Glomus Tumors of the Head and Neck: Anesthetic Considerations

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I. Introduction

The resection of glomus tumors of the head and neck presents potentially life threatening anesthetic challenges and risks. The last review of these lesions in the anesthesia literature was in 1983. (1) Since then the perioperative care of these patients has undergone significant change, reflecting increased understanding of the nature of these lesions. For example, the association of delayed gastric emptying with resection of the tumors has only recently been appreciated. Associated anesthetic risks mandate familiarity with current information, including the nuances of presentation and management.

II. Historical overview

The history of these tumors is a fascinating one. (2) Nests of paraganglia, believed to be ganglia of the tympanic nerve, were first described in 1840 by Valentin. (3, 4) Carotid body tumors were first reported by Von Luschka in 1862. (5) In 1941, Stacy Guild of the Otologic Research Laboratory of Johns Hopkins University reported upon the presence in temporal bone sections of "flattened, ovoid glomus structures. . .for which the name glomus jugularis is proposed." Guild noted that these tumors "arise in the adventitia of the dome of jugular bulb," immediately below and sometimes extending through the floor of the middle ear. The innervation and blood supply were identified as the glossopharyngeal nerve and the ascending pharyngeal artery and the vascular nature of these lesions was appreciated: "Each glomus. . .consists of blood vessels of capillary or pre capillary caliber with numerous epithelioid cells

between vessels...Usually. . .the vessels are the more prominent feature." Guild noted that the histologic appearance of glomus tissue resembles that of the carotid body. (6) In 1945, Rossenwasser recognized a relationship between a destructive tumor in the middle ear and the glomus jugulare body. (7) In 1953, he argued for a distinction between the glomus jugulare tumor proper and the glomus jugulare tympanicus tumor. (7) The staging proposed by Alford and Guilford embodied this dichotomy, with the tympanicum confined to the middle ear and the jugulare involving the jugular bulb and the skull base. (8) Other classifications have been used, (9) but this basic distinction is still recognized today. (10)

III. Glomus tumors as paragangliomas

Glomus tumors are paragangliomas, which are tumors of neural crest origin, outside the adrenal medulla, capable of secreting catecholamines in autonomic ganglia, the Organ of Zuckerkandl, or adjacent to ganglia. (11) Paragangliomas originate in the head and neck from the brachiomeric family of paraganglia, neuroendocrine tissue which lies along the carotid artery, the aorta, the glossopharyngeal nerve, and the middle ear. (3) Glomus tumors are not the most common paragangliomas. Indeed, the great majority of paragangliomas arise from the carotid body. (12, 13) Glomus tumors can co-exist with other paragangliomas, most commonly those arising in aortic or carotid bodies. Whenever a glomus tumor arises, there is a 10-25% chance of a second craniocervical paraganglioma, usually a carotid body tumor. (14)

IV. Characteristics

Glomus tumors are uncommon, accounting for only 0.03% of all neoplasms and 0.6% of head and neck tumors. (15) The glomus tympanicum, however, is the most common tumor of the middle ear. (15) Glomus tumors are sometimes familial, bilateral, and associated with other neoplasms. They can be malignant, although this is rare. (16, 17, 18) Most frequent in middle age females, they also arise in the pediatric population where they present special problems of both diagnosis and treatment. (19)

V. Clinical manifestations

Clinical manifestations depend upon tumor location. These relate most often to middle ear and cranial nerve dysfunction. Pulsatile tinnitus, conductive hearing loss, aural fullness, discharge or bleeding, and a bluish-red mass behind the tympanic membrane are characteristic of the former. Facial paralysis, dysphonia, marked hearing loss, and pain typify the latter. (17) Neurologic symptoms usually begin after aural symptoms. (20)

VI. Treatment options

The controversy concerning surgical versus radiation treatment continues. With small lesions, radiation is advocated. (21, 22, 23, 24) With younger patients and tumors causing bony destruction surgery is recommended. (25, 10) The risk of recurrence is present but the chance for definitive cure along with long-term compensation for cranial nerve deficits have made the surgical option increasingly attractive. (26, 27) Non-operative treatment is considered appropriate in many cases due to the benign course and the extensive nature of surgery at the skull base--with its considerable risks. (17) Support for conservative care comes from a report of a 40 year history prior to therapy (20) and a relatively low rate of recurrence following radiation treatment. (15)

