a test particle of proper mass m_0 moving radially inwards. It is easy to show that the local velocity v along the trajectory is related to the velocity V at infinity by $(1-v^2)/(1-V^2)=(1-2M/r)$. The local rate of change of momentum is then found to be $m_0M(1-v^2)^{-\frac{1}{2}}r^{-2}(1-2M/r)^{-\frac{1}{2}}$ which may be interpreted as the gravitational attraction. This agrees with the classical value in the appropriate limit. One may interpret $M(1-v^2)^{-\frac{1}{2}}$ as the relative mass of the body as seen by the infalling test particle. The fact that this plays the part of the gravitational mass is one of the results got in a different way by Salzman. In most applications $2M/r \ll 1$, and we may think of r as nearly enough the classical distance. But the presence of the factor $(1-2M/r)^{-\frac{1}{2}}$ does recall the role of the Schwarzschild surface. So as far as this simple case is concerned, it checks that there is no change in the absolute position of this surface as it affects a moving test particle. Naturally, an observer on that particle may describe the surface in various ways depending upon what observations he makes of events in its vicinity.

The simple case might suggest that there is never any need to treat a Schwarzschild mass save with the aid of a standard description of the metric. But this is obviously not the case if we have to treat the interaction of two such masses. And this is where Salzman's work may provide a valuable start. In the first place, it supplies at any rate an approximation to the required field. In the second place, Salzman does indeed obtain by formulating a suitable Lagrangian an approximation to the behaviour of interacting masses. This Lagrangian is closely similar to one formulated by Landau and Lifshitz, which may be taken to confirm the usefulness of Salzman's metric. Incidentally, he concludes that "two-body effects can be important even in the case of a 'test' mass". Also he compares his Lagrangian with that for an analogous electromagnetic problem and pursues somewhat his analogy of gauge functions, but here the physical significance is scarcely clear. At any rate, Salzman has opened up a field in classical general relativity that will undoubtedly be further explored.

Puberty

from our Steroid Biochemistry Correspondent

THE mechanisms responsible for the increased secretion of androgens at the time of puberty are incompletely understood. The use of techniques for taking frequent samples of blood, the development of simple, sensitive and specific assay methods and the polygraphic recording of the stage of sleep has made possible an investigation of hormone secretion throughout a 24 h sleep–wake cycle. These techniques have shown that some hormones, for example growth hormone, luteinising hormone (LH) and follicle stimulating hormone (FSH), are secreted episodically and that their secretion may sometimes be related to the onset of a specific stage of sleep, particularly in the pubertal state. An increase of LH secretion, which may be important in initiating androgen secretion and puberty, occurring synchronously with onset of sleep was demonstrated in pubertal, and sometimes in later prepubertal, boys.

In a recent study (Boyar *et al., J. clin. Invest.*, **54**, 609–618, 1974) plasma LH and testosterone concentrations were measured at 20 minute intervals throughout a 24 h period in six normal pubertal boys. In all six boys there was an increase in LH concentrations with the onset of stage 4 sleep. LH was secreted episodically and although plasma testosterone concentrations did not show such marked variations, every major secretory episode of LH was followed about 20 minutes later by an increase in the plasma testosterone concentrations. Major secretory episodes of LH could occur at times when plasma testosterone concentration was increasing and close to the maximum concentration reached during the 24 hour period, whereas during the day, although testosterone concentrations decreased, no further major episodes of LH secretions were detected. In mid-puberty similar but smaller changes were observed.

To show that the increased androgen secretion was dependent upon sleep-associated secretion of LH, subjects were studied in which sustained sleep was delayed for 3 hours. Under these conditions the first LH secretory episode again coincided with the first sustained period of stage 4 sleep and was followed by an increase in testosterone secretion. Acute sleep–wake reversal studies also confirmed this relationship; concentrations of testosterone were significantly higher during the periods asleep than during the periods awake. In contrast to these results there was no consistent increase in LH secretion with the onset of sleep in sexually mature young men and testosterone secretion occurred equally throughout the 24 hour period rather than being highest during the sleep period.

The secretion of trophic hormones such as LH from the pituitary is often regarded as being controlled by a simple negative feedback mechanism; a high secretion of trophic hormone stimulates secretion of the hormone from the target gland and this increase in turn inhibits secretion of the trophic hormone. This feedback control system seems to be operating in sexually mature young men but such a control system does not seem to explain the changes observed during sleep at puberty when secretory episodes were often maximal at times when plasma testosterone concentrations were high. These observations suggest that at puberty the central nervous system exerts a regulatory influence which is more important than the classical feedback mechanism in controlling the secretion of LH and, indirectly androgens, at puberty. The mechanisms involved in initiating hormone secretion at this time remain to be unravelled.

Binding of streptomycin to ribosomes

from a Correspondent

STREPTOMYCIN is a well-known agent for precipitating nucleic acids. In addition to this general property, the drug is also a specific inhibitor of protein synthesis, and the inhibition is associated with the smaller (30S) ribosomal sub-particle. In *Escherichia coli*, it is known that mutations in protein S12 can confer resistance to or dependence on the drug (Ozaki, Mizushima and Nomura, *Nature*, **222**, 333–339; 1969; Birge and Kurland, *Science*, **166**, 1282–1284; 1969); and that these effects can be suppressed by mutations in proteins S4 and S5 (see for review Garrett and Wittmann, *Adv. Prot. Chem.*, **27**, 277–347; 1973). The question therefore arises as to whether streptomycin binds to the nucleic acid or protein moiety of the 30S ribosome, and this is a problem which has given rise to some controversy.

Gorini and his co-workers believe that in the ribosome streptomycin binds specifically to the 16S RNA. In their latest experiments (Garvin, Biswas and Gorini, *Proc. natn. Acad. Sci. U.S.A.*, **71**, 3814–3818; 1974) they have compared the binding of streptomycin (SM) with that of dihydro-streptomycin (H_2SM), an analogue which can be conveniently labelled with tritium. Both drugs seem to have identical binding sites on isolated 16S RNA, as demonstrated by chasing experiments, and both induced quantitatively identical 'misreading' effects, when tested with 30S particles from various different streptomycin resistant strains of *E. coli*, which bind varying low amounts of streptomycin as compared to the wild type. But the binding affinity of H_2SM is lower than that of SM, since the former but not the latter could be removed from 16S RNA or 30S subunits by simple dialysis. This effect enabled the authors to make a very interesting experiment: 30S sub-particles from wild type or streptomycin