SPECIAL ARTICLE

Effects of Carotid Sinus Reflex on Cardiac Impulse Formation and Conduction

By Arthur J. Linenthal

REFLEXES arising from stimulation of pressor receptors in the carotid sinus produce marked effects on the heart, on the blood pressure, and on the brain. Various aspects of these reflexes have been extensively described by others.1-3 This report is concerned primarily with the effects of carotid sinus stimulation on cardiac impulse formation and conduction in man: the anatomy, physiology, and pharmacology of the reflex, and the clinical significance of these potent cardiac effects.

Anatomy

The carotid sinuses, slight bulbar enlargements of the internal carotid arteries at the bifurcation of the common carotid arteries, contain receptors that are stimulated by stretching of the sinus wall brought about by pressure changes in the vessel.4 Pressor receptors are also present in the walls of the common, internal, and external carotid arteries for a short distance above and below the sinuses. Afferent impulses from the carotid sinus are transmitted to the brain stem over the carotid sinus and glossopharyngeal nerves. Efferent impulses to the heart are transmitted over vagal pathways. Within the heart postganglionic parasympathetic fibers are distributed to the sinoatrial node, the atria, and the atrioventricular node; there appears to be no parasympathetic innervation of the ventricles.

In the brain, impulses may also go to the vasomotor center and to the cerebral vessels.

Physiology

Carotid sinus pressure may cause systemic vasodilatation and cerebral vasoconstriction as well as a variety of cardiac effects. The changes in cardiac impulse formation and conduction are due to simultaneous vagal stimulation and sympathetic inhibition during pressure, and to sympathetic rebound stimulation after pressure is released. As shown by the magnitude of the maximum cardiac effects, the activity of the carotid sinus cardiac reflex varies greatly in different people. In general, the effects are greater in older individuals, particularly those with hypertension or coronary artery disease. The segment of the reflex are responsible for hyperactivity of the reflex has been established in a patient with angina pectoris:4 the increased effect was localized either in the intrinsic cardiac nerves to the atrioventricular node or in the node itself. Less commonly, hyperactivity of the reflex may be associated with lesions of the carotid sinus (such as aneurysm or tumor), or with central nervous system lesions (such as tumor or syphilis).

Studies in our laboratory with a technic of controlled, measured pressure on the carotid sinus have provided a quantitative analysis of various aspects of these effects on cardiac impulse formation and conduction in man. During normal sinus rhythm the most common reflex vagal effect on the heart is a negative chronotropic one, the production of a slower sinoatrial rate, manifested electrocar-
Figs. 1 and 2. (See legends on opposite page.)
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Diagnostically by prolongation of the P-P interval. This effect may occur with either right or left carotid sinus pressure. In different individuals the amount of maximum slowing may vary markedly; it may be so slight as to be detected only by electrocardiographic measurements or it may be manifested by cardiac asystole lasting up to 10 seconds and associated with cerebral ischemia and syncope. When pressure on the carotid sinus is raised instantly to an effective level and is maintained at this level for 3 seconds, the maximum prolongation of the P-P interval is often observed in the first cardiac cycle and routinely occurs by the end of pressure (fig. 1). When pressure is discontinued, the sinoatrial rate returns to its control level within 6 to 7 seconds and may then show a slight, brief acceleration. The time to reach the maximum effect and the time for the effect to disappear do not depend on the magnitude of the slowing or on the sinoatrial rate before pressure is applied.

When constant pressure is applied for 30 seconds or more, the sinoatrial rate often returns to its control level within 10 to 15 seconds after the beginning of pressure. Occasionally some vagal effect may persist for at least several minutes with continued application of pressure.

The relation between the intensity of the pressure and the magnitude of the sinoatrial slowing is shown in figure 2. With only a slight increase in pressure above the threshold, the effect increases quickly to a nearly maximum degree and then tends to level off. The variation in the response among different individuals and between the right and left carotid sinuses in the same individual (O.B.) is apparent in these data.

