal craniotomy showed an angioma about 3 cm long which appeared as a closely packed mesh of small- to medium-sized tortuous vessels in the first and second temporal gyri. The large vein 8 mm wide drained the angioma into six epicerebral veins filled with red arterial blood. In and around the angioma, arteries could not be distinguished from veins. A fine polyethylene catheter had been selectively placed in the internal carotid artery by the Seldinger guide technique, and the position

![Fig. 1. Angiogram (1.5 sec) showing early filling of abnormally large veins over the hemisphere. Note decreased arterial filling.](image1)

![Fig. 2. Left: Angiograms (2.5 sec) showing widespread filling of tortuous surface veins entering the longitudinal sinus. Right: Angiogram (0.5 sec) showing that the enlarged middle cerebral artery is supplying branches to the angioma in the Sylvian region.](image2)
of the catheter checked with 2 ml of Coomassie blue dye.* As previously described\textsuperscript{12,22} this produces a flush in the cutaneous territory of the ophthalmic artery over the forehead, upper eyelid, and side of the nose. The catheter was irrigated intermittently during surgery with a dilute solution of heparin in normal saline (25 mg in 200 ml). (This solution can also be delivered continuously by slow drip from a plastic container wrapped by a pressure bag.) By this injection of 2 ml of Coomassie blue into the internal carotid catheter, two main arteries to the angioma were identified (Fig. 4 left). As shown on one of the rapid sequence photographs, the dye could be seen in the feeding vessels before it bypassed to the draining veins. These arteries were then exposed by dissection in the Sylvian fissure where they branched from the middle cerebral artery. When both arteries were closed, the blood in the large draining veins turned from bright red to reddish blue and the distended abnormal veins collapsed.

The effect of this arterial occlusion on the cerebral circulation was then examined by three procedures: fluorescein angiography of the brain, measurement of focal cerebral

* Coomassie blue has since become unavailable from the supplier, and we use Evans blue or Cardio green for this purpose.
blood flow by clearance of xenon and measurement of focal cerebral circulation transit time by the use of Neohydrin-Hg.

**Fluorescein Angiography of the Brain (Technique).** Comparison of the epicerebral and cortical microcirculation changes before and after the arterial clippings were defined with fluorescein angiography. Our earlier technique was used with several modifications. A Kodak-Wratten 47A filter was placed in front of a stroboscopic speed light which had a flash duration of 115 msec. This filter transmitted wavelengths around 460 nm and a small percentage of the red part of the spectrum. A Kodak-Wratten 21 orange and 2-B filters were used in front of the camera lens. A small amount of light in the red band got through to the film, so that a picture of the bloodstreams within the cerebral vessels was obtained. A Nikon motor-driven camera was used, which shot up to 5 frames per sec on Kodak High-Speed Ektachrome color film. Forced development of this film extended the rating to about ASA 1280. An interval timer consisted of a Cadmium photocell discharging with each flash of the strobe light that was coupled to the camera shutter. This registered a signal on a Sanborn recorder with fast-moving paper. The time interval between photographs was determined from the calibrated distance between the signal peaks. Thus, serial pictures were taken rapidly during the arterial phase and slowly during the venous phase of the angiogram. Ciné filming of the angioma was made with similar activating and absorbing filters and an Arriflex movie camera. Magnification of photographs was calculated from strips of flexible transparent membrane squared to a known scale and placed on the curved surface of the brain over the vascular landmarks.

**Fluorescein Angiography of the Brain (Results).** Fluorescein angiography gave a more striking display than Coomassie blue of the shunting blood flow through the angioma. The early phases after the fluorescein injection showed flow into the venous side of the circulation that was well advanced at 1.6 sec (Fig. 4 right). But at this time no flow was visible in the cortical microcirculation and only a few of the ascending branches of the middle cerebral artery were filled. Thus, the shunt flow at this stage was not contributing to perfusion of the microvascular bed of the cortex.

