Table 1. Lowest gibberellin dosages causing complete inhibition of bud development in *Prunus* species.

Species	Floral buds		Vegetative buds	
	Mg/lit.	No. of applications	Mg/lit.	No. of applications
Peach	>500	2	>500	2
Apricot	50	2	250	2
Cherry	250	1	>500	2
Almond	50	2	250	2
Plum	50	2	250	2

stamens, and pistils were clearly distinguishable (Fig. 1*e*). However, none of the buds from treated branches showed signs of even the first phase of floral differentiation. The central regions were occupied by growing points of the vegetative, rather than the floral, form (Fig. 1*f*). Bracts were still being cut off from the growing points. No primordia of flower buds appeared in the axils of any bracts. The following spring a few flowers were formed, showing that floral differentiation and development had occurred in the exceptional bud. In vegetative buds from treated branches, microscopic structure was indistinguishable from that of control buds. In the spring there was no evidence of any previous damage to these buds.

In 1959, the effects of gibberellin were investigated in almond (*P. amygdalus* cv. Jordanola), plum (*P. domestica* cv. President), and peach (*P. persica* cv. Fay Elberta) as well as apricot and cherry. Treatments included 50, 250, and 500-mg/lit. concentrations; some branches of each species received a single application at full bloom, and other branches were sprayed a second time approximately one week later. [For details of treatment procedures and other types of gibberellin effects, see Crane *et al.* (3)].

The effects of the treatments on bud development were studied in the latter part of September. In all the species floral development in the controls had advanced to stages comparable to those illustrated for the apricot and cherry in the 1957 experiments. More than a dozen buds of treated and control branches were dissected under a stereoscope (×20) to determine whether flower buds had formed and whether vegetative buds appeared capable of survival and future development. Criteria used in the latter evaluation were color (whether a healthy green, or yellowish or brown) and texture (succulent versus granular and partially desiccated). The lowest dosages which inhibited bud development so severely that continuing development was considered unlikely are given in Table 1.

As expected, the five forms of *Prunus* studied here exhibited variation in response to gibberellin dosage. In the peach, two applications of gibberellin, of 500-mg/lit. concentration, were not sufficient to influence floral or vegetative bud development. In the apricot, almond, and plum, however, two applications, of 50-mg/lit. concentration, completely inhibited flower bud development. The cherry was intermediate in its response to dosage.

The inhibition of lateral bud development by gibberellin was not an aspect of general growth restriction. Excessive growth was stimulated in other plant regions; the higher the dosages the more extensive such growth and the greater the bud inhibition. Internodes lengthened in some spurs and long shoots. Stem diameter increased in certain species; in the apricot this was found to result from stimulated cambial activity (*4*). Petiole length, or diameter, or both, were increased in some cases. In the cherry (the only one of these species in which terminal buds remain viable from year to year), length growth of spurs was stimulated by some gibberellin doses, resulting in about twice the number of nodes as in control spurs. Similarly in some other species, the vegetative bud immediately below the dead terminal bud of a spur developed into a short branch.

Inhibition of cell division was an immediate effect of gibberellin, leading to restriction of lateral bud development. This was apparent from the greatly reduced zones of cells capable of dividing in treated buds, and also from retarded formation of leaf and bud scale primordia. The failure of inhibited vegetative buds to survive and develop the following year suggests either a toxic effect or one of prolonged starvation, either of which could have blocked cell division. The situation in lateral buds of *Prunus* is in sharp contrast to that in terminal buds of the rosette plants *Hyoscyamus niger* and *Samolus parviflorus*, in which gibberellin greatly stimulated mitosis in subapical regions (*5*). It contrasts also with the stimulated cell division implicit in excessive growth of terminal buds in the cherry after gibberellin treatment. Apparently physiological or anatomical differences, or both, between terminal and lateral buds may influence the effects of gibberellin. In *Prunus*, lateral bud inhibition can scarcely be considered a matter of intensified apical dominance, as indicated by the following evidence. When excessive terminal growth of cherry shoots was stimulated, development of lateral vegetative buds was not blocked. Also, in other species, when the first subterminal vegetative bud on a spur failed to develop, even after high gibberellin doses, the other lateral buds were nevertheless inhibited.

That gibberellin may have blocked floral initiation by affecting other processes than those concerned with cell division alone is suggested by the inhibition of floral bud development by considerably lower dosages than those required to suppress vegetative bud development. Reasons for considering floral initiation, rather than floral differentiation, as the blocked phase are as follows. Gibberellin was applied during the floral initiation period in both years. The interval between floral initiation and differentiation is 3 months in cherry, 4 in peach and plum, and 6 months in apricot and almond (*6*). An all-or-none effect in flower bud formation was noted; all treated floral buds examined were either as advanced in development as controls, or showed no evidence that floral differentiation had begun. It appears, therefore, that gibberellin may have interfered in some manner with processes concerned in floral initiation.