A second, less common reflex vagal effect on the heart during normal sinus rhythm is interference with atrioventricular conduction, shown in the electrocardiogram by prolonged P-R intervals, nonconducted (dropped) sinoatrial beats, varying degrees of partial heart block, or complete atrioventricular block. Complete heart block is usually followed within a few seconds by the appearance of idioventricular beats; occasionally ventricular standstill may persist long enough to produce symptoms of cerebral anoxia (faintness, dizziness, syncope, convulsions). Many features of this effect on conduction are similar to those of the sinoatrial slowing: it may occur with either right or left carotid sinus pressure, and its appearance and disappearance follow a pattern similar to that of the slowing. Although these 2 effects often occur at the same time, they are quite independent. Prolongation of the P-P interval usually occurs with less intense pressure than is required to prolong the P-R interval. In certain individuals, however, marked prolongation of the P-R interval and even dropped beats may occur with little change in the sinoatrial rate.

Other changes in sinoatrial rhythm produced by carotid sinus pressure are related to its varying action on other cardiac pacemakers as well as on the sinoatrial node. For example, a greater vagal depression of the sinoatrial node as compared with lower pacemakers may permit the escape of an atrioventricular nodal rhythm or an idioventricular rhythm during sinoatrial slowing. After the end of pressure, on the other hand, when the vagal influences have disappeared, rebound increased activity of the previously inhibited sympathetic stimuli may excite the sinoatrial node less than it does other centers, resulting
in extrasystoles or runs of atrial, atrioventricular nodal, or ventricular tachycardia.

Carotid sinus pressure may also markedly affect the heart during various cardiac arrhythmias. The rapid ectopic impulse formation of paroxysmal atrial tachycardia or paroxysmal atrioventricular nodal tachycardia may be stopped abruptly and sinoatrial control may be restored. Ventricular tachycardia, on the other hand, is not affected. During atrioventricular nodal rhythm associated with absent sinoatrial activity, carotid sinus pressure may slow the atrioventricular nodal rate and may lead to an idioventricular rhythm.

In addition to these effects on abnormal types of impulse formation, carotid sinus pressure applied during partial atrioventricular or ventriculoatrial block may cause an increase in the conduction defect. Although reflex vagal stimulation has no effect on the ectopic impulse formation in atrial fibrillation or atrial flutter, it may increase the degree of block and thereby slow the ventricular rate. In contrast to this depressing action of carotid sinus pressure on atrioventricular conduction, in some patients with transient left bundle-branch block reflex vagal stimulation may temporarily restore normal intraventricular conduction without changing the rate. Another effect of carotid sinus pressure on atrioventricular conduction is seen in the production of anomalous atrioventricular conduction (Wolff-Parkinson-White syndrome) in susceptible individuals.

Pharmacology

Various commonly used drugs may influence the cardiac effects of carotid sinus pressure. Vagomimetic drugs such as acetyl-beta-methyl-choline and neostigmine, digitalis glycosides, and veratrum alkaloids may all potentiate these effects. Previously ineffective pressure on the carotid sinus may produce marked cardiac effects when reapplied following the administration of one of these agents. Even sympathomimetic drugs, by their vasoressor action, may stimulate pressor receptors and produce reflex vagal effects on the heart.

On the other hand, vagolytic drugs such as atropine, and sympathomimetic agents such as epinephrine, ephedrine, and phenylephrine may decrease or block completely the carotid sinus cardiac reflex. This blocking effect may not be the same on all parts of the cardiac impulse-forming and conducting system. Atropine, for example, in submaximal doses may interfere with a reflex vagal depression of the atrioventricular node without altering the effect on the sinoatrial node. Under these circumstances carotid sinus pressure which previously produced only slight sinoatrial slowing without a shift in the pacemaker, after atropine may slow the sinus node similarly and also lead to atrioventricular nodal rhythm. Drugs such as epinephrine and ephedrine may have a 2-fold action: they may counteract the depressing effect of reflex vagal stimulation on the sinoatrial node and they may stimulate the more prompt escape of lower pacemakers. In experimental animals quinidine also has been found to block the cardiac effects of direct electric stimulation of the vagus nerves; a similar effect in man probably occurs only with very large doses. Procaine hydrochloride injected into the wall of the carotid sinus or infiltrated around the nerves from the carotid sinus will temporarily block the reflex activity.

