Changes of Ventricular Impulse Formation during Carotid Pressure in Man

By M. Golbey, M.D., C. P. Ladopulos, M.D., F. H. Roth, M.D., and D. Scherf, M.D.

No proof exists that carotid sinus pressure influences ectopic automatic ventricular impulse formation in man. Two patients are described who revealed during carotid pressure slowing of a ventricular parasympathetic focus and a change of the area of impulse formation respectively.

There appears to be general agreement that in the mammalian heart vagal stimulation has no effect on ventricular contractility. This was demonstrated by Drury who used the myocardiograph of Cushny and was confirmed by optical determination of the intraventricular pressure of the artificially driven heart; when a cardiac alternans existed and weak faradic stimulation of the cardiac sympathetic nerves for a few seconds abolished the alternans and strengthened the contractions of the ventricle, stimulation of the vagus nerves with the strongest possible currents was devoid of effect. Maximal stimulation of either vagus or of both simultaneously had no effect on ventricular excitability or conduction velocity of the dog heart.

However, some instances are mentioned in the literature where vagal stimulation apparently has had an effect on ventricular ectopic impulse formation. Thus, a ventricular paroxysmal tachycardia was repeatedly abolished by carotid pressure. In this case it may be argued that it was not the increase of vagus tonus during the carotid pressure, but the simultaneous decrease of sympathetic tonus which caused the action. In a patient with complete A-V heart block, ventricular tachycardia and ventricular fibrillation were registered during attacks of the Stokes-Adams type. These attacks appeared during straining at stool and could always be induced by gentle digital stimulation of the rectum. The action of an autonomic reflex can hardly be excluded and the vagus nerves may have played a part. The appearance of auricular or ventricular extrasystoles during carotid pressure is common.

In the dog heart pretreated with aconitine, in amounts too small to cause any visible change in the electrocardiogram, brief faradic stimulation of the vagus nerve regularly led to the appearance of ventricular extrasystoles. Once extrasystoles were present vagal stimulation increased their number. The possibility was discussed that acetylcholine formed in the auricle during vagus stimulation may cause this reaction in the ventricles. It is known that under certain conditions in man, and in dogs under influence of aconitine, acetylcholine causes ventricular extrasystoles to appear.

The effect of vagal stimulation on ventricular automaticity has been repeatedly investigated, and the older literature has been reviewed by Erlanger. This author studied the chronotropic effect of the vagus during complete A-V block in the dog. He found an occasional but insignificant diminution of the rate "which developed slowly and reached its maximum later than the slowing of the auricles."

Similar results were obtained in two series of experiments performed on dogs, wherein complete A-V block was produced by severing the bundle of His or both bundle branches; prolonged vagal stimulation with strong faradic currents caused a slight but distinct slowing of the ectopic ventricular rhythm. However, in most experiments this action appeared after a latent period of three to five seconds, which makes a direct vagal effect improbable. Therefore these results were explained by the assumption mentioned above, namely, that acetylcholine, released in the auricle, is responsible for these minimal effects in the ventricles.

From the Department of Medicine, New York Medical College (Metropolitan Hospital Division), New York, N. Y.
Finally, the rate of the ectopic beats in a case of parasystole was studied during the administration of atropine, digitalis, pilocarpine, Doryl (carbamylcholine chloride) and carotid sinus pressure. During carotid sinus pressure a slowing effect on the ectopic ventricular centers could be calculated and this was interpreted as a result of direct action of the vagus.

In the present report two instances are described in which carotid sinus pressure influenced the automatic impulse formation in the ventricles with a clarity hitherto not described.

**Clinical Observations**

**Observation 1.** A 71 year old white man was admitted to the hospital for the fourth time because of shortness of breath and ankle edema. The heart was moderately enlarged, and a soft blowing systolic murmur was heard over the aortic and the apical areas. The blood pressure was 140/80. The electrocardiogram showed a sinus rhythm with frequent ectopic ventricular beats. The P-R interval was 0.24 second. Following administration of digitalis and salt-free diet marked improvement was noted.