At 2.2 sec (Fig. 5 left), there was evidently both shunt flow and perfusion flow. The fluorescein had advanced further into the large tortuous surface veins with mixing nonlaminar flow and spiral streaming of the dye, both mural and axial, within these venous channels. There was also extensive filling of the cortical circulation within the middle cerebral distribution. Many of the ascending and the posterior branches of the middle cerebral arteries were filled with fluorescein, and much of the middle cerebral territory was visualized. When this was compared to a similarly timed photograph at 2.18 sec after occlusion of the arterial feeders (Fig. 5 right) the change was clearly evi-

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**FIG. 5.** Left: Fluorescein angiogram (2.2 sec) showing that there is mixing flow in the large surface veins, early arterial filling and some flow into the microcirculation of the cortex only in the middle cerebral artery distribution. Right: Fluorescein angiogram (2.18 sec) after clipping of the arterial supply to the angioma. Note absence of shunt flow into the abnormal veins, less filling at this stage of the arteries and cortical microcirculation, but early filling of the "watershed" area supplied by the anterior cerebral artery.
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Fig. 6. Left: Fluorescein angiogram (3.3 sec) before clipping of the arterial supply to the angioma. The angioma has cleared, there is mixing flow in the large surface veins, but the superior margin of the hemisphere fails to fill. Right: Fluorescein angiogram (3.8 sec) after clipping of the arterial supply to the angioma. The shunt flow into the veins is absent, and the superior margin of the cortex is densely filled. The area of nonfilling in the parietal lobe appears to be in the distal territory of the posterior cerebral artery.

dent. There was filling of the arteries of the middle cerebral group and some that appeared near the midline in the "watershed" area from the anterior cerebral artery. There was filling of the cortical microcirculation, although over a much smaller area at this particular time after the injection. The most obvious feature, however, was the lack of shunt flow into the large surface veins, which now remained dark and collapsed.

Later at 3.3 sec (Fig. 6 left), before the arteries had been occluded, there was good filling of the microcirculation of the surface of the hemisphere with the important exception that the cortex along the midline did not fill. This area of supply from the anterior cerebral artery was, however, well filled after the arterial clipping, as seen in Fig. 6 right at 3.28 sec. This change in the microcirculation might have been inferred from the x-ray angiogram where the anterior cerebral artery showed poor filling before but better filling after operation (Fig. 3). The veins again showed a contrast with fluorescein streaming into the large surface veins toward the midline before the arterial clipping. After clipping, these veins had not yet begun to fill from the capillary bed. In later phases, the mixing flow patterns reverted to a laminar flow in the ascending surface veins.

Before the arterial clipping, compression of the large vein draining the angioma during the fluorescein angiogram produced a more dense cortical capillary phase and a slower venous clearance time, although the change was not as striking as that after the arterial closure. This no doubt provided some reduction of the shunt flow by decreasing the venous outlet from the angioma.

These anatomical views of the abnormal shunt flow through the angioma and its draining veins relative to the cortical microcirculation perfusion flow were then supplemented for accurate times and measurement by studies of the perfusion flow and transit time carried out by the radioisotopic tracers.

Focal Cerebral Blood Flow by Xenon\textsuperscript{133} Clearance (Technique). Four scintillation probes\textsuperscript{*} with appropriate rate meters and magnetic tape system were used to monitor the passage of the radioisotopic tracers through the brain. \textsuperscript{12} The probes on phantom studies were found to record gamma counts mainly from a volume of brain tissue about 2 cm in diameter. The small size of the detector crystals (3 × 7 mm), the high ratio of blood flow in gray as compared to white matter (about 6 to 1), the inverse square law affecting the gamma radiation with increasing distance from the probe, and the fact that arteries and veins with radioisotopic tracer flowing through them acted more like a point source than an extended source of gamma emission, were all factors tending to enhance the localization characteristics of the detector system.\textsuperscript{18}

The surface of the brain was covered with

\* Manufactured by Nuclear Chicago, 2000 Nuclear Drive, Des Plains, Illinois 60018
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a thin plastic film upon which the probes were placed in close contact with the underlying cortex at four sites: 1) near the large T-shaped vein draining the angioma, 2) near one of the tortuous veins on the inferior frontal cortex, 3) on the anterior temporal region over the angioma, and 4) on the parietal cortex (Fig. 7).

The probes were positioned in a holder during which perfusion flow was measured after injection of xenon$^{133}$ in normal saline (2 mCi in 2 ml) into the carotid catheter. The 10-min clearance curves were used to calculate perfusion flow using Zierler’s formula.$^{25,58,59,60}$

**Focal Cerebral Blood Flow by Xenon$^{133}$ Clearance (Results).** Before closure of the arteries, the xenon curves showed high “shunt” peaks, indicating short-circuit flow. Similar but lower peaks can normally be observed over the main cerebral arteries.$^{12,20}$ Although the largest peak was recorded over the angioma, a high peak was also noted from just behind the angioma over the large draining vein. Sharp but lower peak counts were obtained from both the frontal and parietal regions. The shunt flow therefore involved a wide region of the right hemisphere (Fig. 8).