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2 November 1959

The Sun Azimuth Compass: One Factor in the Orientation of Homing Pigeons

Abstract. In accordance with the theory of the sun azimuth compass (*1*), displaced homing pigeons are misled in a predictable way if their "internal clock" has been reset by exposure to a time-shifted sequence of day-night cycles.

Several species of birds have been trained to respond to particular compass directions in stationary training cages (*2*). By resetting the birds' chronometers, or "internal clocks," predictable deviations from the training direction were obtained (*3, 4*). The question as to whether homing by free flying pigeons could be influenced in a similar manner has not previously been clearly answered, the relevant experiments (*5*) having been inconclusive.

Recent work at Wilhelmshaven, Germany, and Durham, North Carolina, has answered this question. The results described below were obtained from 55 releases under sunny conditions over a period of 4 years. In Fig. 1*A*, results from Germany are involved exclusively; in Fig. 1*B* and 1*C*, about 21 percent of the results were obtained in North Carolina. For each release, two groups of about 18 pigeons each were prepared simultaneously in the following manner: both experimental and control flocks were placed in separate, light-proof rooms, within which artificial day-night cycles could be provided. For the experimentals tabulated in Fig. 1*A*, light was switched on and off 6 hours earlier than sunrise and sunset, respectively; for those in Fig. 1*B*, 6 hours later than sunrise and sunset, respectively; and for those in Fig. 1*C*, the day-night sequence was completely reversed (shifted 12 hours). The control flock was provided with a light-dark schedule which followed the natural day. For releases involving 6 hours of time shift, both groups were confined for at least 4 full days; for a shift of 12 hours, they were confined for at least 7 days. Preliminary work had shown that these periods were sufficiently long for shifting to take full effect (*4*).

All of the birds, in groups containing roughly equal numbers of experimentals and controls, were displaced to release points in various directions from home and at distances varying from 5.5 to 100 miles from home. The releases were timed to fall within the light period which the artificial and the natural day had in common. Experimental and control birds were released singly and alternately, and were followed with field glasses to determine their bearings until they vanished. Observers at the loft recorded arrival times of the birds which were individually marked.

Even normal pigeons almost never head exactly in the actual direction of home. Influences, so far unknown, cause deviations which vary from one place to another but which are, within certain limits, characteristic of each release point. At three release points out of the 17 involved in this report, the mean local deviations from the home direction exceeded 55°; at 14 release points they ranged up to 55° right or left of the true home direction. Therefore, the departures are clearly oriented in the general direction of home, but for a strictly experimental basis of comparison, the bearings of the experimental birds at vanishing are plotted in Fig. 1 with reference to the combined mean vanishing bearings of the controls and not to the actual home