It may be stated here that administration of digitalis leaf up to 0.1 Gm. daily caused characteristic RS-T segment changes, but did not modify the existing arrhythmia. Analysis of the arrhythmia revealed the presence of ventricular parasystole. The ectopic beats appeared at different phases of diastole; sometimes they coincided with the normal QRS complex so that combination beats resulted when parts of the ventricles were activated by the sinus impulse and parts by the ventricular ectopic impulse. Often two ectopic beats appeared in succession, thus enabling us to determine directly the duration of the ectopic interval. When sinus beats appeared between two ectopic beats the interval between the latter was a multiple of an ectopic interval measured directly. Thus, all criteria are fulfilled for the diagnosis of parasystole.

Vector analysis revealed that the vectorcardiogram of the ectopic beat was displaced anteriorly, to the right and upward. The terminal portion of the ventricular loop was described slowly and lay to the right and anterior to the zero point. This type of loop is similar to that seen in right bundle-branch block and indicates that the ectopic beat originated in the left ventricle.

Figure 1, like all other reproduced tracings of the patient shows lead II. The basic rhythm is a sinus rhythm with a rate of 75 per minute. Five ectopic beats are present and appear at different times during ventricular diastole. The ectopic intervals measure 346* (3 × 115 plus 1), 232 (2 × 115 plus 2) and 240 (2 × 115 plus 10). The ectopic interval directly measured on this day was 115. It varied on different days; it was as short as 86 and as long as 123.

Often series of ectopic beats appeared in succession (fig. 2). The longest series of uninterrupted ectopic beats numbered 60. Occasionally a sinus beat succeeded in reaching the ventricle as in figure 2a. After four ectopic beats with an interectopic interval of 100, a premature contraction appeared which was due to an abnormally conducted sinus beat. The latter does not interfere with the ectopic rhythm which therefore was protected from outside impulses according to the rules of the parasystolic mechanism.

Figure 2b was taken while the patient was receiving digitalis. The P-R interval was still 0.24 second. In this tracing sinus beats interfered with ectopic beats and the ectopic interval was 86. The interval between the last ectopic beat of the first group (of three) and the first ectopic beat of the second group (of four) measures 254, which is almost exactly as much as three ectopic intervals.

Figure 2c shows a constant ectopic rhythm with an interval of 94. After the second, fifth and eleventh ectopic beats, abnormal beats appear, resembling the ectopic beats but representing, as in figure 2a, abnormally conducted sinus beats.

While so far the tracings do not reveal anything unusual when compared with other instances of parasystole, the response to carotid pressure was quite abnormal. Invariably the ectopic rhythm was slowed down during the pressure and reached its normal values as soon as carotid pressure was interrupted.

* All figures represent hundredths of a second.
FIG. 1. Tracing showing parasystole with simple interference of two rhythms.

FIG. 2. Interference of ectopic rhythm with sinus beats. The latter are conducted within the ventricle in such a manner that they assume the shape of the ectopic beats.

FIG. 3. Carotid sinus pressure causes slowing of ectopic rhythm. The three strips are continuous. At the beginning of the figure 3a two ectopic beats follow each other and permit measurement of the ectopic interval. It is 108. The arrows in figure 3a and figure 3c indicate the beginning and the end of the pressure on the right carotid sinus. During the pressure, the sinus node is inhibited, the sinus rhythm is suppressed and an undisturbed ectopic rhythm is registered, the ectopic interval being 112, 112, 118, 120, 124, 126, 130, 132, 134, and 134. After termination of the carotid pressure (fig. 3c) a sinus beat is again seen between two ectopic beats, the ectopic interval measuring 112. The interval between the ectopic beats at the end of figure 3c, which are separated by two sinus beats measures 222, that is $2 \times 111$.