After clips closed off the arterial feeders, the profile of the curves changed dramatically with the shunt peaks now being absent and the rate of perfusion flow being increased from all four regions. For the postclipping measurement, 5 mCi in 5 ml of xenon$^{133}$ were injected with the same probe positions. The pCO$_2$ had dropped from 30.5 to 23.0, and the pO$_2$ from 175 to 141. The recorded temperature, pH, and blood pressure remained the same as for the preclipping condition. Thus, the increase in flow values occurred despite a lower arterial pCO$_2$, which would have the effect ordinarily of reducing regional flow (Fig. 9).

**Cerebral Circulation Transit Time by Extracranial Recording (Technique).** Preoperative circulation studies were made by two gamma detecting scintillation probes placed over each side of the forehead so that counts were recorded separately from each cerebral hemisphere.$^{57}$ Neohydryn-Hg$^{197}$ was injected into the antecubital vein with a blood pres-
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sure cuff on the arm. The isotope was delivered into the lumen of the vein while the cuff was inflated above venous pressure, then quickly released to provide a more compact intravenous bolus of the tracer. The curve on the right side of the head showed a peak 1.5 sec earlier, a peak count 68% higher, and an inflow slope more than 200% more rapid than the normal left side (Fig. 10). Two months after operation the curves from either hemisphere were similar.

**Cerebral Circulation Transit Time by Intracranial Recording (Technique).** With the miniature probes at the same sites on the surface of the brain as for the xenon studies, Neohydrin-Hg (1.9 ml containing 190 µCi) was injected rapidly into the internal carotid arterial catheter. These curves were compared with a similar volume and dosage injected with the same probe positions after the arterial occlusions.

**Cerebral Circulation Transit Time by Intracranial Recording (Results).** The transit curves were analyzed by the height-area method to give a blood flow rate. The following points were noted (Fig. 11). The probes over the draining vein and the angioma gave the highest count rate, the highest

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**Fig. 8.** Perfusion clearance curves from intracarotid xenon\(^{133}\). Note absence of "shunt" peaks after the arterial clipping and increase of the perfusion flow rates.

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**Fig. 9.** Xenon\(^{133}\) clearance studies. Comparison of the perfusion flow values calculated before and after arterial clipping.

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**Fig. 10.** Xenon clearance curves. Comparison of the perfusion flow values calculated before and after arterial clipping.
count peak (30,300 and 24,600 per min), and showed the most rapid rise to a peak count rate, which occurred at 1.2 sec, and over the angioma itself at 0.9 and 1.5 sec as a double peak. After the arterial closure, these curves reverted to a more normal profile, although over the angioma itself there was still an earlier peak at 0.9 sec.

The probe near the red vein in the anterior frontal region gave a count rate less than
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Fig. 12. Comparison of cerebral blood flow rates showing reduced bypass flow after arterial clipping, as calculated from transit time curves measured from the brain surface (as shown in Fig. 11).

the two previous probes but still higher than normal at 10,500 counts per min. Peak time was early at 2.3 sec. After the arterial closure, the single peak was replaced by the more usual double peak of cortical and venous curves with a prolonged venous washout slope.

From the parietal cortex the peak times were less abnormal although the venous peak was early, and after clipping the transit curve again had a more prolonged venous washout time.

The shunt flow was thus reduced to similar levels at all four regions, with reduction being greatest through the angiomna itself (Fig. 12). Another significant feature was the obliteration of the shunt flow peaks after the arterial clipping so that the profiles of the curves returned closer to normal with the typical arterial and venous peaks. The lengthening of the venous washout curves was consistent with the longer perfusion time. The 4-sec transit time in the presence of arteriovenous shunting was replaced by a total transit time of 10 sec in the parietal area, 12 sec in the frontal area, and 15 sec in the two temporal areas near the angioma. Allowance is made in estimating these times for the higher background activity produced by the first injection of Neohydrin-Hg197.