VII. Diagnosis

Diagnostic evaluation following a suggestive otologic examination has four major goals: to determine the size and extent of the disease, including intracranial extension; to detect associated lesions; to define collateral circulation; and to assess major vasculature. (5) These determinations currently rely upon several modalities, including computerized tomography (CT) and three dimentional computerized tomography (3D-CT), magnetic resonance imaging (MRI), angiography, and magnetic resonance angiography. High resolution, contrast enhanced, computed tomography best shows the extent of disease and possible tumor erosion of the skull base. It is especially useful in the differentiation of glomus tympanicum from glomus jugulare tumors. Magnetic resonance imaging demonstrates tumor vascularity and three-dimensional relationships of the tumor to surrounding vascular structures. Gadolinium-enhanced magnetic resonance imaging is useful for the detection of small tumors of the middle ear. (28) Angiography reveals the vascular extent of the tumor, jugular vein patency, and carotid artery involvement, with bilateral evaluation of the carotid bifurcation indicated. (29, 30) Magnetic resonance angiography is sometimes useful in predicting the need for temporary cross-clamp or even permanent sacrifice of the internal carotid artery. Jugular vein phlebography can detect finger-like tumor projections which may extend to the heart or embolize at the time of excision(31) but this technique has become virtually obsolete with the advent and effectiveness of 3D-CT for tumor mapping and surgical planning. (32) Radionucleotide scintigraphy can provide additional functional information. (33) Whatever imaging techniques are used, the risks of recurrence make radiographic follow-up necessary. (34)

VIII. Anesthetic risk

Anesthetic management in patients with glomus tumors is a formidable challenge. This relates to the following important risks associated with their resection:

Catecholamine secretion producing symptoms of a pheochromocytoma: The relationship between glomic and true chromaffin tissue has been noted. Suspicions regarding catecholamine secretion were raised when Terracol, Huppler, and Rosenwasser noted intraoperative blood pressure elevations with the surgical manipulation of some glomus tumors. (35, 36, 7) Duke et. al. first reported that glomus tumors can independently secrete large amounts of norepinephrine and can present clinically in the same way as a pheochromocytoma. (37) This is not surprising given their relationship to neuroectodermal and chromaffin tissue but it is rare; only 1-4% of glomus tumors mimic a pheochromocytoma. (38, 39) Catecholamine levels of three to five times normal are required to produce clinical symptoms and glomus tumors may secrete amounts several times normal and be subclinical. (38)

Serotonin secretion producing a carcinoid-like syndrome: The endocrine capability of glomus tumors is not restricted to catecholamine secretion. It also includes the release of 5-hydroxytryptamine (serotonin), kallikrein, and 5-hydroxytryptophan, a precursor of serotonin and histamine. Although several cases of carcinoid syndrome have been associated with carotid body tumors, (40, 41) carcinoid syndrome caused by glomus tumors was not reported until 1980. (42)

Carcinoid tumors most often originate in the small intestine but can arise from any organ system. (43) A consistent history includes bronchoconstriction, tricuspid regurgitation and / or pulmonic stenosis, abdominal pain and explosive diarrhea resulting in dehydration and electrolyte abnormalities, violent headaches, cutaneous flushing, and hypertension, hepatomegaly, and hyperglycemia. (42, 44)

Intraoperatively, histamine and bradykinin released during surgical manipulation can cause profound hypotension and even shock. (43) Interestingly, bronchoconstriction in this setting is often not responsive to conventional therapy such as corticosteroids. Catecholamines may exacerbate the problem by provoking the release of serotonin and kallikreins. (44) However, the use of inhaled β–agonists does counteract the bronchoconstricting effects of histamine, bradykinin, or serotonin and are likely to be the most effective therapy. Alternatives would be inhaled anticholinergics, such as ipratropium bromide (Atrovent), since both histamine and bradykinin induce bronchoconstriction in response to the stimulation of afferent cholinergic pathways (with the subsequent release of acetylcholine causing bronchoconstriction). (45)