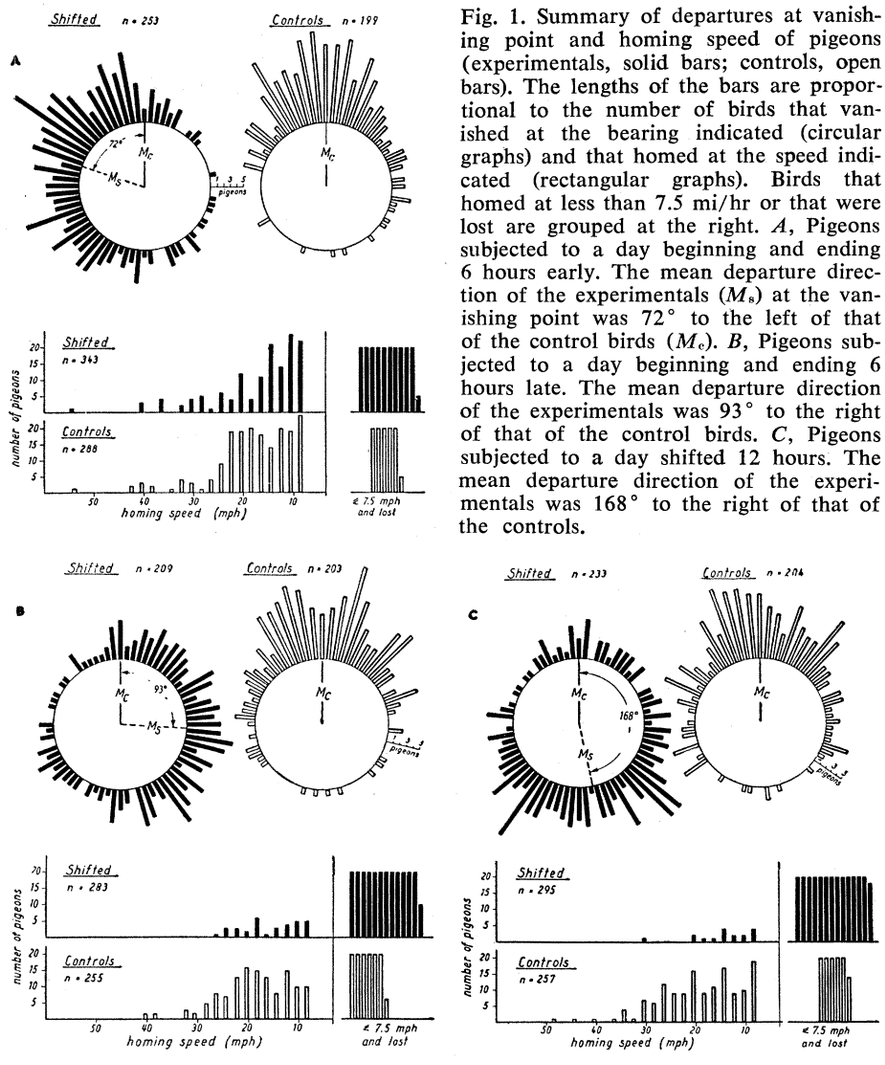

Fig. 1. Summary of departures at vanishing point and homing speed of pigeons (experimentals, solid bars; controls, open bars). The lengths of the bars are proportional to the number of birds that vanished at the bearing indicated (circular graphs) and that homed at the speed indicated (rectangular graphs). Birds that homed at less than 7.5 mi/hr or that were lost are grouped at the right. *A*, Pigeons subjected to a day beginning and ending 6 hours early. The mean departure direction of the experimentals (M_s) at the vanishing point was 72° to the left of that of the control birds (M_c). *B*, Pigeons subjected to a day beginning and ending 6 hours late. The mean departure direction of the experimentals was 93° to the right of that of the control birds. *C*, Pigeons subjected to a day shifted 12 hours. The mean departure direction of the experimentals was 168° to the right of that of the controls.

direction. The figures therefore give the mean deviation of the experimentals. But they show a falsified degree of scatter of the experimentals as compared with the controls, because the average deviation of the experimentals varies somewhat among separate releases. If one summarizes the mean scatter of each single release, the experimentals turn out to have only a slightly increased scatter.

Extrapolating from the results obtained from the stationary cage experiments (*4*), we would expect the birds whose day had been advanced 6 hours to deviate about 90° to the left of the controls, while those whose day had been retarded 6 hours should deviate 90° to the right, and the reversed-day birds should shift 180° from the direction chosen by the controls. The departures summarized on the accompanying graphs show that this expectation is largely realized. This fact is underscored by a drastically decreased homing performance among the experimentals. Reports on the lost experimentals show that they continued to move roughly in the direction chosen when they were released (*4*). Clearly, then, the sun azimuth compass is one basic mechanism in pigeon orientation. However, a small proportion of experimentals were able to home rapidly despite their shifted day. These statements suggest that other factors, as yet unknown, may be operative as well.

Nevertheless, by supporting the postulated function of the sun azimuth compass, the findings could be interpreted according to Kramer's idea of "map and compass" process (*6*) in which orientation is supposed to consist of two steps, one establishing the geographical position of displacement and the other defining compass directions. About the first step we do not yet know anything (*7*).

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Adaptation of Cardiac Output to Peripheral Runoff Studied in Intact Dogs

Abstract. A pump with sinusoidal piston movement was connected to the abdominal aorta of intact anesthetized dogs, causing slow oscillations of arterial blood pressure. Evidence was found for the existence of a mechanism which enables the heart to adjust its output immediately to changes of peripheral outflow.

In previous publications (*1*) Wetterer and I described a method by which we produced slow, nearly sinusoidal oscillations of the arterial pressure in intact anesthetized dogs. At that time it became apparent that these oscillations do not cause any reflex activity in the cardiovascular system and that the vasomotor tone is unchanged. Since recent studies of the performance of the heart (*2*) emphasize the importance of experimentation in intact animals as compared to experimentation in preparations such as Starling's, it became obvious to us that this method would be most suitable for such studies.