Clinical Applications

Carotid sinus pressure is of clinical value principally in the diagnosis and treatment of certain cardiac arrhythmias and in the diagnosis of the carotid sinus syndrome.

Digital pressure on the carotid sinus is performed with the patient either supine or in a semirecumbent position and with the head slightly rotated away from the side to be stimulated. The carotid sinus can often be felt as an expansile mass situated below the angle of the jaw usually at the level of the thyroid cartilage. Frequently it is impossible to feel the actual bulbus enlargement and one must be guided by the point of maximum
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carotid pulsation. The greatest difficulty is experienced in patients with short, thick necks. Pressure is applied toward the vertebral column by rubbing vigorously with several fingers over the carotid sinus and several centimeters above and below. It is important to retract the carotid artery laterally to prevent the vessel from slipping medially. When pressure on either carotid sinus alone is ineffective; both carotid sinuses may be gently pressed simultaneously. In its clinical uses effective carotid sinus pressure need not be applied for more than a few seconds and ineffective pressure should not be applied for more than 10 seconds.

During application of carotid sinus pressure the physician should observe the cardiac activity by listening to the heart beat or by watching the electrocardiogram. Transient dizziness or lightheadedness occasionally occurs when there is no ventricular beat for 2 or 3 seconds. Excessive cardiac effects which may lead to syncope and convulsions must be avoided by stopping the pressure when the first definite effect is observed. Rarely, complete atrioventricular block and ventricular standstill may persist following carotid sinus pressure, especially in patients with Stokes-Adams disease who are subject to spontaneous attacks of ventricular standstill. External stimulation of the heart by slapping the precordium or with an electric pacemaker is useful in such circumstances to terminate the standstill and to resuscitate the patient.

Untoward effects of carotid sinus stimulation most commonly occur when the pressure is prolonged and intense, when it is applied with the patient in the sitting or standing positions, when it is applied to both carotid sinuses simultaneously, or when the patient is elderly. In older patients with generalized arteriosclerosis, carotid sinus pressure of excessive duration with complete occlusion of the carotid artery has been followed by transient or permanent hemiplegia. Syncope and convulsions may also occur in the absence of significant cardiac slowing, as a result of a direct effect on the brain. Pressure on the carotid sinus may also stimulate reflexes to the coronary arteries, to other major vessels, to the bronchial smooth muscle, and to the gastrointestinal tract; resulting manifestations may include precordial pain, hypotensive symptoms, wheezing, and gastrointestinal symptoms.

Diagnosis of Cardiac Arrhythmias. Changes in cardiac impulse conduction as a result of carotid sinus pressure may be helpful in the diagnosis of certain cardiac arrhythmias. The precise electrocardiographic recognition of a supraventricular arrhythmia, for example, may be difficult when the P waves are obscured by rapid ventricular complexes. Carotid sinus pressure, by interfering with atrioventricular conduction, may slow the ventricular rate enough to reveal the electrocardiographic configuration of the supraventricular activity. This effect is most often useful in the diagnosis of atrial flutter with 2:1 block and may also be helpful in paroxysmal atrial tachycardia with 2:1 block. The marked, irregular ventricular slowing which occurs with carotid sinus pressure and disappears at once when pressure is stopped is so characteristic of atrial flutter that it can be used at the bedside to differentiate this arrhythmia from other rapid, regular tachycardias.

Because of its effect on cardiac impulse formation and conduction, carotid sinus pressure may be useful in the analysis of complex arrhythmias. For example, transient slowing of a dominant supraventricular pacemaker may reveal a ventricular parasystolic focus, and prolongation of ventriculoatrial conduction may demonstrate retrograde conduction as the mechanism of coupled beats.

Treatment of Cardiac Arrhythmias. Carotid sinus pressure often terminates paroxysmal atrial tachycardia and paroxysmal atrioventricular nodal tachycardia. This simple procedure can be carried out by the patient himself as well as by the doctor and, if effective, may be extremely valuable when attacks recur frequently and drug prophylaxis is unsuccessful. There is no necessary relation
between preexisting hyperactivity of the carotid sinus reflex and the efficacy of carotid sinus pressure in stopping an episode of tachycardia. The effect usually consists of an immediate restoration of sinoatrial rhythm, occasionally with very slight preliminary slowing of the ectopic pacemaker. Rarely is there significant slowing of the arrhythmia if normal sinus control is not restored. Once normal sinus rhythm returns usually it persists, although occasionally it may be transient and carotid sinus pressure may need to be repeated.