The rationale for these choices merits brief review and discussion. Histamine stimulates afferent nerve fibers and subsequent reflex bronchoconstriction through cholinergic mechanisms. In addition, histamine induces bronchospasm by directly stimulating H1 receptors on airway smooth muscle. Bradykinin has not been shown to directly stimulate airway smooth muscle but when bradykinin is inhaled bronchoconstriction results. The bronchoconstriction is likely mediated through cholinergic reflex pathways. The rationale for using anticholinergics for either histamine or bradykinin induced bronchoconstriction is to block reflex induced cholinergic discharge that would cause bronchoconstriction. However, both histamine and bradykinin also cause the release of bronchoconstricting neuropeptides through local axon reflex loops. These are not inhibited by anticholinergics. Inhaled β–agonists are preferable because they blocks both of these pathways. Serotonin acts similar to bradykinin and histamine in that it too causes bronchoconstriction, through the stimulation of afferent nerve fibers and cholinergic pathways. The bronchoconstrictive effects of serotonin, too, are either attenuated or abolished by β–agonists. (46) , (47) , (45)

Aspiration or airway obstruction following tumor resection: Cranial nerve deficits can either manifest at the time of presentation or occur following tumor resection. Preoperatively, the vagus nerve is most commonly affected, with the glossopharyngeal and hypoglossal nerves also at risk. Intraoperative sacrifice of cranial nerves is necessary in a relatively high percentage of cases involving resection of lesions of the jugular fossa. The vagus nerve fares most poorly with glomus vagale tumors and meningiomas but even with glomus jugulare tumors it is only preserved about half of the time. (48)

Sacrifice of one of the cranial nerves IX, X, XI, or XII is well tolerated both acutely and over the long-term but impaired sensory and motor function, especially of the vagus, does increase the risk of postoperative aspiration, (14) which may occur in nearly 25% of patients. (38) Aspiration in this setting is a consequence of both loss of airway tone and of sensation. The risk of airway obstruction following cranial nerve injury is less than the risk of aspiration but is nonetheless real. Unilateral cord paralysis, which in an adult usually does not result in complete airway obstruction, certainly can produce obstruction when combined with airway edema or laryngeal distortion.

Primary thyroplasty and vocal cord medialization procedures (medialization laryngoplasty with silastic) are specifically indicated either in the event or anticipation of tenth nerve deficit. They medialize the paralyzed vocal fold so that the normal vocal fold can meet it in the midline. This narrows the airway somewhat by changing the position of the paralyzed vocal fold from a paramedian or lateral position to a median one but it can help avoid the need for a tracheostomy while improving glottic competence and thereby improving

coughing, swallowing, and speech. (48) At present, however, tracheostomy is not routinely performed over vocal cord medialization regardless of approach. (49, 50, 38)

Tracheostomy is currently used very infrequently for glomus tumor surgery, usually only for infratemporal fossa surgery involving very large lesions(38) (50) Tracheostomy does not prevent aspiration but does ensure airway patency and, in the event of aspiration, facilitates airway suctioning and care. In addition, it isolates the airway from the skull base. If a patient with a communication between the nose and the brain coughs (even if the communication is small) a pneumocephalus can result. With a cuffed endotracheal tube, aspiration can be controlled quite effectively. (51) Low pressure, high complicance cuffs and frequent external monitoring and control of cuff pressure minimize ischemia of the tracheal wall. (52) , (53) , (54) , (55)

Impaired gastric emptying: Cranial nerve integrity and airway competence are especially important in view of the recent suggestion that patients following glomus tumor removal may be at increased risk for postoperative ileus on the basis of elevated cholecystokinin (CCK) levels. Slowed gastrointestinal motility in patients with partial airway compromise on the basis of cranial nerve injury, especially to the vagus nerve, obviously places them at risk for the aspiration of gastric contents postoperatively. Jackson et. al. observed that the perioperative management of patients undergoing surgical resection of a glomus tumor is associated with gastrointestinal complications such as pancreatitis, cholecystitis, and ileus far exceeding what would be expected. In terms of postoperative ileus, they postulate a fall in CCK (which they found to be approximately twice as great in these patients compared to patients with nonglomus skull base tumors) may be causative: "Elevation of serum gastrin levels is followed by an increase in specific receptors on target cells. If the same holds true for CCK receptors, a reduction in serum CCK to "normal" levels would result in a reduced receptor occupation and (possibly) failure of gastric emptying, diminished pancreatic secretory activity, and lack of gallbladder contraction." (56) In order to minimize the potentially significant risk of postoperative aspiration, prudent postoperative care may entail not only nasogastric suction but also a period of parenteral nutrition.

