DO WE HAVE TO TREAT POST-TRAUMATIC SEIZURES FOLLOWING THE FIRST SEIZURE? – NO

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There is no simple yes or no answer to this question. This question meets an other, more important one: how can we influence epileptogenesis, since the available AEDs prevent only ictogenesis.
We have two kinds of post-traumatic seizures: 1) immediate (within 24 h) and early (1-7 days) and 2) late seizures. Traumatic brain injury (TBI) has an acute brain tissue damage related direct effect, proportionally ictogenic with the lesion size, being analogue with the other symptomatic seizures. Available AE drugs are able to protect against the early seizures (ES) but not against the late seizures (LS). LS develops during a certain ripening period underlying by epileptic transformation of the involved brain tissue against which administration of AE drugs does not have protecting value.
Inhibiting early ictogenesis with AE drugs is reasonable but not preventive against LS ie (PTE). Undergoing experimental research work tries to understand how, if at al the early period plays role in epileptogenesis leading toward PTE. Another line of research try to find markers of epileptogenesis as targets for diseases modification, effecting on epileptogenesis. Recently the available new AEds (for example levatiracetam) are under scrutiny to find disease modifying effect of them beside the known anti- ictogeneic effect.
PTE is an important health issue:

1) accounts for 10-20 % of symptomatic epilepsy and 5% of all epilepsy (in USA and EU cca 0.5 million people live with PTE)
2) PTE may serve as a model to understand more focal epieptogenesis in general.
3) Search for disease modification in PTE may help to prevent epileptogenesis and not only ictogenesis in other epilepsies as well.