The transit time studies thus compliment the xenon perfusion values in demonstrating the increase of microcirculation perfusion following the reduction of the arteriovenous shunt.

Excision

After examining the effect of the arterial occlusion on the circulation, the two feeding arteries were permanently clipped and divided. With the collapse of the vessels of the angiomna and of the large draining veins, the removal was readily made, being extended to include the mesial temporal region which had given electographic abnormality.

Postoperative Course

The postoperative x-ray angiogram confirmed the complete removal of the malformation. The branches of the anterior cerebral artery of the right hemisphere filled more completely than before (Fig. 3 right). The patient was maintained on Dilantin 100 mg three times a day and phenobarbital 60 mg at night. In the second month after operation, the patient had one episode which may have been a seizure when he drank a large amount of beer. The electroencephalogram 4 months after operation showed no epileptiform abnormality. He still had pain and stiffness of the right hand secondary to occlusion of the right brachial artery following vertebral angiography. His neurological examination otherwise was normal. When last seen 2 years after operation, the patient reported that his seizures had been reduced from the preoperative frequency of one-to-three per month to about the same number in a period of 3 months, with these being nocturnal attacks.
temporal electrographic abnormality has not been present since operation.

Discussion

Red Cerebral Veins

Red arterial blood in the veins that drain vascular malformations of the brain have long been familiar to neurosurgeons. Cush- ing and Bailey,5 Dandy,7 and a number of later authors11,31,54 referred to veins filled with blood of an arterial color, often pulsatile, and under increased pressure. The rapid arteriovenous shunt flow through these lesions has been well described in arteriographic studies and the structural basis for this examined in pathophysiological speci- mens.21,24,26,30,38,40-42,46

This venous hyperoxia may be localized with small angiomas or, as in the present example, appear to involve the veins over the entire hemisphere. In drawing attention previously to the significance of red veins occurring with a variety of cerebral lesions or pathophysiological changes, we separated out those veins related to structural shunts as found in arteriovenous malformations and vascular tumors, from others related to more complex metabolic and microcirculatory changes.13 Under this latter category we included the red veins related to the post-ictal state of the cerebral cortex, which Penfield13,44 referred to as “reactive hyperemia,” a term widely used for post-occlusive effects on the peripheral circulation (see Krogh31). Reactive hyperemia in the brain was produced by Penfield and Erikson44 in experimental animals either after asphyxia or by occlusion and release of large arteries. Red veins have also been observed in conditions where the oxygen uptake of the tissue might be expected to be decreased,13,19,20 for example, in relation to reduced metabolism (as in hypothermia), reduced tissue volume (as in a scar or cyst), or reduced capillary volume (as in microvascular sludging or embolization). In most of these circumstances, however, as contrasted with reactive hyperemia, the focal cerebral blood flow in the area concerned or in the brain affected as a whole is decreased rather than increased.15 Waltz27 has elaborated further on the occurrence of red veins in experimental ischemia.

Some confusion has arisen from the intro-duction of the term “luxury perfusion”32 stimulated by the observations in man13,43 and in experimental animals56 of bright red venous blood under a variety of circum- stances all having the common feature of increasing the oxygenation of the cerebral or jugular venous blood. It refers mainly to the condition described by the term reactive hyperemia. But in many of the conditions above, where red veins are noted to be present, the perfusion flow is reduced.15,16 In the case of an arteriovenous malformation, the excessive flow is obviously nonperfusion shunt flow.

Further difficulty in the definition of focal changes in cerebral blood flow derives from the fact that some types of lesions, as for example a glioma, are heterogeneous in relation to the features producing red draining veins, since the microcirculation in and around the lesion contains shunt flow, delayed perfusion flow, and severe decrease of flow with ischemia or even infarction.16 The resolution of most extracranial techniques for measuring cerebral blood flow locally is not sufficient to define these contrasting levels of flow rates in the microcirculation in and around the lesion.19,20,27,28

Cerebral Steal Syndrome

The idea that an arteriovenous malforma- tion can interfere with the circulation of the surrounding brain or indeed of the opposite hemisphere has been supported mainly by arteriographic evidence. The concept has led to some colorful terms, such as “a parasite on the circulation,”47 “a thirsty lesion,”71 or, as Murphy37 first expressed it, “a steal” from the brain. He referred particularly to the demonstration by Norl6n,38 among oth- ers,6,31 that the diameters of the arterial feeding vessels were reduced following surgical excision and the remainder of the cere- bral arteries filled more completely.