Increased intracranial pressure: While intracranial extension of these tumors is usually extradural and confined to the posterior fossa, they can extend to the middle fossa. Significant intracranial hypertension with glomus tumors is rare. When it does occur, it is usually due to tumor occlusion of venous sinuses. In the past, the sitting position was frequently utilized to facilitate exposure. The lateral position is most frequently used today to decrease the risk of venous air embolism.

Threat of venous air embolism: Venous air embolism is a risk during many types of surgery.((57) , (58) , (59) , (60) , (61) , (62) , (63,, 64) , (65)) Air embolism has been reported in craniofacial procedures((66)) and head and neck trauma((67)) but the risk during resection of glomus tumors has not been quantitated. This risk is probably heightened with involvement of the jugular vein or need to open the sigmoid sinus, a not infrequent surgical maneuver given the propensity of the tumor for extension. (68) Proximal and distal ligation of the vein and packing of the wound prior to tumor manipulation or resection is mandatory to diminish the risk of air embolism. Lateral positioning intraoperatively is most appropriate, the sitting position being unnecessary and even contraindicated.((51))

Substantial blood loss: The resection of these highly vascular lesions can be associated with substantial blood loss. Blood replacement in one report averaged 3-8 units and length of operation ranged from 8-26 hours. (38) The sigmoid sinus, the inferior petrosal sinus, and the tumor itself are the major sources of bleeding. (48) In one patient in one series, 5000 cc of blood was lost in one hour. (1) In another patient, up to 14 units of blood were transfused. (69) Massive blood loss is of particular concern with intracranial extension requiring opening of the sigmoid sinus or intrajugular extension.

Preoperative embolization diminishes intraoperative blood loss and shortens operative time. (70, 71) Despite these advantages, many surgeons choose not to embolize these tumors because of the risk of associated morbidity: vascular injury, stroke, death. (72) Other strategies to combat significant blood loss during surgery include external carotid and jugular vein ligation and hypotensive anesthesia. (73)

IX. Anesthetic management

Anesthetic management stems directly from preoperative concerns. One of the most important involves excess catecholamine or serotonin secretion. Both, especially catecholamine secretion, can be life threatening. A careful history and physical examination are crucial in addressing this issue. Suggestive symptoms include severe hypertension, sweating, myocardial infarction, and cardiovascular collapse for catecholamine excess (11, 74) and diarrhea, flushing, and headaches for serotonin excess.

The question of when and what to test is an important one. Should serum and urine catecholamine levels be checked in all patients with glomus tumors or only those with symptoms? Levit suggests that all patients with craniocervical paragangliomas should have

serum and urine levels checked. (75) Ghaleb, however, recommends formal laboratory testing only with a suggestive clinical history. (1)

The problem with this later strategy is that undiagnosed pheochromocytomas lead to high perioperative morbidity and mortality. (76, 77, 78) While there are fewer reports of undiagnosed catecholamine secreting glomus tumors, there is every reason to believe perioperative morbidity would be high without proper preoperative preparation and, just as with pheochromocytomas, would sharply diminish with proper preoperative pharmacologic preparation. (79) Further, Jackson, who routinely tests serum catecholamines in patients with glomus tumors regardless of the history and physical examination, has found catecholamine levels as high as eight times normal without clinical symptoms. (51) This suggests the need to preoperatively check serum catecholamine levels in all patients with suspected glomus tumors. The cost for this is ninety-six dollars at the University of Iowa but given the potential perioperative morbidity of an undiscovered catecholamine secreting glomus tumor, the additional expense would seem justified. The biochemical diagnosis of catecholamine secreting tumors has been outlined elsewhere. (79) In terms of serotonin secreting tumors, the number of reported cases is fewer and while the morbidity can be significant the chance of it is very small. Therefore, a routine plasma serotonin level in the absence of clinical symptoms is probably unnecessary.