FIG. 4. Carotid pressure slows ectopic rhythm and, after it is discontinued, there is seen interference of ectopic, parasystolic rhythms with sinus rhythm simulating bigeminal rhythm. The three strips are continuous. The arrows again indicate beginning and end of the pressure. The ectopic intervals before the pressure in figure 4a were constantly 108. During the carotid pressure they measure: 110, 112, 112, 110, 116, 120, 112, 110, 128, 116, 116, 112, 116, 116 and 116. On this occasion the pressure was on the left carotid sinus; usually, pressure on this side was less effective than on the right in prolonging the interectopic intervals.

Figure 3 shows such an experiment and figure 4 also represents the effect of carotid sinus pressure this time performed during the presence of an ectopic rhythm. The slowing of the ectopic rhythm during the carotid sinus pressure is obvious.

After discontinuation of carotid pressure in figure 4, an interesting picture appears with a
sinus beat interpolated between each group of ectopic beats without disturbing the ectopic rhythm. A bigeminal rhythm is simulated.

In figure 5 the result of carotid pressure obtained on four different occasions is reproduced. It is evident that the slowing of the ectopic impulse formation begins immediately after the onset of carotid pressure and quickly disappears after its discontinuation. The slowing of the rate amounted in one experiment to 31 per cent of its value before the carotid pressure.

Another interesting feature which could repeatedly be observed in this patient is depicted in figure 6. One must assume that the sinus beat broke into the ectopic center, disturbing its impulse formation. The protection of the ectopic center, which makes parasystole possible was broken. Actually, parasystole disappeared for a moment and the sinus beat disturbed the ectopic impulse formation as it always does in a normal heart. This phenomenon was observed repeatedly but only during carotid pressure, and in more than 30 tracings from this patient it was never seen spontaneously.

Observation 2. A 72 year old patient was admitted to the hospital with congestive cardiac failure and severe Cheyne-Stokes breathing due to coronary sclerosis. A complete A-V block was found, occasionally showing varying forms of ventricular extrasystoles and of automatic ventricular beats. Figure 7 shows the effect of right carotid sinus pressure. The four strips are continuous. Before the carotid pressure the tracing (lead III) shows complete heart block with ventricular complexes which remain identical. During carotid pressure the P waves disappear, the ectopic beats assume a different form and remain so until 14 seconds after the end of the pressure; with the appearance of a ventricular extrasystole the original ventricular automatic beats recur. The ectopic intervals in figure 7 measure: 188, 196, 192, 192, 192, 188, 192, 192, 192, 192. Pressure on the left carotid artery had the same effect.

This observation shows a distinct effect of carotid pressure on the activity of the ventricle. The change of form of the ventricular ectopic beats during carotid pressure could be explained...
by shifting of focus or by disturbance of conduction. We are inclined to assume the first possibility. Changes of focus from beat to beat are known to occur in heart block and are explained by the presence of a bilateral bundle-branch block. That the change of focus during carotid pressure was not fortuitous is demonstrated by the fact that it could repeatedly be elicited by carotid pressure while it was not observed in this form spontaneously.

It may be argued, that changes of form of the ventricular complexes without changes of rate speak in favor of a disturbance of conduction and against a change in the focus of impulse formation. It is however established that under different experimental conditions ventricular centers, situated in either ventricle form impulses with the same rate.

**Discussion**

The tracings obtained in the first patient illustrate an instance of parasystole showing the following unusual features:

1. During carotid sinus pressure a pure ectopic rhythm was elicited, the basic rhythm being depressed.

2. During carotid sinus pressure there was progressive lengthening of the interectopic intervals, that is, slowing of the ectopic rhythm.

3. During carotid pressure, for a brief period a loss of the protection of the ectopic center from other impulses (protective block) occasionally was seen.

4. Arrhythmias appeared owing to the interference between ectopic and sinus rhythms which imitated bigeminal rhythms.

In the second patient carotid pressure led to the appearance of varying forms of ectopic beats with little or no change of rate. Whereas spontaneous changes or variations of the form of the ectopic ventricular beats were occasionally seen in this patient, the gradual change from the normal form as noted during carotid sinus pressure, and the return to normal when the pressure was discontinued were not seen spontaneously.