The excessive shunt flow through the an- gioma estimated by various methods for total cerebral blood flow has ranged from between 1 and 3 liters of blood per min, producing an over-all increase in cerebral blood flow to as high as three times or more the normal volume.2,3,33,34,51,54,55

In some instances, the more convenient application of radioisotopes to the jugular sampling method made it possible to define the reduction in shunt flow following Surgi-
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cal removal of the arteriovenous malformations, so that the method served as a useful index of the completeness of excision. The rapid carotid-brain and brain-jugular time as well as the more rapid transit time over the hemisphere containing the angioma can also be readily measured by isotope methods.

In reference to the red veins occurring with arteriovenous malformations, we suggested that the term "cerebral steal" was a useful one to describe this idea of the shunt flow through the angioma producing some impairment of the circulation in this surrounding normal brain. The changes recorded in our present case lend support to this concept, the proof of which has so far been somewhat elusive. The conversion of the shunt flow to more effective perfusion flow was demonstrated both on the diffusible xenon clearance curve studies as well as by nondiffusible tracer curves obtained from the transit time values of Neohydrin-Hg.

Moreover, the cessation of excess shunt flow into the large surface veins with more adequate filling of the "watershed" areas of the hemisphere are consistent with the same trend.

This single example, although intensively examined, needs to be supported by additional examples of angiomas of different varieties and in different locations in the hemisphere. It is the effect on the microcirculation, however, that has been measurable here, so that the basis for improved perfusion flow has been carried one stage beyond the evidence of pure arterial filling observed on arteriogram films.

The use of the term "cerebral steal" or "intracerebral steal" in relation to changes in focal cerebral blood flow through ischemic lesions is perhaps less appropriate. The major factor in this circumstance, as we have noted elsewhere, is the remarkable change in flow, both retrograde and anterograde, in the collateral vessels leading into the microcirculation of the ischemic cortex. Another feature of the ischemic exclusive lesions is the absence of structural arteriovenous shunts so characteristic of angiomas and vascular tumors.

Conclusions

From the previous literature and from our present findings, the signs of the "cerebral steal" syndrome can be summarized as follows:

1. Rapid brain circulation transit time and short carotid jugular interval, as measured by radioisotopes.
2. Early uptake on the brain scan.
3. High oxygenation of jugular venous blood, increased by hyperventilation.
4. Visualization of the arteriovenous malformation on angiography with early filling of veins and relatively poor filling of normal arteries, as well as cross-over flow from the opposite side.
5. At craniotomy, filling of red veins with turbulent pulsating arterial blood under increased pressure and increased oxygen levels, which, on occlusion of the arterial supply or excision of the malformation, turn blue, collapse, and exhibit more normal laminar flow.
6. High rate of non-nutritional, non-perfusion shunt flow through the malformation and the draining veins.
7. Conversion of shunt flow to perfusion flow after the arterial obliteration of vessels supplying the lesion or excision of the malformation.

Clinical correlates include progressive symptoms and signs (focal seizures, neurological deficit, memory impairment) without clinical evidence of hemorrhage. It is recognized that, in some instances, small subclinical hemorrhages, focal thrombosis, enlargement of the angiomatosus vessels, and increased venous pressure may contribute to this progressive course. The significance of these factors awaits further studies.

Summary

The changes in focal cerebral blood flow before and after obliteration of the arterial inflow to an arteriovenous malformation of the temporal lobe have been observed and measured by fluorescein angiography and by radioisotopic transit-time and clearance-time blood flow values. Pre- and postoperative intravenous injection of radioisotopes is a simple and useful means of assessing differential transit times in the circulation of the two hemispheres taken separately.

The significance of red veins with hyperoxygenated blood in relation to arteriove-
nous malformations has been discussed and differentiated from other conditions in which red venous blood can also be observed. It is important to distinguish between red veins caused by structural shunts, as in arteriovenous malformations, and those associated with metabolic or vasomotor changes either locally or generally in the brain.

The use of the term "cerebral steal" indicates the concept that excessive shunt flow through an arteriovenous malformation occurs at some expense to the circulation of locally or generally in the brain. The features of the "cerebral steal" syndrome in the presence of an arteriovenous malformation have been outlined.

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