Significant elevations of urine or serum catecholamines or their metabolites require the same preoperative pharmacologic stabilization in these head and neck tumors as for a pheochromocytoma. There are several agents and protocols for treatment. The most traditional rely upon α and β adrenergic blockade. One recommended regimen involves phenoxybenzamine one to two weeks prior to elective surgery, starting with an initial dose of 10 mg twice a day and gradually increasing to final doses of 40-100 mg / day. Phenoxybenzamine blocks both postsynaptic α-1 receptors and presynaptic α-2 receptors, the latter leading to enhanced catecholamine secretion and possible tachycardia. Prazosin, a selective α-1 antagonist, causes less tachycardia than phenoxybenzamine. It also is shorter acting, resulting in less prolonged postoperative hypotension. Usual initial doses are 1 mg three times a day with final daily doses of 8-12 mg. Labetalol, a combined α and β blocking agent, has been used successfully in the preoperative preparation of pheochromocytoma. B-adrenergic blockade is not required in most cases but if utilized these drugs should never be started prior to the institution of α blockade. Unopposed α-agonism in the setting of β–blockade can lead to severe vasoconstriction and hypertensive crisis and, because the β–blocked heart may not be able to compensate for the increased systemic vascular resistance, myocardial ischemia, infarction,

and failure can occur.(62, 63) Recently, other therapies have been suggested for the preoperative preparation of patients with pheochromocytoma, for example calcium channel blockade and magnesium sulfate. Nicardipine used orally in the preoperative period (60-120 mg / 24 hours) and intravenously intraoperatively, 2.5-7.5 µg / kg / min by infusion, has been used to successfully control pheochromocytomas. (80) Magnesium sulfate in combination with fentanyl has also been used successfully as an adrenergic antagonist intraoperatively at a dose of 40-60 mg / kg intravenously followed by a continuous infusion of 1-2 gm / hr. In some such cases, sodium nitroprusside may be necessary to control hypertension. (81)

Serotonin secreting glomus tumors should be identified if at all possible by clinical history preoperatively. Dehydration or electrolyte abnormalities should be corrected. Somatostatin inhibits the release of serotonin but it has a very short plasma half-life. Octreotide (Sandostatin, Sandoz), a long acting somatostatin analogue with no major side effects, decreases the storage and release of mediators in the perioperative period and a regimen consisting of 100 µg subcutaneously, two to three times daily, should be started two weeks prior to surgery in symptomatic patients. (82) Intraoperatively, histamine and bradykinin released during surgical manipulation can cause bronchospasm, decreased pulmonary compliance, profound hypotension, and even shock. Therapy must be centered upon either blocking the release of chemical modulators or blocking their actions. Again, octreotide is useful, with 100 µg intravenously at induction recommended. (83) Severe hypertension is of particular concern. 5-Hydroxytryptamine may increase vascular response to catecholamines by stimulating the release and inhibiting the uptake of noradrenaline and potentiating post-junctional α-1 adrenoceptor mediated response to both epinephrine and norepinephrine. (84) Ketanserin, a selective antagonist at 5-HT2 receptors, α-1 adrenoceptors, and H1 histamine receptors, may offer the best pharmacologic profile to treat a hypertensive carcinoid-like crisis. An infusion of 5 mg / hr is suggested. (85, 82) Postoperatively, octreotide should be continued for 72 hours and slowly weaned over several days.

Involvement of cranial nerves that may predispose to airway obstruction or aspiration may occur in one of several ways: tumor invasion preoperatively, nerve injury or sacrifice intraoperatively, or tissue edema causing nerve palsy postoperatively. Whatever the mechanism, ninth, tenth, or twelfth cranial nerve dysfunction can predispose to airway compromise--either by aspiration or obstruction. Preoperatively, a careful history and physical examination focused upon the potential problem of airway compromise is required. If the patient has had previous head and neck surgery, an assessment of vocal cord function and position is important. Intraoperatively, the anesthesiologist should be aware of nerve

sacrifice or inadvertent nerve damage. Postoperatively, the dynamic nature of edema around cranial nerves one to three days following the procedure must be appreciated by both the anesthesiologist, surgeon, and nursing staff. Frequent observation for stridor and wheezing is key.

The risk of aspiration caused by gastroparesis is increased in these patients. The findings of Jackson et. al. ((56)) suggest heightened awareness and caution, conservative airway management including a tracheostomy or endotracheal tube, intermittent nasogastric suction, and postoperative total parenteral nutrition.