Thus in both subjects an effect of carotid pressure on ectopic ventricular impulse formation was observed; in observation 1 this effect was demonstrated more clearly than ever before. This was due to the fact that for the first time the lengthening of the ectopic intervals during carotid sinus pressure could be measured directly. We are fully aware that the observed changes during carotid pressure could be attributed to inhibition of the cardiac accelerator nerves, which has been demonstrated with the oscillograph during carotid pressure, and not to a vagal effect. The immediate slowing of the heart after carotid pressure started and the rapid return of heart rate to the normal level when the pressure was discontinued (fig. 5) speak against an interpretation of the effect.
of carotid pressure as a pure sympathetic nerve phenomenon. However, the possibility that the changes during carotid sinus pressure are caused by the release of acetylcholine in the auricle cannot be denied. The gradual increase of interectopic intervals and the fact that this increase occasionally lasts longer than the carotid pressure support this contention (fig. 3). An action of the carotid pressure on the coro-
nary circulation could not explain these find-
ings, for a vasoconstrictive effect upon it is un-
proven, and the changes could not appear so rapidly if the coronary circulation were involved.

The appearance in parasystole of an un-
disturbed ectopic rhythm during carotid sinus pressure has been observed clinically7,18 and experimentally.14 Our first patient shows this phenomenon with rare clarity. The occasional break of the protection of the ectopic center, usually and, in our opinion, incorrectly named "protective block," has been observed previously.12,15 The fact that here it was observed only during carotid sinus pressure is of interest, but a discussion of the possibilities involved would be mere speculation.

The occasional appearance of a bigeminus-like rhythm in observation 1 (fig. 4) is fortuitous and does not permit the interpretation of a bigeminal rhythm on the basis of parasystole. This has been repeatedly attempted without success.

Conclusions

A patient with a classic type of ventricular parasystole is described. Carotid sinus pressure inhibited the sinus rhythm and permitted the ectopic parasystolic rhythm to proceed undisturbed. During carotid pressure the rate of the ectopic rhythm was slowed by as much as 31 per cent of its rate prior to carotid pressure. When carotid pressure was discontinued, the ectopic rhythm returned to its pre-existing rate.

Carotid sinus pressure also caused a break in the protection of the ectopic center (protective block). This phenomenon was seen several times during carotid pressure and never without it.

A patient with A-V block is described in whom carotid sinus pressure repeatedly caused the form of the ventricular complexes of the ectopic center to change without any change of rate. This phenomenon is explained by a shift of the pacemaker.

Sumario Español

Se describe un paciente con la variedad clásica de parasistole. La presión sobre el seno carotídeo inhibió el ritmo sinuatrial y permitió el ritmo parasistolico ectópico proseguir sin interrupción. Durante la presión carotídea la frecuencia del ritmo ectópico fue retardada por tanto como un 31 por ciento de su frecuencia antes de la presión sobre el seno carotídeo. Cuando la presión sobre el seno carotídeo fue descontinuada, el ritmo ectópico retornó a su frecuencia preexistente.

Presión sobre el seno carotídeo también causó una descontinuación del centro ectópico (bloqueo protector). Este fenómeno fue observado repetidas veces durante la presión sobre el seno pero nunca sin esta.

Un paciente con bloqueo A-V se describe en el cual la presión sobre el seno carotídeo repetidamente causó la forma de complejos ventriculares del centro ectópico cambiar sin producir cambio en la frecuencia del pulso. Este fenómeno se explica por un cambio en el pacificador del corazón.

References


Changes of Ventricular Impulse Formation during Carotid Pressure in Man
M. GOLBEY, C. P. LADOPULOS, F. H. ROTH and D. SCHERF

Circulation. 1954;10:735-741
doi: 10.1161/01.CIR.10.5.735

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1954 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/10/5/735

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/