A simple method for the simultaneous recording of blood pressure and heart rate

Blood pressure and heart rate can be simultaneously and accurately recorded, over a wide pressure range, using a single pressure transducer input incorporated into the circuit outlined in Fig. 1.

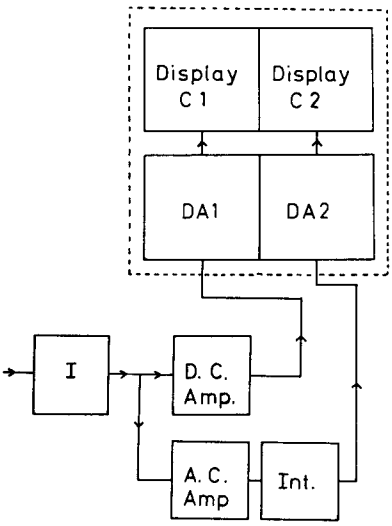

FIG. 1. Schematic diagram of the apparatus used for the simultaneous measurement of blood pressure and heart rate.
I = Input source: a pressure transducer (Bell and Howell: Type 4–326–L212). D.C. Amp = a blood pressure amplifier ('Devices' Type D.C.2D). A.C. Amp = an A.C. amplifier ('Devices' Type A.C.7). Int = a ratemeter ('Devices' Type 2751). D.A.1 and D.A.2 = driver amplifiers for channels 1 and 2. Displays C1 and C2 = display channels 1 and 2.

The equipment shown in broken outline represents the components of a Devices (M2 type) two channel pen recorder fitted with D.C.5 pen driver amplifiers.

Because the A.C.7 amplifier is not used as a differential amplifier, a shorting link must be placed between pins 1 and 2 of the 3 way jack plug which is inserted in the "signal input 1" jack socket (see diagram of input signal paths, Fig. 7, 46 Devices, M2R Manual).

Description of system. The pulse pressure impulses are converted, by means of the input transducer, into an electrical signal which is then used as the primary signal for activating both the pressure amplifiers and the rate meter. To record heart rate, the signal is passed into the amplifier, and from thence into the integrator before being fed into the second channel of the pen recorder. Blood pressure is displayed on Channel 1.

If pulse pressures are low, the signal will be insufficient for feeding directly into the integrator and this will fail. Therefore, a means of amplifying the pulse signal is essential and for this reason the A.C.7 amplifier is incorporated into the system. This amplifier also has useful time constant and filter facilities for exclusion of unwanted signal components.

Thus simultaneous blood pressure and heart rate measurement and recording can be made and, unlike ECG-actuated heart rate meters, distortions and artifacts which may arise during electrical stimulation of the preparation are isolated.

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Antagonism by methysergide of the 5-hydroxytryptamine-like action of toxic doses of fenfluramine in dogs

Fenfluramine produces anorexia in experimental animals and in man, but does not stimulate the central nervous system (Le Douarec, Schmitt & Laubie, 1966; Colmore & Moore, 1968). It has been reported to cause depletion of catecholamines both centrally and peripherally (Ziance & Kunnard, 1968; Duce & Gessa, 1966) and in contrast to amphetamine to release brain 5-hydroxytryptamine (Opitz, 1967; Duhault & Verdavainne, 1967). Also in contrast to amphetamine it lowers body temperature in experimental animals (Jespersen, Bonaccorsi & Garattini, 1969; Bizzi, Bonaccorsi & others 1969).

The effect of fenfluramine alone and in combination with methysergide (a specific 5-HT antagonist) was studied in 5 male beagle dogs by recording the rectal temperature and observing gross behaviour. (±)-Fenfluramine hydrochloride or saline was given subcutaneously after recording of the basic temperature, and 2½ h later methysergide bimaleate was administered intraveneously. Each dog was tested 3 times at 7–10 day intervals.

Fenfluramine causes hypothermia in dogs—as in other species—with maximum effect 2½ h after injection. Methysergide given alone does not influence body temperature at the dose tested, but when given 2½ h after fenfluramine a clear-cut and statistically significant antagonism of the fenfluramine-induced hypothermia was revealed. [$P < 0{\cdot}05$, $P < 0{\cdot}01$ and $P < 0{\cdot}01$ at 3, 3½ and 4 h respectively (Student's *t*-test)].

Fenfluramine caused changes in behaviour similar to those reported by Bogdanski, Weissbach & Udenfriend (1958) after injection to dogs of 5-HTP, a 5-HT precursor, which at decarboxylation raises the brain level of 5-HT up to 10 times (Udenfriend, Weissbach & Bogdanski, 1957). The fenfluramine-induced reactions were: sedation, mydriasis, apparent blindness, whining by petting, diarrhoea and unwillingness to keep still during measurement of rectal temperature. All these symptoms were more or less improved after methysergide. Two of the dogs behaved quite normally half an hour after the injection, while the other 2 dogs were still partially sedated. The fenfluramine controls continuously showed the mentioned symptoms up to 5–6 h after administration.

Some evidence was obtained that methysergide also antagonized the fenfluramine-induced anorexia, because when food was presented 1 h after the administration of methysergide or saline, none of the 4 dogs treated with fenfluramine + saline showed any interest in the food, while 2 of the 4 dogs treated with the combination ate with fairly good appetite, and 1 dog showed interest without eating.

The antagonism of subcutaneously administered methysergide (0·05 mg/kg) to the fenfluramine sedation and discomfort was confirmed in an experiment with 4 female beagles. The antagonism judged on gross behaviour observations was evident in 3 of these dogs which had received 2·5, 5 or 20 mg/kg fenfluramine subcutaneously about 3 h before. The behaviour of 1 dog given fenfluramine 5 mg/kg was not improved by methysergide.