The possibility of intracranial hypertension must be considered. While rare in these cases, it can be life threatening. Suggestive signs and symptoms should be elicited and the CT scan evaluated for intracranial extension. Elevation in intracranial pressure may be related to obstructive hydrocephalus with ventricular enlargement or to the mass effect of the tumor.

Anesthetic management in the setting of intracranial hypertension secondary to tumor extension should follow the same general principles as for other intracranial mass lesions. (86) , (87) In general, sedative premedicants should be avoided. The relationship of mean arterial pressure to cerebral perfusion pressure emphasizes the importance of an intra-arterial catheter. Induction and maintenance of anesthesia should be tailored to avoid an exacerbation of elevated ICP. While there are many methods to achieve this, one includes spontaneous hyperventilation, thiopental to achieve narcosis and mask hyperventilation followed by neuromuscular relaxation with a long-acting nondepolarizer. The slow administration of fentanyl or sufentanil, laryngotracheal lidocaine followed by an assessment of mean arterial pressure, and volatile anesthetics by mask (if necessary) are important adjuncts to control a hyperdynamic response to laryngoscopy. With intubation achieved and hyperventilation established ($PaCO_2$ 25-30 mm Hg), volatile anesthetics in moderate dose can be used with safety. The administration of mannitol and placement of a lumbar subarachnoid catheter for withdrawal of cerebrospinal fluid are other useful adjuncts in controlling intracranial pressure. Avoidance of dextrose containing intravenous solutions is recommended. (88, 89, 90)

It is important to appreciate the risk of massive blood loss. Besides the standard monitors, an arterial line, Foley catheter, and CVP (or a PA catheter) are suggested. Substantial blood loss can occur rapidly and necessitate transfusion. Volume status should be monitored with a CVP and replaced through a large bore intravenous catheter. Before placement of a central line, tumor involvement of both the internal jugular vein and the superior

vena cava should be assessed. If the internal jugular vein but not the superior vena cava has been invaded by tumor, the contralateral basilic, external or internal jugular vein can be used to gain central access. If both have been invaded, the femoral vein should be used. The importance of central placement mandates chest radiography or fluoroscopy when a basilic vein is used, given the tendency for the catheter tip to progress to a noncentral location, usually in the neck. (1) Two large peripheral intravenous lines should be placed. Blood salvage for transfusion, in the presence of tumor, is not usually done but controlled hypotension with deep inhalation anesthesia and sodium nitroprusside may be useful to decrease blood loss. (91) , (92) , (93) , (94)

Air or tumor embolism is extremely rare in these cases, partly because the patient is most frequently placed in a supine or lateral position with the head secured by either a Mayfield skull clamp or horseshoe head rest. (51) With invasion of the internal jugular vein, tumor embolism is nevertheless a risk. This may explain several reported instances of sudden death and emphasizes the importance of early proximal and distal vein ligation and even cardiopulmonary bypass in such cases. (31, 95) With proximal and distal vein ligation prior to tumor resection, the risk of air or tumor embolism is greatly diminished. (51)

If the carotid artery is to be occluded intraoperatively, some form of assessing the adequacy of cerebral perfusion may be warranted. This may include EEG, somatosensory evoked responses, arterial stump pressure assessments, transcranial Doppler measurement of middle cerebral artery flow velocities, or possibly the direct measurement of CBF. Monitoring for cerebral well-being during carotid occlusion has been discussed in great detail in the medical literature and a recent review of this subject is suggested. (96, 97, 98, 99, 100, 101, 102)

X. Conclusion

Glomus tumors are paragangliomas of the head and neck and they present multiple and challenging anesthetic risks. Clinical manifestations depend upon tumor location but most often relate to middle ear and cranial nerve dysfunction. The treatment trend is increasingly surgical, partly because of the increased effectiveness of preoperative imaging and tumor mapping with the CT and 3D-CT scans, MR imaging, angiography, and magnetic resonance angiography. Anesthetic risk includes catecholamine secretion producing symptoms of a pheochromocytoma, serotonin secretion producing symptoms of carcinoid syndrome, aspiration following tumor resection, impaired gastric emptying, increased intracranial pressure, the threat of venous air embolism, and substantial blood loss. Of the many significant recent developments adding to the understanding of these lesions, the recognition that they may

predispose to gastroparesis and new concepts concerning the testing for and treatment of catecholamine and serotonin excess are perhaps most important to the anesthesiologist.

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