Twelve experiments were performed on large dogs anesthetized with 30 mg of Nembutal, or 3 mg of morphine sulfate, plus 200 mg of sodium barbital per kilogram. A pump with a nearly sinusoidal piston movement (a specially adapted Harvard respiration pump) was connected to the abdominal aorta by way of a short polyethylene tube of 3.2-mm inside diameter via the right femoral artery. The volume displacement of the pump was variable within the range of 50 to 100 ml. The piston movement was continuously recorded by means of a linear differential transformer. The pump was operated with frequencies from 0.18 to 0.30 cy/sec and in operation caused a slow oscillation of the mean arterial pressure by alternatingly taking out and reinjecting blood. Arterial blood pressure was measured in the aortic arch by means of a miniaturized catheter tip manometer with a natural frequency of 500 cy/sec (*3*). The manometer was introduced through the left femoral artery. A catheter tip flowmeter with a natural frequency of 160 cy/sec (*4*) was placed in the ascending aorta close to the aortic valves by way of the left carotid artery. Clotting was prevented by the injection of 5 mg of heparin per kilogram. Calibration of the flowmeter was accomplished *in situ* by taking simultaneous dye-dilution curves (*5*). Zero flow in the ascending aorta was obtained by arresting the heart for a few seconds by stimulation of the left vagus nerve.

Our recordings taken during operation of the pump show that the oscillation of the aortic pressure, while accompanied by significant changes of aortic flow, is not reflected in the pressure in the right atrium. They demonstrate further that operation of the pump does not cause any changes in heart rate. This, as well as the fact that the arterial pressure after the pump is stopped returns within 1 second to the level recorded before the pump was started, indicates that the pressure oscillation does not produce reflex changes in the circulatory system.

For the evaluation of the recordings the area under the curve of the aortic flow, which was taken as a measurement of cardiac output, was measured with a planimeter, and each stroke volume was calculated. The volume exchanges of the pump were derived from recording the piston movement.

It was found that for any given diastolic aortic pressure, the algebraic sum of cardiac output and volume injected or withdrawn by the pump during the same systole was a constant. This is shown in Fig. 1*A*, where stroke volume plus pump volume is plotted against diastolic aortic pressure P_d. It can be seen that values which were obtained with three different pump frequencies follow closely the same line. This shows that although the rate of inflow or outflow produced by the pump was greatly changed by changing the pump frequency, any variation of total systolic inflow into the arterial system was fully and immediately compensated by the heart. Since the effect of the pump represents in a sense a continuous change of the peripheral outflow such as would result from a change of the peripheral resistance, one must conclude that this mechanism adjusts cardiac output immediately to changes in peripheral outflow.

The constancy of the total systolic inflow for a given diastolic pressure must necessarily lead to a constancy of the aortic pressure pulse with respect to this diastolic pressure, since, as stated above, the vasomotor tone and therefore all other parameters, such as the systolic runoff and the elasticity coefficient of the arterial system, are unchanged by the operation of the pump. This is demonstrated in Fig. 1*B*, where systolic aortic pressure P_s is plotted against diastolic pressure P_d in order to show values obtained during injection as well as withdrawal of the pump. The ratio P_s/P_d was found to be changed only during the brief periods of respiration of the animal, where systolic pressure rose to relatively higher values. Therefore, only pressure cycles without respiratory activity were evaluated (*6*).

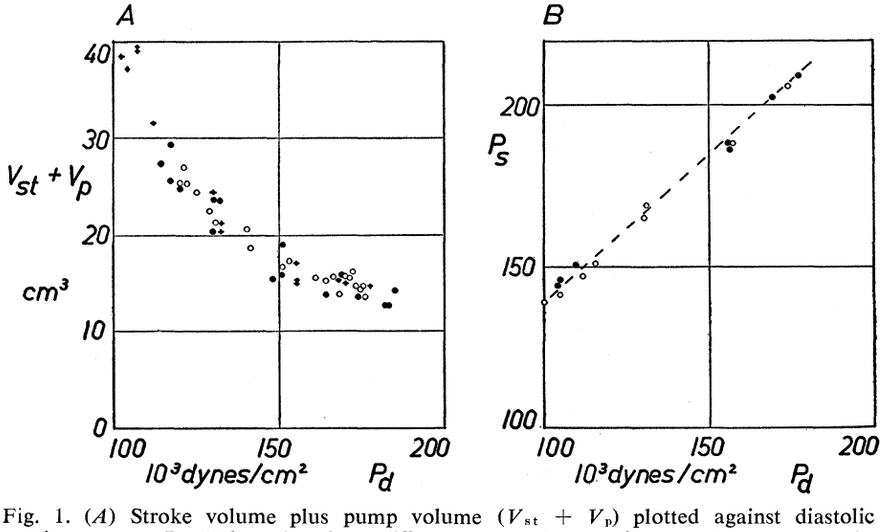

Fig. 1. (*A*) Stroke volume plus pump volume ($V_{st} + V_p$) plotted against diastolic aortic pressure P_d. Values for three different pump frequencies: +, 0.28 cy/sec; ●, 0.22 cy/sec; O, 0.16 cy/sec. (*B*) Systolic aortic pressure P_s plotted against diastolic aortic pressure P_d; O, during withdrawal; ●, during injection of pump.

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