The simplicity and safety of carotid sinus pressure make it the initial procedure of choice to be tried in all cases of paroxysmal supraventricular tachycardia. The absence of an effect does not exclude the possibility of an active response to another means of producing reflex vagal stimulation, such as pressing on the eyeballs, inducing vomiting, breathing deeply, expiring forcibly against the closed glottis after a deep inspiration (Valsalva maneuver), and swallowing a large bolus. Failure of these measures at one time during an episode of tachycardia does not preclude success later in the same attack; accordingly, they should be tried repeatedly before resort to other forms of therapy. If a vagomimetic drug or a digitalis glycoside is administered, previously ineffective reflex maneuvers should be repeated, since they may then be effective.

**Diagnosis of the Carotid Sinus Syndrome.** An occasional person with a hyperactive carotid sinus reflex may develop the carotid sinus syndrome; that is, spontaneous symptoms occur as a result of stimulation of the carotid sinus by such means as a tight collar, pressure from shaving, or turning the head. The symptoms may include dizziness, faintness, paresthesias, syncope, and convulsions; most often they are due to marked cardiac slowing, but they may also result from a drop in blood pressure without change in the heart rate, or from a reflex effect directly on the brain. Treatment may consist of specific drugs such as atropine or ephedrine, surgical denervation of the carotid sinuses, or irradiation of the carotid sinus area.

The diagnosis of this syndrome depends upon repeated, exact reproduction of the spontaneous symptoms by manual pressure on the carotid sinus. A careful analysis of each suspected case is essential, since many individuals who have symptoms from pressure on the carotid sinus do not have spontaneous symptoms, and a hyperactive carotid sinus reflex may be present along with some other disturbance causing cerebral symptoms. Conditions such as epilepsy, hypoglycemia, postural hypotension, and vasovagal syncope must be differentiated.

In patients with Stokes-Adams disease, attacks of syncope due to ventricular standstill may be precipitated by spontaneous stimulation of the carotid sinus. Diagnostic carotid sinus pressure should be applied with great care in such patients and with an external cardiac pacemaker at hand, since, if ventricular standstill occurs, it may last long enough to cause severe cerebral symptoms.

**Diagnosis of Acute Myocardial Infarction during Left Bundle-Branch Block.** The electrocardiographic diagnosis of acute myocardial infarction may be difficult in the presence of left bundle-branch block. In certain individuals with left bundle-branch block carotid sinus pressure may paradoxically restore normal intraventricular conduction. If the block can thus be temporarily abolished, the electrocardiographic changes of acute myocardial infarction may become apparent. Carotid sinus pressure should be applied cautiously in patients with acute myocardial infarction, since the cardio-inhibitory reflex may be quite active.

**Miscellaneous Cardiovascular Effects.** Carotid sinus pressure has been found to be effective occasionally in terminating attacks of angina pectoris. This effect is independent of any action on the heart rate and has been attributed to an interruption of sympathetic reflex arcs or sensory pathways. The relief of pain is not known to be specific for cardiac pain and some patients with angina pectoris
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are not helped. Accordingly, and in view of the hazards involved, carotid sinus stimulation is not considered to be clinically useful in the diagnosis or treatment of angina pectoris.

Carotid sinus pressure has also been found to terminate attacks of acute pulmonary edema in certain hypertensive patients. Prolonged application of pressure is described and this is undoubtedly hazardous. As in the case of anginal attacks it seems likely that this effect is independent of any action on the heart rate.

REFERENCES

BEING TRUE TO ONE'S CONVICTIONS

THOMAS H. HUXLEY

English biologist, 1825-1895

Sit down before fact as a little child, be prepared to give up every prejudiced notion, follow humbly wherever and to whatever abysses nature leads, or you shall learn nothing. I have only begun to learn content and peace of mind since I have resolved at all risks to do this.—Life and Letters of Thomas H. Huxley. From Great Companions. Readings on the Meaning and Conduct of Life from Ancient and Modern Sources.
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